Tuberculosis in childhood / by Dorothy Stopford Price ; with a chapter on tuberculous orthopaedic lesions and other contributions by Henry F. MacAuley.

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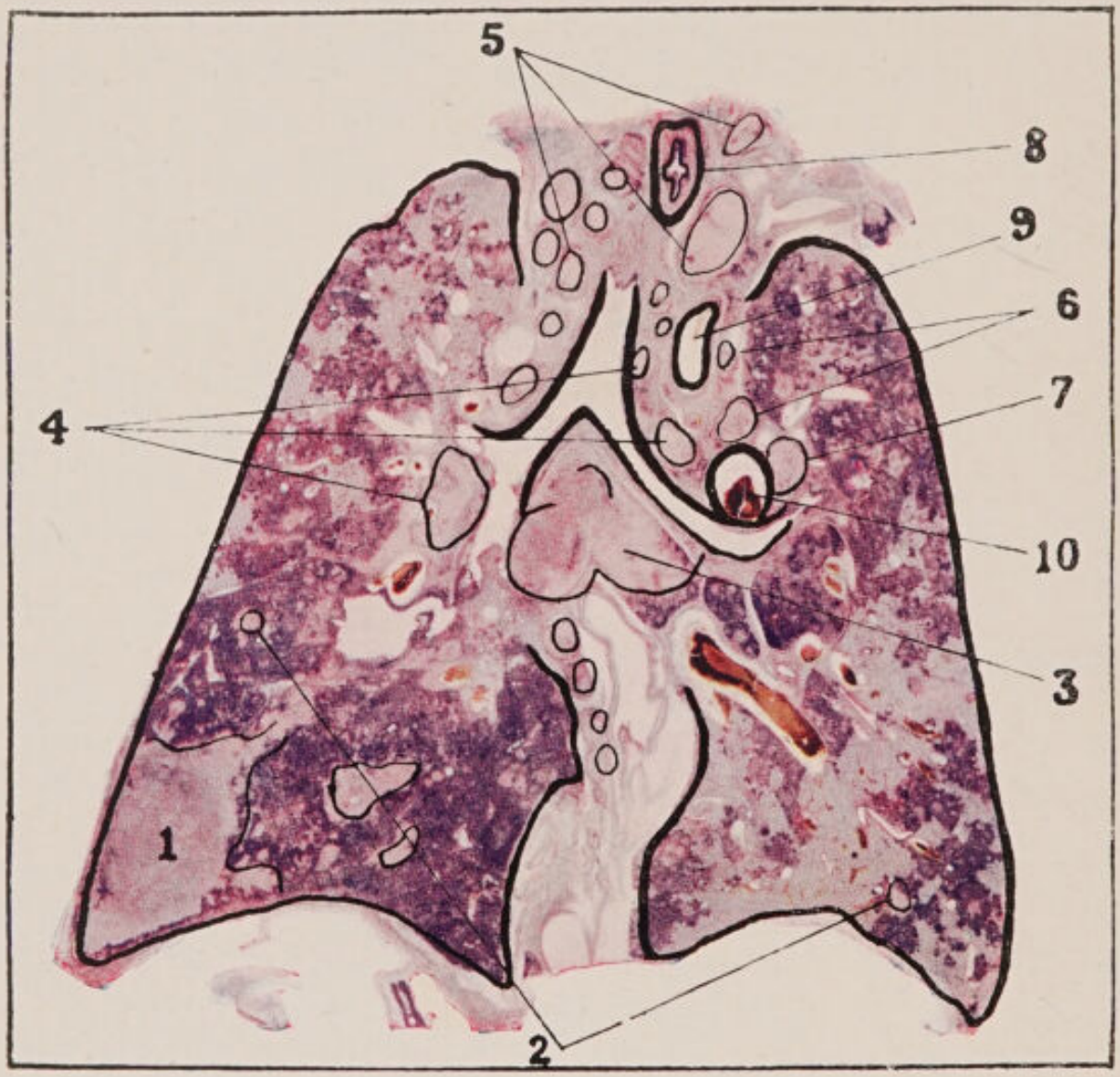

MEDIASTINAL GLAND TUBERCULOSIS IN AN INFANT.

1, Primary focus; 2, Hæmic foci; 3, Bifurcation glands; 4, Tracheobronchial glands; 5, Paratracheal glands; 6, Arcus aorticus gland; 7, Ductus Botalli gland (6 and 7 constitute the para-aortic group); 8, Oesophagus; 9, Aorta; 10, Pulmonary artery. The tuberculous infection has spread from the primary complex (1, 3) to all mediastinal glands (4, 5, 6, 7), and hæmic foci have occurred (2). ($\times \frac{4}{5}$.)

(*By courtesy of Dr. Stephan Engel.*)

TUBERCULOSIS IN CHILDHOOD

BY

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With a Chapter on Tuberculous Orthopædic Lesions and other Contributions

BY

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SECOND EDITION, FULLY REVISED
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PREFACE TO SECOND EDITION

THE whole book has been revised and brought into line with recent advances in our knowledge of childhood tuberculosis. The chapters on Classification, Epidemiology, and Tuberculin Tests, together with the sections on Erythema Nodosum, Phlyctenular Conjunctivitis, B.C.G., and Prognosis, have been entirely re-written. Many more figures have been incorporated from the experience of the author, and also from other workers in the school of tuberculosis which is developing in Ireland.

The line of approach to tuberculosis—namely, through the primary type—which was advocated in the first edition, has already begun to show good results when applied in practice; this approach may also be applied with success to prevention and treatment of tuberculosis in the young adult age period as well as in infancy and childhood.

D. S. PRICE.

DUBLIN,

December, 1947.

PREFACE TO FIRST EDITION

WHILST dealing for a number of years with sick children, the writer was forcibly impressed by the fact that many vague symptoms of ill health besides actual disease could be attributed to infection by the tubercle bacillus. Inquiries showed that scattered throughout the world literature were many helpful articles on the subject of tuberculosis in childhood, but few books existed except those written in foreign languages. Nine years' clinical investigation, aided by visits to foreign clinics, has strengthened the writer in the opinion that too little attention is paid to the subject in this country. This book, therefore, is intended to be a brief practical guide to the diagnosis and treatment of tuberculosis in children. The primary focus in every situation is discussed, but special emphasis is laid on the pulmonary lesion, on account of the predominance of inhalation as a mode of entry. There are also described methods of spread from the primary complex, with resultant lesions: lesions which may be either secondary (blood-borne) or tertiary (bronchogenic).

There is no condition which demands correct diagnosis more urgently than does primary tuberculosis in children and adolescents. Every case of bone and joint tuberculosis, of tuberculous meningitis, and of tertiary phthisis bears witness to the inaccuracy of the statement that primary tuberculosis is a benign and self-healing condition; many of such infections, indeed the majority, are overcome by individual natural resistance, but a number of untreated cases fail to heal and become the cause of protracted ill health; unfortunately a certain proportion of cases end fatally. Yet when once a system of early diagnosis is introduced, and treatment afforded during the danger period early in the primary infection, the condition does become a benign one—that is to say, extension of lesions is avoided,

and the death-rate from tuberculosis in infancy, childhood, and adolescence falls. Rest is the important factor in bringing about this improved state of affairs.

The methods employed for recognition of the primary focus are simple and within the scope of every practitioner. Reliance is placed most of all on the tuberculin skin test, and secondly on radiological appearances; interpretation of the latter must depend on a knowledge of the tuberculin reaction, otherwise children may be condemned to months of inactivity on account of conditions which are non-tuberculous. On the other hand there may be seen another distressing picture, that of a child in the stage of primary tuberculosis which is unrecognized, and who is denied the rest which is necessary to heal the lesion and so to overcome the toxic effects which accompany the implantation of the tubercle bacillus on virgin soil; the child's whole future health may depend on a short period of rest at this moment.

Tuberculosis presents the greatest danger to life during the age periods of infancy and adolescence. Thirty years ago Ghon found that 90 per cent of infants so infected died. During the past four years, working on the lines indicated in this book, it has been possible in the Tuberculosis Department of St. Ultan's Infant Hospital to reduce the mortality-rate from tuberculosis in the 0–1 year period from 77 per cent to 28 per cent. In the Children's Extern Department of the City of Dublin Hospital, children aged 3–14 years suffering from primary tuberculosis have been treated by bed-rest, mostly in their own homes; amongst cases with definite radiological signs of pulmonary involvement, extension of lesions has been avoided, with a death-rate of 1·5 per cent. Adolescent primary tuberculous lesions have been treated on the same lines, with promising results.

The last chapter of the book has been devoted to the subject of early diagnosis and treatment of orthopædic tuberculous lesions; these often come first under the eye of the physician, although they are really surgical conditions. This chapter has been written by Mr. H. F. MacAuley,

the well-known orthopædic surgeon, who possesses a wide experience of bone and joint tuberculosis in children, and whose clinical work has been attended for many years with outstandingly successful results. The writer owes most grateful thanks to Mr. MacAuley for so kindly contributing to the book, and for his collaboration on abdominal, renal, and cervical gland tuberculosis in the chapter on extra-pulmonary tuberculosis.

The writer wishes to thank three friends in particular for help and encouragement: Dr. Walter Pagel, who has explained the pathologico-anatomical processes in tuberculosis in so illuminating a manner, and Professor Arvid Wallgren, who has given a lead in research into the clinical aspects of tuberculosis in children. To Dr. Pagel the writer is indebted for photographs and reports on five anatomical specimens; to Professor Engel for the section of a child's lung in colour reproduced for the first time.

The sincere thanks of the writer are also expressed to all the colleagues and friends in Dublin and in other parts of Ireland who have helped with the X-ray films and by co-operation and useful discussion and criticism.

D. S. PRICE.

DUBLIN,

April, 1942.

CONTENTS

TUBERCULOSIS IN CHILDHOOD

CHAPTER I

CLASSIFICATION OF TYPES

The classification followed in this book will be that of three types of tuberculosis :—

Type I.—A primary focus with lymphatic spread to the corresponding lymph-gland (primary tuberculosis).

Type II.—Dissemination by the blood-stream.

Type III.—Bronchogenic tuberculosis—restricted to the lungs (isolated phthisis, so-called " adult-type ", which is however not infrequently observed in children).

It is not always possible for the clinician to draw an arbitrary line between pathological types, but, speaking broadly, this is not difficult in the majority of cases. The tuberculous process is the same no matter what the route of entry ; for the sake of clearness in this chapter, however, it will be simplest to concentrate on the pathological processes as they occur in the lung, for it is by the respiratory route that the vast majority of children are infected.

Tuberculous processes have been the subject of research in many countries for over forty years. Indeed in 1876 Parrot produced a treatise on the primary lung and bronchial gland focus. Kuss (1898), Albrecht (1909), and Ghon (1916) further investigated the primary complex. In 1917, working in the light of knowledge provided by these earlier investigators, and greatly enriched by his own original observations, Ranke produced his classification of pulmonary tuberculosis, dividing the disease into three stages, primary, secondary, and tertiary. To-day this is, in broad outline, still a satisfactory theoretical working basis for the problem of the development of tuberculosis.

Whilst an understanding of the pathologico-anatomical processes forms the basis for the clinical handling of the disease, at the same time it must be clearly understood that throughout these types or stages the individual reaction of the infected child will vary according to the degree of natural or inherited resistance, of hypersensitivity, and of acquired immunity which is present in each case. Furthermore, clinically it is not possible to be aware of all the pathological changes which are going on in the patient.

Type I. Primary Tuberculosis.—The lesion is localized in the lung, and under this heading are included: (*a*) primary complex; (*b*) mediastinal gland involvement; (*c*) primary cavities.

a. Primary Complex.—The first type begins with the primary lodgement of the tubercle bacillus in the body. In most cases this occurs by inhalation, with implantation in the lung parenchyma. A *primary focus* is formed at the site of lodgement, whence infected material drains by the efferent *lymph-vessels* to the nearest lymph-gland at the lung root, the *primary gland.* This triad of events forms an entity which is known as the *primary complex.* This complex usually heals by fibrosis and calcification.

b. Mediastinal Gland Involvement.—If the primary gland fails to heal there may occur extension into neighbouring or contralateral glands. The clinical picture of mediastinal gland tuberculosis is often seen in children. These glands in their turn may heal or they may liquefy and discharge their contents into the blood-stream or, more rarely, into a bronchus.

c. Primary Cavities.—If healing of the primary focus does not take place, there may arise softening (liquefaction) and cavity formation. This condition is seen in infants and young adults. Primary cavities often disappear quite suddenly; on the other hand they may lead to a fatal termination by bronchogenic spread.

During the incubation period, i.e., the time interval between actual bacillary invasion and establishment of tuberculin hypersensitivity, and before the primary stage is clinically apparent, the child is *anergic* and gives a negative response to tuberculin; when the tuberculin reaction becomes positive, the child becomes *allergic* to tuberculin, and the primary complex may be recognized clinically. With this development of allergy the child suffers from a primary tuberculous infection.

Type II.—This is manifest when the tubercle bacilli which are circulating in the blood-stream form metastases. Bacillæmia can occur in two ways:—

a. From the primary focus bacilli enter a branch of the pulmonary artery or vein.

b. From the mediastinal glands bacilli can reach the main lymphatic trunks and thence enter the superior vena cava.

Massive flooding of the circulation from (*a*) and especially (*b*) leads to multiple and widespread metastases. Less heavy infections lead to solitary or few metastases. Between these two extremes there are many varieties and degrees of secondary dissemination, some benign, some less benign, and others fatal; for example, skin tuberculosis, bone and joint lesions, miliary disease, and meningitis. Type II is seen at its worst in young children, and so it is of great importance. If its manifestations appear within three months of first infection, whilst the child is still in a highly hypersensitive condition, and before any appreciable degree of immunity has developed, then the risk of meningitis or miliary spread is greatest; this danger, however, becomes increasingly less as the months go by, and clinical experience shows that after the lapse of a year danger of dissemination is almost negligible.

Type III.—This arises when there is a fresh focus in lungs which have already been primarily infected; this new focus may be due to recrudescence of a pre-existing lesion or to a new infection from without ('endogenous' or 'exogenous' reinfection). If *endogenous*, the focus arises from a hæmic or bronchogenic spread; the latter occurs as a rule from a liquefied primary focus ('primary cavity') or from the glandular component after its rupture into a bronchus or else from a liquefying early post-primary ('Simon' or 'Assmann') focus. If *exogenous*, it is either true reinfection (i.e., a group of foci, pulmonary or glandular, of the primary complex type which develop when allergy has waned completely), or else it is 'superinfection' (i.e., a new exogenous focus developing while allergy is still active); in the latter case no complete primary complex will develop, but only a focus in the lung. It should be mentioned that in the writer's opinion neither true reinfection nor exogenous superinfection are of great practical significance, although with continued exposure they may occur in exceptional cases. In children the most usual type of reinfection is endogenous.

Whatever the mode of infection, however, the fact remains that the lung tissue of an allergic (already infected) child will react to the new infection in a manner which differs from the response given to a primary infection (*see* Koch's phenomenon, p. 108). Type III lesions differ from primary in the following ways: (1) Some degree of acquired immunity is present in the former, expressed by the freedom of the extrapulmonary organs, i.e., isolation of the process to the lung, namely, the organ which has the least natural resistance to the tubercle bacillus; (2) The root glands are not involved in Type III lesion; (3) The foci which initiate Type III lesion show a marked tendency to liquefaction, in contrast to the focus of primary tuberculosis, which tends to fibrosis and calcification; (4) From a cavitated Type III lesion bronchogenic aspiration leads to the formation of new foci in other parts of the lungs, which in their turn cavitate and form typical 'phthisis'.

Thus it may be seen that whilst primary tuberculosis under adverse conditions tends towards generalization, Type III lesion restricts its spread to one organ, usually the lung. Although possessing some degree of resistance which is successful in isolating the disease to the lung, the subjects of bronchogenic phthisis nevertheless often succumb to the disease. In children it is obvious that when Type III phthisis is present it must follow primary infection without a long interval, and we know that the nearer to the primary infection the more vulnerable the child; this is one of the main reasons why in the child the course of bronchogenic phthisis is rapid and the tissue reaction infiltrative.

The classification of tuberculosis into 'childhood' and 'adult' types is not possible and should be abandoned. Pagel (1946) sums up the question in these words: "There can be no discussion of the fact that at the site of primary infection a primary complex will develop at any age . . . In the white race no age- or race-determined preference for either dissemination or bronchogenic tuberculosis can be found. In the age groups from 20 to 50 the reviewer's [Pagel] material shows the number of cases of disseminated tuberculosis to be equal to that of bronchogenic tuberculosis . . . The importance and frequency of disseminated forms in the adult, their chronicity and often benign course, reveal the fallacy inherent in the distinction of a childhood type with predominant dissemination, and an adult type with predominant restriction to the lung."

CHAPTER II

EPIDEMIOLOGY

The type of tuberculosis which is usually seen in childhood is the primary lesion. Of late years Type I tuberculosis is also becoming more common amongst young adults. Price finds in Dublin that at least 50 per cent of positive child reactors to tuberculin have a demonstrable lesion, either healed or active. Amongst these demonstrable cases 80 per cent have Type I primary lesions; the remaining 20 per cent are suffering from bone, joint, or abdominal (other than primary abdominal) lesions, or phthisis, miliary disease, or meningitis.

Children, unlike adults, cannot be classified as sputum-positive and sputum-negative cases. The 80 per cent or so of primarily infected cases are not and never have been sputum-positive; positive sputum is rare in children and when present indicates nearly always an advanced stage of the disease; it is only children suffering from bronchogenic and sometimes from disseminated tuberculosis who show tubercle bacilli in their sputum. Notification of primary tuberculosis, even where there is a demonstrable active lesion, is not compulsory; this non-notification is to be deplored from the view-point of prevention, for it is of paramount importance to heal the disease in this early stage; in achieving such healing by prompt treatment, extra food and money allowances are an asset to the patient, and the benefits conferred far outweigh any possible harm which notification may do to him; money spent at this stage will prove a national economy in the long run.

The importance of early diagnosis of Type I tuberculosis cannot be over-emphasized. Many children pass through primary tuberculous infection without illness and with no demonstrable lesion. But not all. At the time of infection a proportion of cases suffer an illness, definite or indefinite—namely, primary tuberculosis. By means of careful clinical and radiological investigation, it is possible to select those cases which require treatment and those which require supervision. In this matter it is obvious that knowledge of past and present

tuberculin reactions is of the utmost value in assessing the case. Even with the aid of tuberculin test, radiography, and clinical investigation, it is not possible to give a firm prognosis in every case; bone and joint lesions and meningitis do arise in cases under treatment. Still the development of secondary metastases is a rare occurrence in the child who has been treated both early and adequately during primary tuberculosis. On the other hand, all untreated cases of primary tuberculosis do not prove benign, for a proportion proceed to an extension of lesions.

In any community the incidence of tuberculous infection amongst the childhood population may be estimated by ascertaining the number of positive reactors amongst representative groups of children, healthy as well as sick. Positive reactors may be sub-classified in various ways: according to age; according to locality (urban or rural); in groups of supposedly healthy children or hospital patients; as contacts of known 'open' cases, or as unsuspected and leading to the discovery of an 'open' case. Positive reactors must be examined both clinically and radiologically and then divided into healed, active, or occult (no demonstrable lesion). The active cases must further be classified as Types I, II, and III. Only when this has been completed, will there be reliable statistics as to the tuberculinization and tuberculosis incidence in any given community. Wide surveys to discover tuberculin skin reactions, therefore, are a necessary preliminary to the success of a comprehensive campaign against tuberculosis in children.

Tuberculin surveys may be carried out amongst small children in welfare centres, and amongst older children in schools. In some countries or localities, routine tuberculin testing has been adopted; acting on the knowledge thus gained precautionary measures are taken, with the result that childhood tuberculosis in such countries is restricted to the primary type of lesion, and later on is noticed a marked decrease in the incidence of surgical tuberculosis. Others are content with the investigation of contacts, i.e., those children who come from a tuberculous milieu, sought for in the homes of notified adult cases. This latter method is a practical one, but its success depends on notification of all sputum-positive cases, which is in fact often incomplete; furthermore under this scheme extrafamilial and chance infections are frequently overlooked. As a general rule the incidence of positive reactors at all ages is higher amongst

urban dwellers than in rural groups; Rich (1944) shows this to be the case in the United States; Daniels (1944) finds it amongst Irish and Welsh, but not amongst English and Scottish nurses in England; rural figures from Ireland are shown in *Table I.*

Table I.—Tuberculin Testing in a Rural District in Eire (*Crowe*, 1942)

	Age in Years		
	0–5	5–10	10–15
Positive ...	4 (17 per cent)	10 (30 per cent)	18 (49 per cent)
Negative ...	19 (83 ,, ,,)	23 (70 ,, ,,)	19 (51 ,, ,,)
Total ...	23	33	37

	Age in Years		
	15–20	Over 20	Total
Positive ...	7 (44 per cent)	28 (67 per cent)	67 (45 per cent)
Negative ...	9 (56 ,, ,,)	14 (33 ,, ,,)	84 (55 ,, ,,)
Total ...	16	42	151

A marked reduction in the incidence of positive reactors has been observed universally during the past thirty years. In 1909 in Prague, Vienna, and Paris 70 to 94 per cent of children at 12 years reacted positive. Whereas in the past it was considered a waste of time to tuberculin test children in cities because nearly all reacted positive, now the employment of the test gives useful information; a practical point of value in testing is that it reduces by half the numbers who need radiological examination, for only positive reactors need be sent forward for this investigation. To-day in many cities less than half the population at school-leaving age are positive reactors, and in some rural districts only 10 to 20 per cent. Rich (1944) quotes 50 per cent positive at the age of 20 in the States; Alston (1946) found 56·6 per cent positive amongst 800 boys aged 6 to 16 years in an Industrial School in County Dublin; Price (1945), amongst 172 healthy girls entering a State school aged 14, found that

12·7 per cent were positive, and on leaving aged 18 that 30·8 per cent were positive. In *Table II* may be seen results of some recent tuberculin surveys.

Table II.—Tuberculin Reactions of 1330 Dublin Children Comparison with Other Surveys
(*Kavanagh*, 1946)

Age Group	1944–5	1941	1939	1939	1944
	Dublin Hospital (Kavanagh)	Irish Paed. Association	Belfast (Shaw)	London (Bradshaw)	St. Andrew (Mackenzie Institute)
	Mantoux 1–100	Hamburger Percutaneous	Mantoux P.P.D. 2 strengths	Mantoux 1–1000	Patch
	1330 cases	1121 cases	1634 cases	2827 cases	491 cases
	per cent	per cent	per cent	per cent	per cent
0– 1	2·2	4·6	—	—	—
1– 3	12·0	15·9	—	16·8	13·4
4– 6	31·0	27·1	—	39·1	25·0
7– 9	42·5	34·7	—	—	32·7
10–13	55·1	46·1	—	47·2	41·5
0–13	27·9	20·5	34·0	—	31·0

Series of tests in England and Ireland have been made amongst hospital patients rather than amongst healthy children. Capon (1938) found a positive reaction in 78 per cent of suspected tuberculous children in hospitals in Liverpool. D'Arcy Hart (1932) found a positive reaction to undiluted old tuberculin amongst London children in 97 per cent who were suffering from clinical tuberculosis; and again, in his extensive survey, amongst 490 clinically non-tuberculous controls, he found that in the 0–2 age period 6·5 per cent, 3–5 age period 18 per cent, and 6–10 age period 38 per cent reacted positive to undiluted old tuberculin.

In recent years there has been a decrease in the incidence of positive reactors amongst children. This decrease follows a general decline in the incidence of tuberculosis and a reduction in the number of sputum-positive cases. This change has been brought about through early diagnosis and modern methods for rendering infectious cases sputum-negative, by improved standards of living, and by an increase in tubercle-free milk supplies, with pasteurization of milk in large cities. Thus it has

come to pass that in most countries the primary infection, previously considered to be the prerogative of children, is still to be faced by half the population during adolescence or young adult life. This ' late ' primary infection brings its own problems. The decline of primary infection during the years of childhood will in all likelihood continue during the next quarter century, and tuberculosis in the child will become increasingly rare. As a result of this, the young adult population will be laid open to outbreaks of primary tuberculosis on exposure to infection, and, unless these cases are recognized and treated, there will occur an increase of the more serious forms in this age period. The susceptibility of the uninfected young adult will have to be protected by B.C.G. vaccination. It is in this manner that the Danes are dealing with the problem on the Island of Bornholm ; the island is now largely free from tubercle bacilli, and its young inhabitants fall a prey to the disease in considerable numbers when they go to Copenhagen or other cities ; such infection of young adults is now prevented by B.C.G. vaccination prior to leaving the island (Holm, 1946).

Contact.—The most fruitful field in which to seek for tuberculous infection and disease in children is amongst contacts of sputum-positive cases. The source of infection is to be found most frequently in the home, but it may also be found in schools or amongst neighbours. Kayne (1935) showed home infection to be high amongst young children, whilst extra-domiciliary infection increases in later childhood ; in the latter event it may be due to a single or ' chance ' exposure, which is difficult to trace. In well-to-do families disasters may arise from contact with a sputum-positive maid ; parents have been known to refuse to part with a satisfactory help or children's nurse, in spite of the warning of the family physician. Another very possible source of danger is the grandparent with chronic cough.

Examples of Intrafamilial Infection.—

Opie and McPhedran (1935) report from Philadelphia :—

450 home contacts 0–14 years	87·5	per cent positive to tuberculin.
486 non-contacts 0–14 years	53	per cent positive to tuberculin.

Enid Williams (1938) reports from Cardiff that amongst 80 child contacts, aged 9 months to 16 years, 55 per cent were positive to tuberculin ; those under 5 years showed a positive percentage of 66. Further figures are given in *Tables III and IV*.

Table III.—London Investigation of Contagion, 1929. Tuberculin Reactions in Home Contacts and Non-contact Controls
(*From Schlesinger and Hart*, 1930)

Group	Number Aged 0–10	Number Positive	Percentage Positive
Home contacts of pulmonary tuberculosis	68	48	59
Home contacts of open pulmonary tuberculosis (bacilli in sputum)	42	29	69
Home contacts of open pulmonary tuberculosis (sputum negative or unknown)	26	11	42
Home contacts of non-pulmonary tuberculosis	26	4	15
Non-contact controls	438	80	18
Bedroom contacts of open pulmonary tuberculosis (bacilli in sputum) ...	34	24	71

Table IV.—Contacts Found in 78 Tuberculous Infants under 1 Year
(*Price*, 1938)

Source of Infection	Deaths	Recoveries
Mother	19	9
Father	9	4
Uncle, aunt, brother, sister ...	9	5
Unknown	23	—

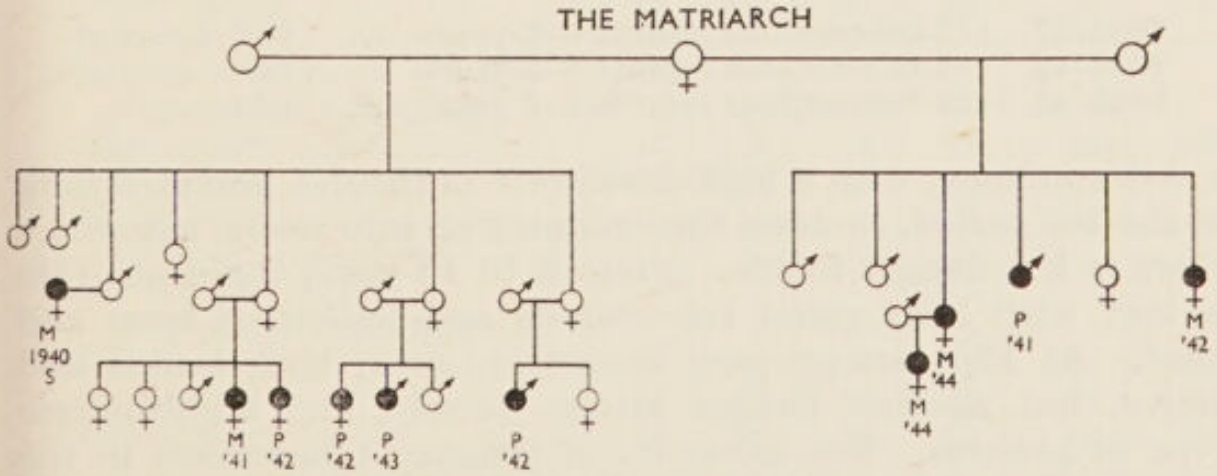
The havoc wrought by intra-domiciliary infection will be further exemplified by two instances from the author's experience :—

1. In 1942 two children aged 1–2 years were admitted to St. Ultan's Hospital, one dying of miliary tuberculosis, and the other, who recovered, with a tuberculous cavity. They came from a hostel where unmarried mothers lived with their babies. A survey was made of all the children in the hostel, after the source of infection, a young mother, had been removed to a sanatorium. Amongst 61 children under 6 years of age 19 were found to be positive to tuberculin (31 per cent) ; of these 19 children 13 showed radiological evidence of primary tuberculosis of the lung ; all were treated in preventoriums and were cured. In 1945 another young mother was taken to a sanatorium suffering from phthisis from the same hostel. All children were again

tested ; amongst 79 children under 6 years (excluding previous positive reactors who remained well) 36 were positive (45 per cent) ; of these 36 it was necessary to treat institutionally 18 suffering from primary tuberculosis ; in addition, one child of 2, who was negative to Moro test but positive to Mantoux 1–100, was found to have miliary disease and died in hospital. Twelve of the remaining negative reactors have received B.C.G. vaccination on a voluntary basis to date.

2. Appended is a family tree to demonstrate the number of persons who were infected by the introduction of a young tuberculous girl through marriage into a tubercle-free family. Even though she died three months after her marriage in 1940, the infection she introduced was handed on for the ensuing 5 years, mainly through the medium of the youngest girl of the second family, who died in 1942. It is worth noting that the infection selected those in the age groups 0–5 and 14–20 years, as represented by the adolescents of the second marriage and the grandchildren of the first marriage of the matriarch. It appears now that the ' epidemic ' is over ; all survivors are well since 1945, and all babies born since then have remained negative reactors ; the primary cases have healed. B.C.G. vaccination of the new babies is being considered, but not urged. The total casualties from this one case were 4 dead and 5 infected with primary tuberculosis.

SPREAD OF TUBERCULOSIS IN A FAMILY, 1940–5



M, Dead ; P, Primary ; S, Source of infection.

Examples of Extrafamilial Infection.—Fitzpatrick (1946) reports from Cork the case of a child aged 8 years, symptomless but sputum-positive, whom he found to be suffering from bilateral

bronchogenic phthisis. He examined her 38 classmates aged 7 to 9 years; all were positive reactors; 3 had healed primary complexes and 3 had small hilar opacities. Holm (1946) reports an epidemic of tuberculosis, including several cases of erythema nodosum, in a school; amongst 105 pupils who had been negative two months previously 66 then gave a positive reaction. The source of infection was found to be a teacher, with minimal apical lesion, who had held classes in a basement room which had been blacked-out.

Age.—There are two particular danger periods when infection by the tubercle bacillus not infrequently ends fatally; these are early infancy and adolescence. Thirty years ago it was said that of infants infected with tuberculosis 90 per cent died. After the employment of B.C.G. preventive vaccination, Wallgren (1936) showed that the infant tuberculosis death-rate in Gothenberg fell from 3·4 per thousand in 1922–6 to 0·3 per thousand in 1934–6, in the absence of a corresponding fall in the adult death-rate. In *Table V* it may be seen that a substantial reduction in the infant tuberculosis death-rate can be effected by early diagnosis followed by treatment.

Table V.—Result of Early Diagnosis and Treatment; 356 Tuberculous Infants in St. Ultan's Hospital (*Price*, 1946)

		Tuberculosis Mortality
1933–37	78 tuberculous infants 0–1 year ...	77·0 per cent
1938–45	46 tuberculous infants 0–1 year ...	34·7 ,, ,,
1933–37	115 tuberculous infants 0–3 years ...	60·7 per cent
1938–42	82 tuberculous infants 0–3 years ...	18·0 ,, ,,
1943–45	159 tuberculous infants 0–3 years ...	22·6 ,, ,,

After infancy, with a high death-rate in the 0–1 period, falling in the 1–3 period, comes a time running on into school age where there is less danger to life. From 6 to 12 years the death-rate is low, with hilar gland tuberculosis and lesions of bone and joint. At 12 years primary lesions are seen, both healed and active, but another danger arises—namely, the bronchogenic type of phthisis. For examples of primary tuberculosis in the school age period *see Table VI.* In *Table VII* are shown the total deaths from tuberculosis in various countries at different age periods, in the year 1944; of interest is the fact that B.C.G. vaccination has been used extensively in Denmark since 1940; note the low incidence of meningitis in that country.

Table VI.—Results of Treatment by Home Rest in Primary Tuberculosis

(*Price*, 1946)

	Tuberculosis Mortality	*Tuberculosis Morbidity*
1933–9—		
200 children 3–14 years with radiological evidence of primary tuberculosis		
1946—		
Reviewed	1·0 per cent	1·0 per cent

Table VII.—Total Deaths from Tuberculosis in One Year in Various Countries

Country	Age in Years					
	0–1	1–5	5–10	10–15	15–20	20–25
England and Wales (1942) Population 38,243,000						
Pulmonary	64	134	59	144	1530	2484
Central nervous system ...	188	686	287	198	238	87
Other forms	87	238	111	131	265	226
Scotland (1944) Population 4,653,645						
Pulmonary	32	49	19	53	326	504
Central nervous system ...	38	138	91	70	78	40
Other forms	18	58	31	36	57	57
Northern Ireland (1944) Population 1,314,000						
Pulmonary	7	8	5	17	79	133
Meningitis	23	33	26	17	21	9
Other forms	12	14	6	4	15	15
Eire (1944) Population 2,944,000						
Pulmonary	13	23	26	52	277	428
Meningitis	31	87	64	43	58	34
Other forms	14	60	24	25	60	69
Denmark (1944) Population 3,898,000						
Pulmonary	10	17	7	4	43	131
Meningitis	8	23	10	13	4	15
Other forms	4	4	6	3	7	13

Since infancy has been mentioned as the age of greatest danger, a few words must be devoted to the *infant*. It has already been shown that the highest incidence of intrafamilial infection occurs amongst infants and young children ; it is obvious that an infant can scarcely avoid contracting a lung-to-lung infection from a phthisical mother when it is remembered : (1) the low resistance of the infant to all infections ; (2) the fact that the infant is constantly in the mother's arms. Indeed, even a short exposure is very dangerous to an infant, for a single droplet is sufficient to infect it. Whether repeated infection from a phthisical mother to her infant makes the prognosis worse than from a single exposure, is debatable ; the answer probably is yes, if the infant gets repeated doses when in the pre-allergic state.

Example.—Joe O'D., age 8 weeks, died from toxæmia, and autopsy revealed a primary tuberculous cavity in the right lung. He had been exposed to infection from his phthisical mother for the first two weeks of life, after which he had been removed to a tubercle-free environment.

Example.—James M., twin, born in a maternity hospital, was lent by the nurses a few times to comfort a very sick woman in the same ward who had lost her baby. J. M. was admitted to St. Ultan's Hospital aged 16 days, and died 6 months later of disseminated tuberculosis, confirmed by autopsy. The other twin and the rest of the family were free from tuberculosis.

Sex.—In this book, statistics discriminating between the sexes are not employed. There is no particular sex incidence in childhood tuberculosis until puberty, at which age secondary and tertiary manifestations are definitely more commonly seen in girls.

Type of Bacillus.—The human type of tubercle bacillus plays by far the largest role in the causation of tuberculous disease in children. However, the bovine type is also of importance, especially amongst children under 5 years of age. Infection is carried in milk to the mouth, causing cervical gland tuberculosis, and to the intestinal tract, causing abdominal tuberculosis ; from either of these a secondary infection of bone, joint, or meninges may occur. The incidence of bovine infections is higher in rural districts than in cities (*see Table XIV*, p. 98). In some countries, such as Norway, and in large parts of Canada and the U.S.A., by slaughter of tuberculous cattle or by pasteurization of milk supplies to large cities, bovine infection has been practically eliminated. In England (1934) an average of 40 per cent of

cows were found to react positive to the tuberculin test, and were found to be infected during the inspection of carcases. Only a small proportion of these cows, however, yield tuberculous milk at all times—many only excrete tubercle bacilli in the milk at intervals; cows with advanced disease and tuberculous udders which give badly infected milk are becoming more rare. In London, 3·2 per cent of milk samples were found to be infected with tubercle bacilli, and 5 to 10 per cent of cows in England were found to yield tuberculous milk. In four cities in Scotland, samples of milk examined showed that 10 per cent were infected by the tubercle bacillus. In Ireland bacteriological examination of milks in Dublin by Bigger showed 8 per cent of samples to contain tubercle bacilli, and in Cork by Cussen (156 samples by guinea-pig inoculation) showed 6·4 per cent to contain tubercle bacilli. In Scotland there is a relatively high incidence of infection of the lung by the bovine type of bacillus. The same is found in Denmark, but the numbers are lessening owing to an intensive campaign to stamp out tuberculosis in cattle. *Table VIII* (taken from a table by Holm [1946]) shows the results of examinations in the State Serum Institute, Copenhagen, in typing the bacillus from pulmonary and extrapulmonary lesions in children.

Table VIII.—Relative Frequencies of Bovine Type Bacillus in Patients with Pulmonary and Extrapulmonary Forms of Tuberculosis; Data compiled for Patients living on Jan. 1, 1944 (*After Holm*, 1946)

Age Groups	Pulmonary Tuberculosis			Extrapulmonary Tuberculosis		
	Total Human and Bovine	Bovine		Total Human and Bovine	Bovine	
		Number	Per cent		Number	Per cent
Total all ages over 0 –over 70 ...	11,072	384	3·5	1,454	204	14·0
0– 4 years ...	473	18	3·8	91	31	34·1
5– 9 ,, ...	476	15	3·2	71	20	28·2
10–14 ,, ...	519	7	1·3	43	7	16·3
15–19 ,, ...	1,457	51	3·5	130	23	17·7
20–24 ,, ...	2,282	68	3·0	190	24	12·6

CHAPTER III

ROUTES OF ENTRY INTO THE BODY

THE tubercle bacillus may enter the body by the following routes; (1) Inhalation; (2) Ingestion; (3) Skin; (4) Mucous membrane: (5) Placenta and amniotic fluid.

1. Inhalation.—The most usual route of entry is by the air-passages, for inhalation infection is responsible for 80–90 per cent of all cases of childhood tuberculosis. The tubercle bacilli are inhaled and travel down the trachea to enter one or other of the main bronchi; they then pass along smaller bronchi to reach a bronchiole, and finally lodge in an air-cell or alveolus. At the site of implantation in the lung parenchyma, a primary focus is formed, the contents of which are drained by the lymphatic vessel to the regional gland at the hilum of the lung, causing infection of the gland. The bacilli which are inhaled in this manner come from one of two sources: either from fine dust particles which contain dried but living tubercle bacilli, or from moist bronchial droplets coughed out by a phthisical person, and containing living tubercle bacilli.

Figures showing the frequency of primary pulmonary infection in these countries are usually reported from post-mortem material. Autopsy figures, however, do not give a true picture of the incidence of infection as seen by the clinician. Ghon (1912) found it in 92·4 per cent of 184 children at autopsy. Blacklock (1932) found 61 per cent of pulmonary cases in 283 autopsies on tuberculous children. MacGregor (1937) found 60 per cent amongst 233 autopsies on children. These figures from Scotland are low as compared with other countries; possibly this is due to the high incidence of bovine and abdominal lesions in Scotland. Neale (1933) reports 71·6 per cent primary lung infections amongst 300 necropsies on tuberculous children in Birmingham. Price found 78·5 per cent primary lung lesions at autopsy in 28 infants under 2 years in Dublin, and 75 per cent amongst 450 clinically tuberculous children, aged 0–14 years.

a. Dried Dust.—Many observers hold that dried dust is the most frequent source of aerogenous tubercle infection in children.

It has, on the other hand, been suggested that in damp climates the droplet is greater than the dried dust mode of infection. Years ago in Vienna, Cornet examined dust from the floors of houses where lived a phthisical adult, and he found tubercle bacilli present in 30 per cent of the sweepings ; these bacilli remained alive and virulent for from three to four months. He also examined carpets from such houses, and in 35 out of 36 he found tubercle bacilli. More recent investigations have confirmed the accuracy of his findings, and in Paris tubercle bacilli have been demonstrated in the sweepings of public vehicles. Thus it may be seen that children run a risk of inhaling tubercle bacilli in the form of infected particles of dried dust which are stirred up by floor sweeping.

b. Direct Droplet.—Infected droplets may be expelled by a phthisical person from the mouth in speaking, and from a bronchus by coughing. The spoken droplets are usually merely salival, and are less often infected by tubercle than are the bronchial droplets. Cough droplets, although smaller than mouth droplets, contain many more tubercle bacilli—in the large ones several and in the smaller only one or two bacilli. At birth a human air-cell measures about 50μ, and in childhood from 100μ to 200μ. Tests have been made to show that coughed droplets will carry for nearly a yard before falling to the ground. Some of the larger cough droplets may contain depots of bacilli, and some observers are of the opinion that if a heavily laden droplet lodges in a child's air-space, serious results ensue. Others believe that only small droplets containing one or two bacilli can lodge in a child's lung. Tubercle bacilli, which are $2–4\mu$ in length, can live in these droplets for many days.

2. Ingestion.—Ingestion as a mode of entry is second in importance to inhalation. It is responsible for primary abdominal and cervical gland tuberculosis. Here the tubercle bacillus lodges and forms a minute focus in the mucous membrane of the wall of the small intestine, and penetrates the wall, with or without leaving an ulcer. The favourite site is in the region of Peyer's patches, immediately above the ileo-cæcal junction. The corresponding mesenteric gland becomes infected by lymphatic drainage, and thus the abdominal primary complex is formed. Tubercle bacilli which are ingested are conveyed in two ways: they are contained either in infected food, usually milk, or in particles of dirt or dust which are mixed with saliva in the child's mouth.

The incidence of abdominal tuberculosis, according to statistics, varies in different countries, as well as in different parts of the

same country. The influence of rural and urban domicile plays a very important role, as does also the nature of the milk supply. Ghon found in Vienna 2 cases of abdominal tuberculosis amongst 500 tuberculous children. In Sweden to-day the incidence is about 2 per cent of tuberculous children. In Germany 5–10 per cent of tuberculous infections are primarily abdominal. In Scotland, Blacklock found amongst 283 tuberculous cases at autopsy that 35·7 per cent were abdominal infections; and MacGregor found 36 per cent amongst 233 cases at autopsy. In England, Neale found 15·3 per cent primary abdominal infections amongst 300 fatal tuberculous cases. In Ireland, Price found 5 per cent primary abdominal infections amongst 450 clinical cases of tuberculosis (0–14 years).

a. Milk.—It has not yet been proven that tuberculosis can be conveyed through the medium of human milk. The ingestion of milk from an infected cow (or goat) is the most common cause of primary abdominal tuberculosis. Infection can also be caused by ingestion of cream and butter made from tubercle-infected milk. De Grolier (1939) reports tubercle bacilli found in 4 out of 22 samples of cream in Paris. Primary abdominal bovine infection occurs most frequently during the first five years of life. Blacklock found amongst 101 children suffering from primary abdominal tuberculosis that 26 were under 1 year, and 29 between 1 and 2 years. In Eire in the year 1944, 7 children died of abdominal tuberculosis under 1 year, 10 aged 1 to 2 years, and 14 between 2 and 5 years. The highest incidence is between 1 and 3 years; where breast feeding is prolonged, and in cities where welfare schemes provide free clean milk to infants, the incidence during the first year is low.

b. Dirt and Dust.—It has already been shown that in the dwellings of phthisical persons, floor dust can provide infection to small children; this is not only by inhalation but also by ingestion, for at the crawling age bacilli are transferred from fingers to mouth and swallowed in the saliva. Puzzling cases of abdominal infection in children with human organisms can occur in these ways. The habit of blaming the milk supply in all cases of primary abdominal tuberculosis may need reconsideration when figures on a large scale are available to demonstrate the type of bacillus recovered from small children. Phthisical persons wetting teats and soothers in their own mouths before giving them to infants may cause human abdominal infection.

3. Skin.—The intact skin is not permeable to the tubercle bacillus. Two or three rare cases have been reported in which infection took place through the dermis after prolonged friction against an infected surface. For practical purposes this route of entry may be disregarded, but the possibility must be borne in mind, for it has been demonstrated that under certain conditions the skin of a guinea-pig is permeable. If a primary skin infection takes place it must be through an already existing wound; this is not found in children, although it occurs from time to time amongst laboratory and slaughter-house workers. Should it occur, a small abscess would form two to four weeks later at the site of the wound, and the regional lymph-gland would be involved. Infection during the rite of circumcision has been reported in authentic cases.

4. Mucous Membrane.—Entry through the intestinal mucous membrane has already been considered; other mucous membranes where entry in rather rare instances may occur are nose, throat, middle ear, conjunctiva, and vulva.

Nose, Mouth, and Throat.—Entry is rare, except in the tonsil; of this a certain percentage of cases are authentically reported. Oral infections are caused by the ingestion of tuberculous milk or particles of infected dirt or dust. The middle ear in infants is occasionally the site of a primary infection, the tubercle bacilli reaching it from the mouth cavity. The difficulty of oral infection is that the primary focus is seldom visible macroscopically; diagnosis has to be made by the enlargement of the regional glands. The nose is seldom affected. Finkelstein reported one case where a phthisical mother infected her infant by cleaning aphthæ off its mouth with her finger. Primary tonsillar or adenoidal is the most usual type of mouth infection. The tonsil may appear normal to the naked eye, and on section after tonsillectomy the primary focus may not be found; the nature of the infection in these cases is indicated by the involvement of the tonsillar gland, and the upper nodes of the deeper cervical glands. MacGregor in Scotland found primary cervical gland infection in 4 per cent of 233 fatal cases; Blacklock found it in 2·1 per cent of 283 autopsies on tuberculous children, and in only 1 of these was the tuberculous lesion demonstrated (human type). Ghon found 2 cases of primary infection of the tonsil amongst 2114 tuberculous children. Price, amongst 450 clinically tuberculous children, found only 1·1 per cent that appeared beyond doubt to be a primary oral infection. Only at autopsy

can the primary infection of tuberculous cervical glands be relegated beyond dispute to the mouth. Clinically, unless there is an obvious intra-oral focus, one can never entirely exclude the possibility of a primary focus in some other part of the body, with secondary blood-borne metastasis in the cervical glands. Entry has been reported in the gums around a carious tooth; this is of less frequent occurrence than is often suggested.

Conjunctiva.—Primary conjunctival infection by the tubercle bacillus in children is very rare. It occurs in cases of trauma, with coincident or subsequent infection. This may be conveyed by a flick of a cow's tail across the eye, or other injury. In these cases the pre-auricular glands on the affected side are enlarged.

Vulva.—Infection of the vulva is extremely rare, but it has been known to occur in young children from a tubercle-infected floor. The vulval ulcer is usually found only after attention has been directed to the resulting enlargement of the inguinal glands, which is generally quite marked. Nordenskjold (1937) reported one case associated with erythema nodosum, and in so doing surveyed the literature; she found reports of 10 authentic cases. She considered that in extra-pulmonary primary tuberculosis, infections of the skin and superficial mucous membrane account for 0·14 per cent of cases.

5. Placental Transmission and Amniotic Fluid.—Less than 100 cases of intra-uterine and intrapartum infection have been reported in an authentic manner. The infection in these cases is derived either from a placental tubercle, which discharges tubercle bacilli via the umbilical vein to primary lodgment in the fœtal liver, or by ingestion of infected amniotic fluid, with primary lodgment in the intestinal mucosa of the fœtus. (*See* Chap. VI, p. 41).

Lymphatics.—A word must be said about this one-time much discussed mode of entry. All observers are now agreed that there is no primary entry into a lymphatic gland; the involvement of glands is always secondary to a primary focus which must lie in the area drained by that gland. Calmette formulated a 'lymphatic theory', postulating that all lung lesions were secondary to primary lesions in the mesenteric glands or tonsils. This theory is no longer accepted, although it is recognized that cases of primary tonsillar infection may occur; it is also known that the spread from abdominal mesenteric glands to mediastinal glands is of very rare occurrence and only seen in very heavily infected cases; in the Lübeck disaster a few such cases were seen.

CHAPTER IV

DIAGNOSTIC TUBERCULIN TESTS

Rationale.—The rationale of the tuberculin skin test for diagnostic purposes rests on a basis of laboratory and clinical investigation which has been carried on in many countries during the past forty years. Robert Koch discovered the tubercle bacillus in 1882, and later he thought that the tuberculin which he prepared would be a strong poison for a tuberculous and a weak poison for a non-tuberculous subject. He described three types of tuberculin reaction : general, focal, and local. In the general reaction the tuberculin is in the circulation, and a toxic febrile reaction begins after a few hours. In the focal the toxicity closely resembles that of the general, but in addition an inflammatory reaction in the original tuberculous lesion sometimes leads to necrosis. The local reaction shows some inflammation at the site of inoculation, without any general or focal disturbance. In 1907 von Pirquet discovered the ' phenomenon of allergy '. He found that a person previously infected by avirulent tubercle bacilli who was then injected with tuberculin gave a specific tissue reaction at the site of injection. Thus von Pirquet demonstrated that tuberculin sensitivity existed as the result of a true tuberculous infection, such as can only be produced by an infection with living virulent or avirulent tubercle bacilli. According, then, to the originator of the term, ' tuberculin allergy ' means that the individual has experienced a specific body change through infection with *living* tubercle bacilli, and has in consequence a capacity to respond to tuberculin irritation with specific tissue formation. As a result of his observations, von Pirquet devised for diagnostic purposes a method of cutaneous application of tuberculin, which produced a local reaction only. Since 1907, the local type of reaction only is employed for diagnostic purposes, and the focal and general types have been discarded as dangerous.

Tuberculin.—Tuberculin is a product derived from the tubercle bacillus. There are many of these tuberculins, which differ quantitatively rather than qualitatively. *Old tuberculin* (*O.T.*)

was the first tuberculin and it was produced by Robert Koch in 1890. It is a culture of killed tubercle bacilli, filtered to remove the dead bacilli; it still remains the standard for diagnostic tuberculin. In the U.S.A., and also in England and Denmark, another tuberculin diagnostic has been produced in which extraneous proteins have been eliminated (*Purified protein derivative*, *Purified tuberculo-protein*). It has been found that all these tuberculins hold good as tests for both human and bovine types of infecting bacillus. Controls for the cutaneous and intracutaneous tests are seldom now employed, because protein pseudo-reactions have been reduced to a minimum by improved methods in the preparation of tuberculin.

Methods of Application.—

a. Cutaneous Test (*von Pirquet*).—This test is performed by placing two drops of old tuberculin on the skin of the arm, and then scarifying the area with a vaccinator or needle. In from 24 to 48 hours a positive result is shown by the appearance of inflammation around the wound. The diameter of this so-called ' Pirquet's papule ' is measured in millimetres, 7 mm. being positive. This cutaneous method has been given up generally in favour of percutaneous and intracutaneous tests, because of its large percentage of error, that is, failure to respond in positive cases. Galtung Hansen (1944) reports that its action is improved by the addition of 1 drop of 1 per cent solution of adrenaline to 1 c.c. of old tuberculin. Alston's (1946) comparison of these two von Pirquet tests showed little difference (*see Table X*).

b. Intracutaneous Tests (*Mantoux; Purified Protein Derivative*).—

Mantoux Test: Mantoux first published his test in 1908. It is performed by injecting intracutaneously 0·1 c.c. of freshly diluted old tuberculin in varying strengths. The injection is made into the flexor surface of the forearm, and it must be definitely intracutaneous, as demonstrated by the raising of a small white weal at the time of injection. The operator can see best where the needle is going if the bevel is kept turned upwards. The result is read in 48 to 72 hours, but inflammation round a positive reaction will remain for more than a week. The response is positive when an inflammatory area of infiltration surrounded by hyperæmia appears at the site of injection, remains for at least 48 hours, and the infiltration measures not less than 7 mm. across. A slight transitory reaction, due either

to non-specific protein or to trauma, may appear and disappear again in 48 hours. Incorrect interpretations will be avoided if the readings are taken after 72 hours ; this is convenient because, in negative cases, 72 hours after the first test is the time of choice for further testing with a higher dilution. The strengths which are used with old tuberculin are 1–10,000 (0·01 mg.), 1–1000 (0·1 mg.), 1–100 (1 mg.).

It is essential to remember never to employ a dilution of 1–100 (1 mg.) for the first test ; a strong local reaction with transitory pyrexia may ensue. If the first test of 1–10,000 is negative on the third day, it should be followed with 1–1000 ; if again negative, by 1–100. When a positive reaction is observed, it is not necessary to test any further ; indeed the mistake of further tests with stronger doses may lead to severe local reactions in positive cases. It is claimed that the Mantoux is the most sensitive of all tests ; it is true that Mantoux 1–100 will reveal cases still in the incubation stage, and thus allergy may be demonstrated some days earlier than with any other test. Alston found that a diluted intradermal test had a sensitizing effect ; she tested 101 boys with von Pirquet test after a dose of Mantoux 1–1000, and found that the number of Pirquet positives equalled the Mantoux positives, a result not obtained on using the Pirquet as a first test. The Mantoux test is certainly the most reliable for the exclusion of tuberculosis, on account of the high strength of tuberculin which may be reached. To carry this test to its logical conclusion necessitates three injections which will take nine days to complete. It is universally accepted that a negative to freshly diluted 1–100 dilution of Mantoux test means that the individual has not been infected by the tubercle bacillus. A rare exception is ' terminal anergy ' (*see*, ' Lowered Allergy ', pp. 27, 28). Care must be taken to ensure that the testing dilutions are freshly prepared and have not lost potency. Parish and Okell (1938) and Kayne (1938) found that dilutions of O.T. remained stable for 9 weeks in an ice chest but began to deteriorate after 6 weeks at room temperature. Holm (1934) in Copenhagen summarized the results of his investigations, as shown in *Table IX*.

Purified Protein Derivative (*P.P.D.*) : This form of tuberculin was first prepared by Seibert (1926–34) in the U.S.A. The bacilli are grown on a synthetic protein-free medium, lessening the tuberculo-protein and thereby reducing the risk of pseudo-reaction. This test is administered, like the Mantoux, by the

intracutaneous route, and a positive, read in 48 hours, shows an infiltration of not less than 7 mm. in diameter. The advantage of this test, in addition to protein reduction, is that it is made up as a commercial production in tablet form ; the tablets retain their potency for three years, but once dissolved the solution

Table IX.—Holm's Results as to the Stability of Old Tuberculin in Various Dilutions

Dilutions 1 in 10,000	after 14 days lost in strength	20 per cent.
	after 1 month ,, ,, ,,	40 per cent.
	after 2½ months ,, ,, ,,	60 per cent.
Dilutions 1 in 1000	after 1 month ,, ,, ,,	20 per cent.
	after 2½ months ,, ,, ,,	45 per cent.
Dilutions 1 in 100	after 1 month ,, ,, ,,	15 per cent.
	after 2½ month ,, ,, ,,	40 per cent.
Dilutions 1 in 10	remained stable for several months.	

must be used within a few days. Amongst many strengths made, those in general use are 0·00002 mg. and 0·005 mg. The fact that these two strengths may be used without danger of severe reaction when compared with the three dilutions necessary in the Mantoux test is of practical advantage. A standard dose of 100 tuberculin units equals 1 mg. O.T. (Mantoux 1–100 and P.P.D. 0·002 mg.). For further details the reader is referred to the work of Parish et al. and Jensen et al.

c. Percutaneous Tests (Moro; Hamburger; Plaster; Vollmer; Patch).—As a first test the percutaneous has many advantages. Chief amongst these is that it will catch the hypersensitive case. Further, its painlessness will gain a child's confidence at first examination, thus ensuring a return for further testing or treatment. The ointment form of tuberculin does not deteriorate, and in a tube keeps fresh for a year. It is a safe test where tuberculides or abscesses are present ; it avoids sterilization of instruments, and the tube may be carried in the pocket ready for use. Contra-indication for the percutaneous test is skin rashes, such as acne or scabies, where difficulties in interpretation will arise.

Moro Test : Moro first used his test in 1907. It then consisted of equal parts of old tuberculin and lanoline ; the ointment is rubbed into the skin over the sternum. A positive result was indicated by the appearance of a definite folliculitis at the site. Further modifications have improved this test as a diagnostic agent.

The Edinburgh Moro ointment of Sir Robert Philip (obtainable from A. K. Stewart, West Maitland St., Edinburgh) was the same as Moro's original test. Price found this ointment too weak, but when made up, not in equal parts, but in a strength of 75 per cent O.T. to 25 per cent lanoline, it is a good test.

The Dublin Moro ointment originated by Price (1946) has proved a very satisfactory first test, as shown in *Table X*. (It is obtainable from the National Vaccine Institute, 80 Sandymount Road, Dublin). This ointment consists of 2 parts of O.T. and 1 part of wool fat (anhydrous lanoline). The application of the test is as follows : An area of skin over the sternum is cleansed with washing ether ; to this area is then applied a portion of the ointment, the size of a small green pea, which is rubbed in well for half a minute with the first finger ; the area may be conveniently limited and the skin stretched by placing the first two fingers of the left hand on the sternum, and by rubbing the ointment between them. The test is read in 48 hours, although it may show positive after 24 hours and remain for a week. Papules (folliculitis) must be present for a positive result ; mere reddening of the skin is not a positive reaction. In a few cases papules may be felt and not seen, or there may be only two or three papules ; in doubtful cases it is wiser to repeat the same test before proceeding to a stronger one. It is important to issue instructions not to wash the area until after the test has been read. Should this test yield a negative result, it is necessary to follow it with an intradermal injection of 0·1 c.c. of Mantoux 1–100 (1 mg.) for the exclusion of tuberculosis. It is unwise to rely on a negative response to any first test.

Hamburger Test : Hamburger's ointment, produced by Dr. Fresenius, Frankfurt on Main, was the best ointment for tuberculin testing ; it consisted of O.T. evaporated to constant weight and made up as a jelly. Application and interpretation of a positive result was the same as described for the Dublin Moro test. Hamburger's test should also be followed where negative by Mantoux 1–100 intracutaneously.

Plaster Test : Tuberculin containing O.T. and purified tuberculin, standardized at three times the international standard, is put up in tubes at the State Serum Institute in Copenhagen. One drop is laid (not rubbed) on the skin at one side of the sternum, and a square of sticking plaster the size of a postage stamp is laid over it, with a control square of plaster only on

the opposite side. After 24 hours the plasters are removed; after 72 hours a definite folliculitis on the tuberculin side indicates a positive reaction. The tuberculin used in this test is very potent and gives a rapid response. In 536 cases F. D. Hart found this test equal in reliability to Mantoux 1–1000.

Vollmer Patch Test: Vollmer (1937) devised this method for percutaneous application of dried O.T. in the States; it is known as the 'Patch' test and is now sold by Lederle as a commercial product. To the forearm, which has been previously cleansed with ether, is applied a strip of adhesive gauze tape on which there are three squares; the two end squares have been saturated with undiluted O.T., and then dried; the central square is a control of dried glycerin broth. The strip is removed after 48 hours and read after 72 hours. The reaction is positive when the two end squares show a slight reddening and folliculitis, whilst the central control is negative. The patch is reliable only when used in a fresh condition; it tends to deteriorate if kept too long or in a damp place. F. D. Hart (1938) found amongst 536 children under 16 years that the Vollmer Patch test was equal in reliability to the Plaster test and Mantoux 1–1000 dilution. This was not Alston's experience (*see Table X* —the Vollmer Patch test is Dry Patch X). Any test which professes to distinguish between human and bovine infection may prove utterly misleading; the Vollmer Patch test does not profess to do so.

Choice of Test.—The choice of test is a matter of importance to the physician; he may employ whatever test or tests he is familiar with, provided he finishes with 0·1 c.c. intradermal Mantoux 1–100 dilution, that is 1 mg. of O.T. A recent survey by Alston affords information as to the efficacy of some of the tests at present employed (*see Table X*).

Whereas it is true that in epidemiological surveys and in hospital practice three strengths of tuberculin may be employed conveniently, yet the practising physician is advised to choose two reliable tests. It has already been stressed that amongst children there are advantages in commencing with a percutaneous test. Furthermore, if two tests only are to be employed then the first should be sufficiently potent to eliminate (by positive result) all cases which are hypersensitive to tuberculin; it is not unknown to meet with an unpleasantly brisk response to Mantoux 1–100 where the first test has been too inactive to produce a positive reaction in an individual who

was in fact sensitive. One must have fresh and reliable testing material. In the hands of the author, elimination of the hypersensitive individual has been achieved by correct employment of the Dublin Moro ointment as a first test, with Mantoux 1–100 as the second. No percutaneous or other first test should be relied on as a single test, but must always be followed by Mantoux 1–100 intradermally. This final test is necessary for the exclusion of tuberculosis and tuberculous infection. A good percutaneous test appears to be capable of picking out most primary infections of recent date; but there always remains the exception, especially the advanced tuberculous lesion.

Table X.—Comparison of Various Tuberculin Tests (*Alston*, 1946)

Boys—6 to 16 Years

First Test	No. of Cases	Percentage Positive to First Test	Percentage Positive to Mantoux 1–1000 (All Tested)	Percentage Positive to Mantoux 1–100. Only Negatives to 1–1000 Tested
Dublin Moro ...	201	43·0	55·5	61·0
Dry patch X	102	30·0	51·0	56·0
,, ,, Y		5·0		
Jelly Patch	102	26·0	44·0	53·0
v. Pirquet	101	26·0	48·0	51·0
,, c. adrenaline		31·0		
Mantoux 1–10,000 ...	203	29·0	46·5*	55·0

* Negatives only tested.

Amongst a group of 79 children under 6 years who had been exposed to infection, Price found 36 to be positive to Dublin Moro test; further testing of negatives with Mantoux 1–100 revealed one negative to Moro but positive to Mantoux 1–100, and suffering from widespread miliary disease, although walking about; this case would have been missed had only one test been employed. On testing, and even on retesting after an interval of time, it is wise to perform two strengths of tests as a routine on every occasion.

Lowered Allergy.—A lowering of allergy, as evidenced by a corresponding reduction in the response to tuberculin, occurs in certain conditions. It is important to remember this fact, otherwise the clinician will blame the test for "not always working".

1. The most striking example of lowered allergy is that of 'terminal anergy'. This occurs in a terminal phase, in advanced miliary or generalized or pulmonary tuberculosis, or in the last week of tuberculous meningitis. In such cases the child may fail to react to all weak strengths of test, may react only to Mantoux 1–100, or indeed at the end fail to react at all; in the latter event the tubercle bacillus may be demonstrated in the sputum, laryngeal smear, gastric washings, or cerebrospinal fluid.

2. A partial lowering of allergy tends to occur in certain infectious diseases, such as whooping-cough, measles, even pneumonia. The author finds this lowering to be less marked than is reported by some authorities. Suspected cases which react negative during such infections, should be retested a couple of weeks later.

3. The tuberculin response is not always easy to elicit in children who are much debilitated by any illness, extremely anæmic, mentally deficient, or subthyroid.

General Remarks.—Primary tuberculous infection occurs at some time in the life history of every individual. In most countries nowadays less than half the population have been infected at 14 years. It is possible by recognizing the change from tuberculin-negative to tuberculin-positive ('to invert', as it is termed in Scandinavia, or 'Mantoux conversion', as it is termed in England) to assess the moment of primary infection in an individual. For this, repeated tuberculin testing of negative reactors is necessary. Children and adolescents should be tested annually as a routine, and again in the interval should there be any suspicious rise of temperature or unexplained feeling of ill health. Full examination should follow in any case found to react positive for the first time. Just as it is now essential to know whether an individual has been vaccinated against small-pox or immunized against diphtheria, so should knowledge of his tuberculin reaction become part of a child's equipment. In the case of the tuberculin test, knowledge of the reaction will have to be kept up to date, for a negative will at some time become positive. It is only when constant routine testing becomes a universal practice that the bugbear of tuberculosis, with its three types of lesion, will be dealt with in a rational manner—before, during, and after school age.

That a positive reactor remains positive for the remainder of life's span has been assumed from experience, and we have

worked on this assumption for a number of years and must continue so to work. Children vaccinated with B.C.G., however, after a lapse of time may become once again negative reactors ('reversion', as it is called by the Scandinavians). Pathological reports from Terplan (1940) and from clinical workers suggest that after many years a naturally acquired primary complex may heal so completely that the individual loses his sensitivity to tuberculin, becoming once more a negative reactor; he may then be open to a fresh exogenous infection, and possibly may undergo a primary complex for the second time. Rich (1944) says that once a primary complex has made its appearance and has become arrested, it is infrequent that a subsequent one develops. The question of complete loss of sensitivity during childhood and adolescence scarcely arises, at any rate in white races; and it is at these early ages that we are concerned in the recognition of the tissue changes which accompany a primary complex.

The *intensity* of the tuberculin reaction bears no relation to the degree of disease present. An attempt to interpret such relationship may lead to serious mistakes. A child with a strong positive reaction may be found with no active disease, or perhaps a healed focus; another child with weak response to tuberculin may be in an active primary stage with pulmonary infiltration. The type of response depends on factors known and unknown; chief of the known are the freshness of the testing material and the correctness of its application. For no known reason individual sensitivity may in the same person vary from time to time. An exudative type of child generally responds briskly at all times; this type runs a high temperature for slight causes, is susceptible to all infectious diseases, is of the catarrhal type, and may be asthmatic. The intensity of the response to tuberculin, therefore, has no bearing on prognosis and gives no indication of the activity of the disease. Cases must be regarded simply as 'positive' or 'negative'.

Conclusions.—The tuberculin test is an essential diagnostic aid in the detection of all forms of tuberculosis in childhood. It is the only possible method of detecting symptomless primary tuberculosis. In children under 3 years a positive test strongly suggests active disease. In older children it indicates that further examination is necessary to decide whether the infection is active or healed. A large number of cases under 15 years of age are not healed. In the adolescent and young adult age

periods, tuberculin tests are year by year becoming more valuable as primary infection becomes more often postponed to these ages. Tuberculin testing will save the non-tuberculous child from a wrong diagnosis, where tuberculosis is suggested by clinical or radiological examination. It also detects the infected child at an early and symptomless stage, and may thus at times lead to the discovery of an unsuspected sputum-positive adult. The pædiatrician, child welfare officer, tuberculosis officer, and family physician should be in a position to observe the transition from negative to positive in all children under their care, by means of repeated tuberculin tests. A negative test, where at least two strengths have been employed, ending with 1 mg. of old tuberculin intradermally, excludes tuberculosis and tuberculous infection.

CHAPTER V

RADIOLOGY

General Remarks.—Radiology is a most important aid to diagnosis. Serial radiographs will tell more about the condition in a tuberculous child than will physical examination. Treatment must be guided to a very large extent by the radiological findings. In order, however, to make the best use of X-ray information, it is essential to know what deviations from the normal are to be expected in the various stages of pulmonary tuberculous lesions in children. Confusion results if one attempts to seek only for such changes as are seen in adult tuberculosis. At the same time, all interpretations of radiological abnormalities must be checked and guided by the result of the tuberculin test. It is only fair to the radiologist that information as to negative or positive tuberculin reaction be given by the clinician; if the X-ray appearances are suggestive of tuberculosis and the first test is negative, a second and stronger test must be applied. In certain cases radiological diagnosis alone is impossible; for example, between a primary tuberculous infiltration and an acute or unresolved pneumonia; so also are the interpretations of shadows in the hilar region, for whether they are due to tuberculous processes or to non-specific causes cannot always be revealed by the radiograph.

The normal radiological appearances of the thoracic contents in the child will now be considered, together with the most usual deviations seen in tuberculosis. In order to clarify the discussion, three typical age periods will be mentioned: namely, 1 year, 6 years, and 12 years. Technique depends so greatly on the type of plant used, that only a general remark can be made on the subject here. As infants will not breathe as directed, the time element is all-important. Deep inspiration is unusual in infants, but is increased by crying; so the film should be taken in one twenty-fifth to one-fiftieth of a second during full inspiration in the act of crying. For infants a full-sized film must be employed, but in children over 5 years satisfactory results have been reported with screening (Fitzpatrick, 1946) and with miniature radiography (Reynolds, 1946).

THE INFANT UNDER 1 YEAR

Normal.—In the normal infant (*Fig.* 1) the thorax is very short and considerably broader than it is long, owing to the high position of the diaphragm. The ribs are horizontal, and the heart shadow proportionately broadened. The mediastinum resembles a flexible tube or sac; the younger the child the more flexible the mediastinum. The broad heart shadow covers the hilum, so that any mediastinal glands which are seen outside the shadow of the heart must be enlarged; up to 7 or 8 years there is no well-defined hilum, and only fragmentary vascular shadows are seen. At 1 year the bifurcation of the trachea lies at the level of the 3rd or 4th thoracic vertebra. Infants are usually taken lying down, and this increases the broad appearance of the mediastinum, for the full abdomen pushes up the diaphragm still farther. To obtain immobility and a straight spine, the infant may be bandaged to padded splints, which are tied in the form of a cross.

Abnormal.—

1. *Infiltration.*—A primary infiltration, covering a primary focus, may be small or involve a whole lobe or part of a lobe (*Fig.* 2). This shadow is not very dense, but it is not always easy to distinguish it from an atelectasis (which remains longer) or a pleural exudation (which, however, is not very usual in infants), especially if the child is taken in the supine position; a film should be taken in the upright position and fluid looked for in the axilla; the shadow of fluid is usually dense. It is impossible to differentiate radiologically between the shadow of lobar pneumonia and that of a primary tuberculous infiltration (indeed lobar pneumonia may be superimposed in the same or opposite lung). The tuberculin test must be consulted.

2. *Primary Complex.*—When the perifocal infiltration is small or resolving around the primary focus, it may be seen in the lung field as a localized hazy shadow, and at the same time enlarged corresponding hilar glands will be seen; infants tend to show large infiltrations which may conceal these two separate entities. It is doubtful if an uncalcified primary focus is seen radiologically; it is most likely to be always the perifocal infiltration which casts a shadow. Primary cavities of all sizes may be seen in infants if central liquefaction occurs in the primary focus. Healing is frequently by fibrosis, or with so little calcification that it leaves no radiologically visible residue. Calcification will be seen appearing at a later date in

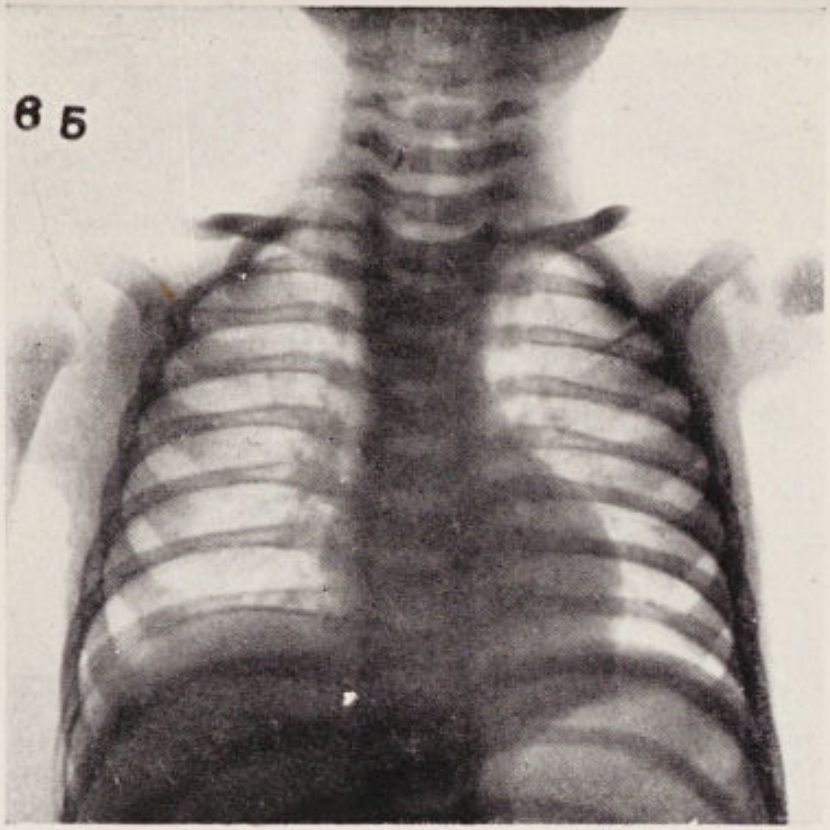
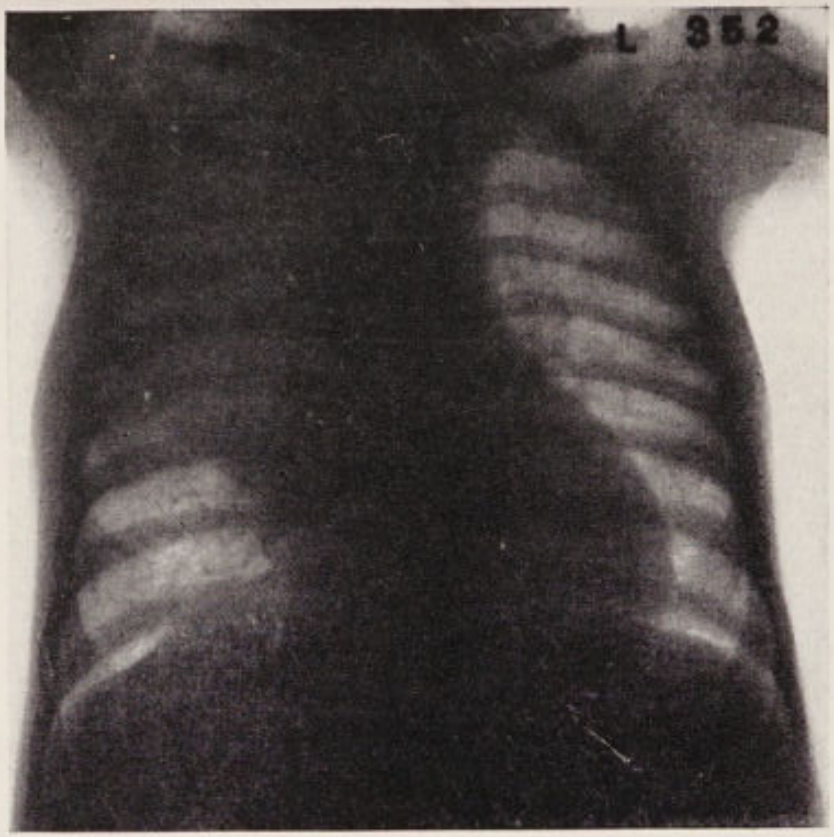


FIG. 1.—NORMAL CHEST, AT 7 WEEKS.



FIGS. 2A-2C.—PRIMARY INFILTRATION.

Fig. 2A.—Mollie N. April 16, 1935; aged 10 months. Mother dying of pulmonary tuberculosis. Hamburger positive. Shadow over right upper and mid lobes represents a primary infiltration (not atelectasis as trachea is not drawn over, and not caseous pneumonia as it had disappeared two months later, and the S.R. was 47 mm. per hour). During the next few months she developed multiple subcutaneous abscesses on limbs and sacral region.

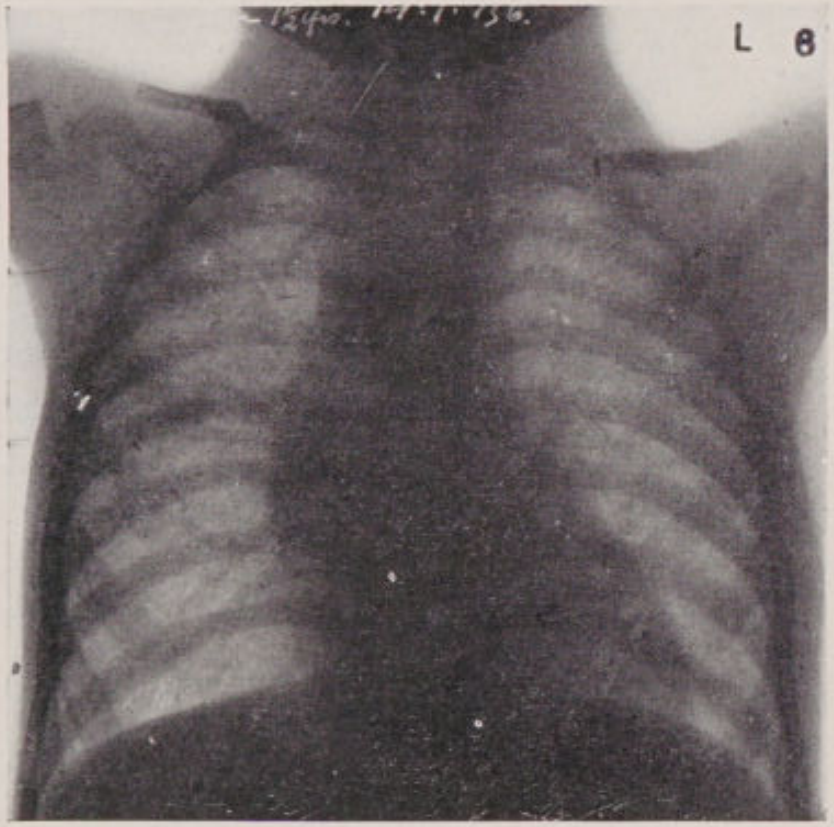


Fig. 2B.—Jan. 14, 1936; aged 1½ years. Right lung clear except for some residual fibrosis. General condition improved, still a few abscesses.

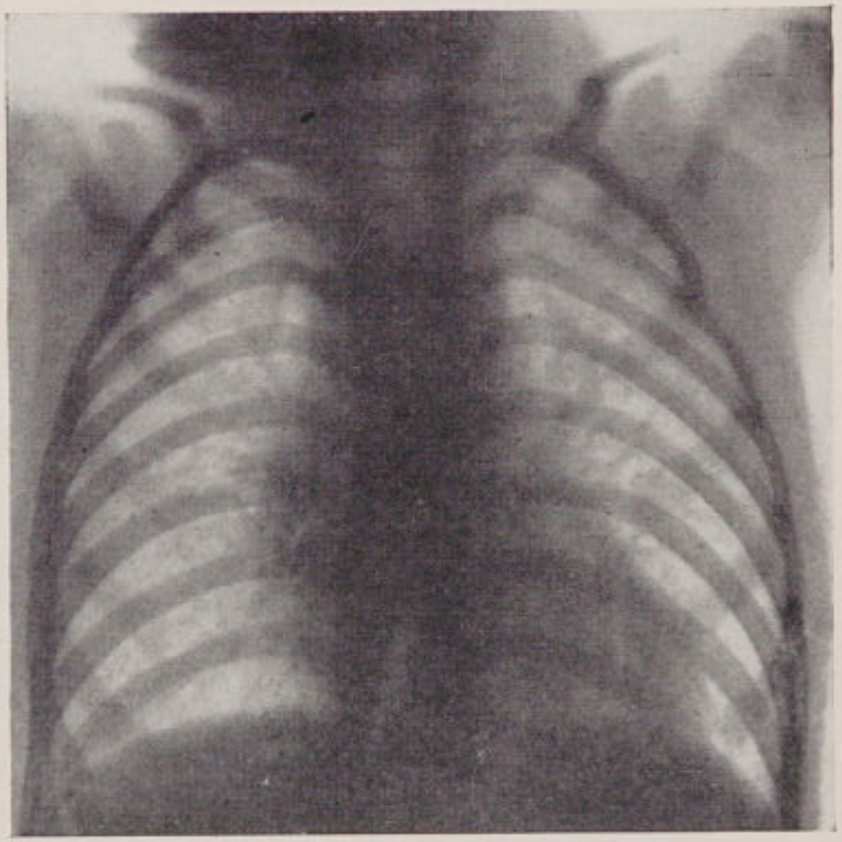


Fig. 2C.—Oct. 17, 1937; aged 3 years and 4 months. Lungs clear; osseous lesions and abscesses healed; sent to an orphanage. One year later chest radiograph the same.

Feb. 10, 1940; aged 5¾ years. Mors from generalized tuberculosis, with recurrence of skin abscesses; chest not X-rayed.

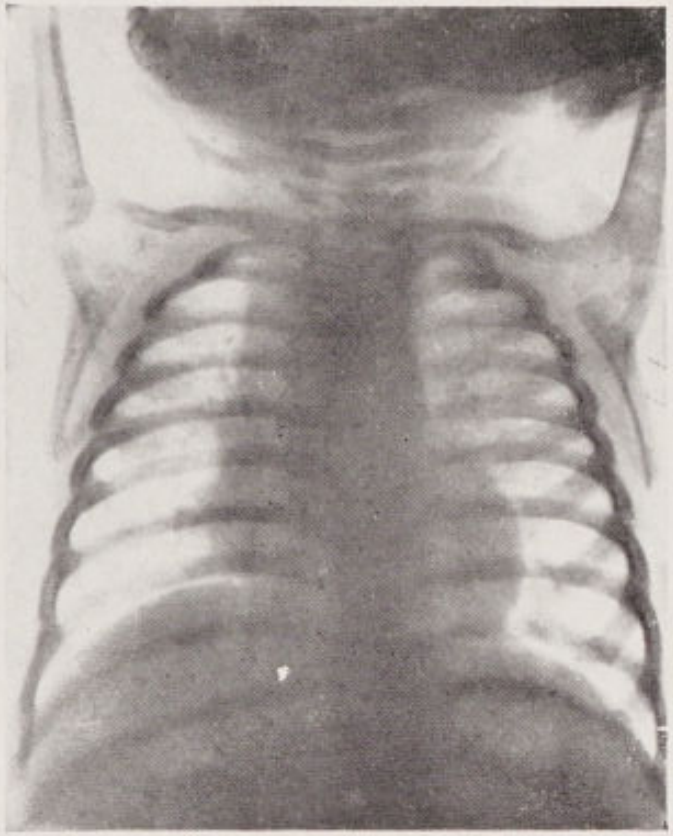
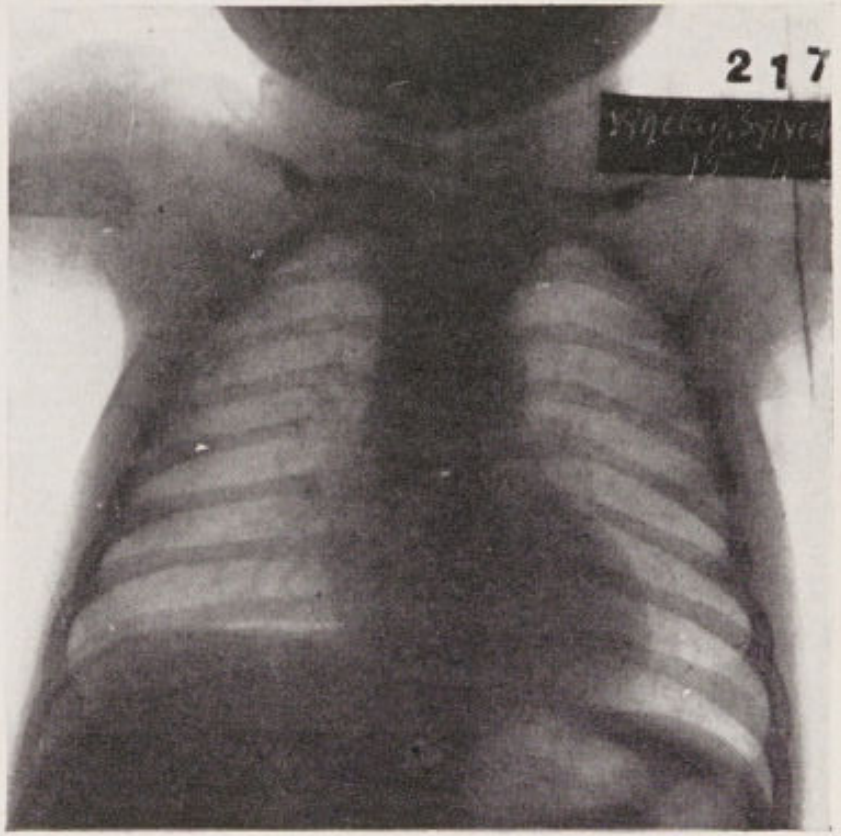


FIG 3.—'CHIMNEY-STACK' SHADOW (CONCEALING CASEOUS PARATRACHEAL GLANDS).

Con. T. Aged 9 months.



FIGS. 4A-4C.—PRIMARY COMPLEX FOLLOWED BY CASEOUS PNEUMONIA.

Fig. 4A.—Sylvester W. Nov. 15, 1938; aged 3 months. (*See Example*, p. 48.) Sister had pulmonary tuberculosis. Radiograph negative, Hamburger negative.

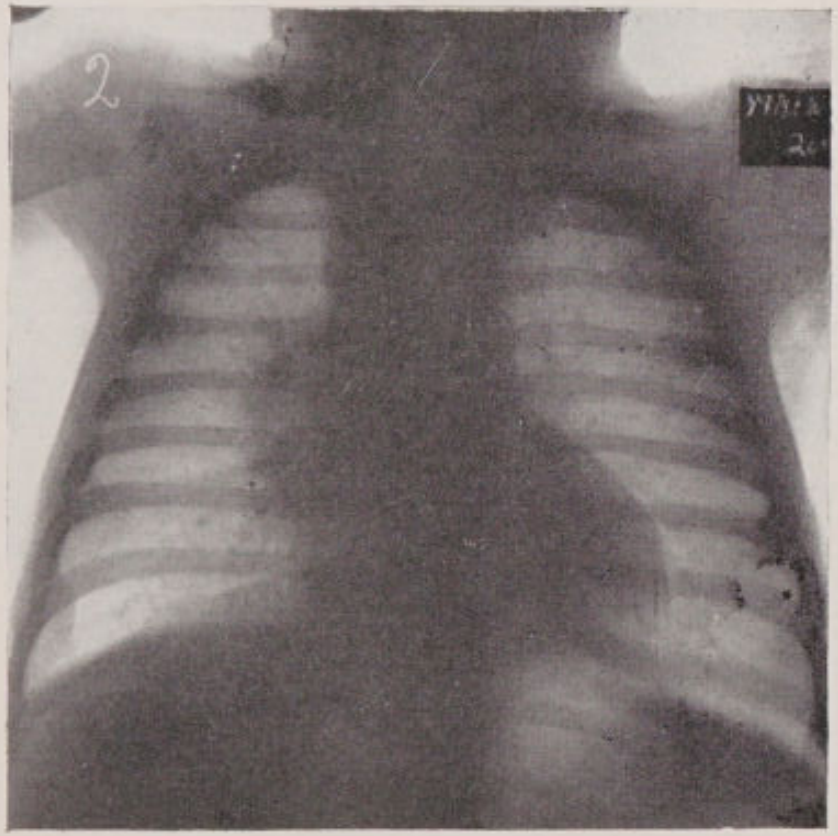


Fig. 4B.—Dec. 20, 1938 ; aged 4 months. Hamburger positive. Film taken during initial fever at the end of incubation period. Primary focus not seen as distinct entity, but suspected to be merged in the shadow at the right hilum, which is that of non-encapsulated caseous glands.

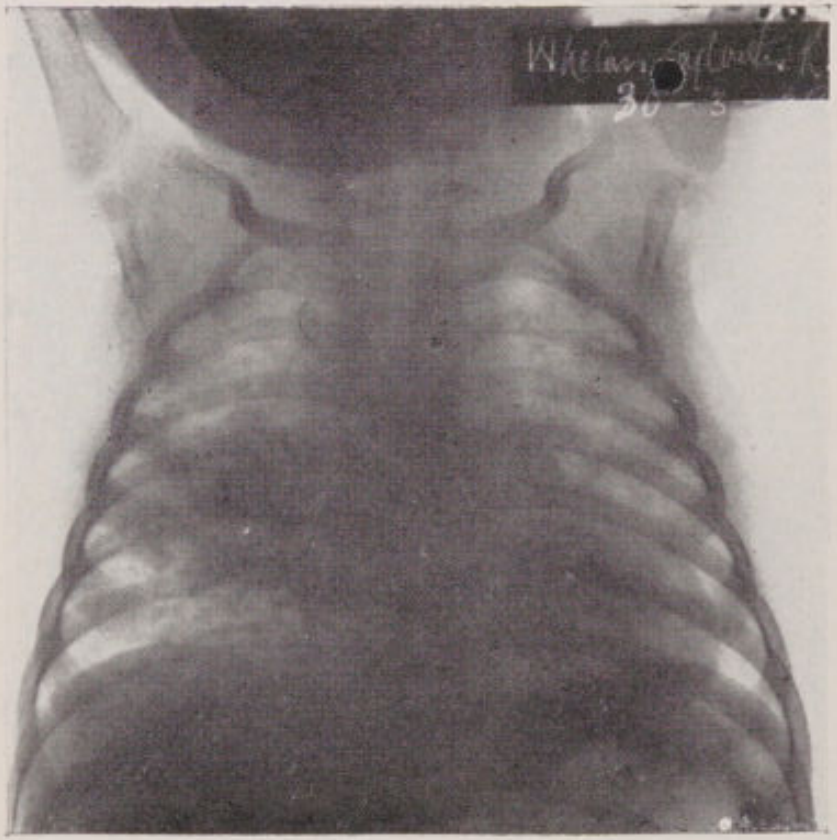
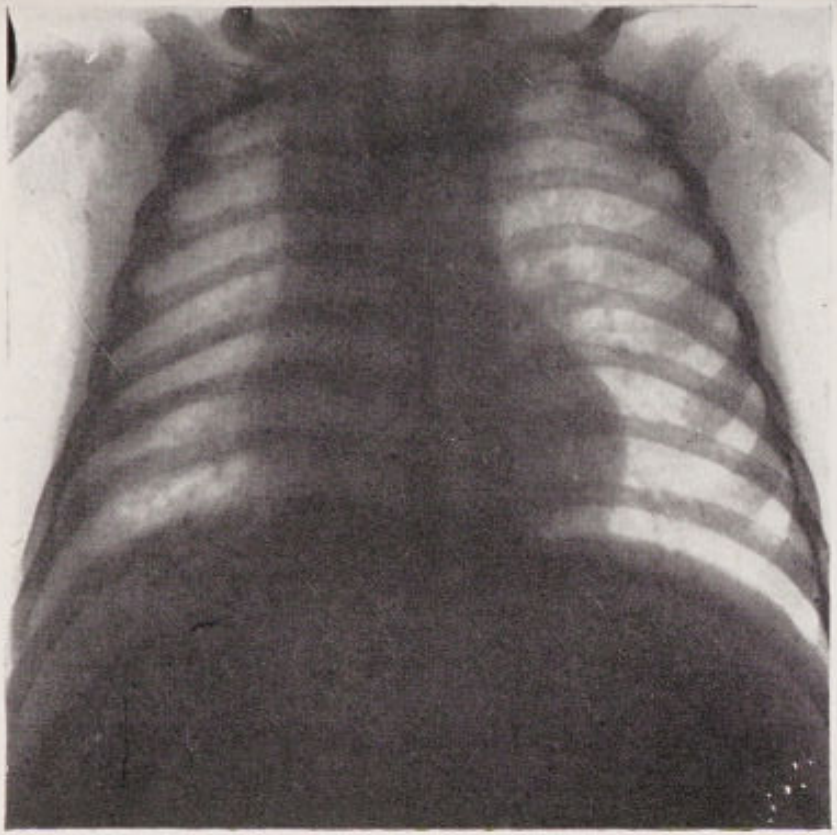
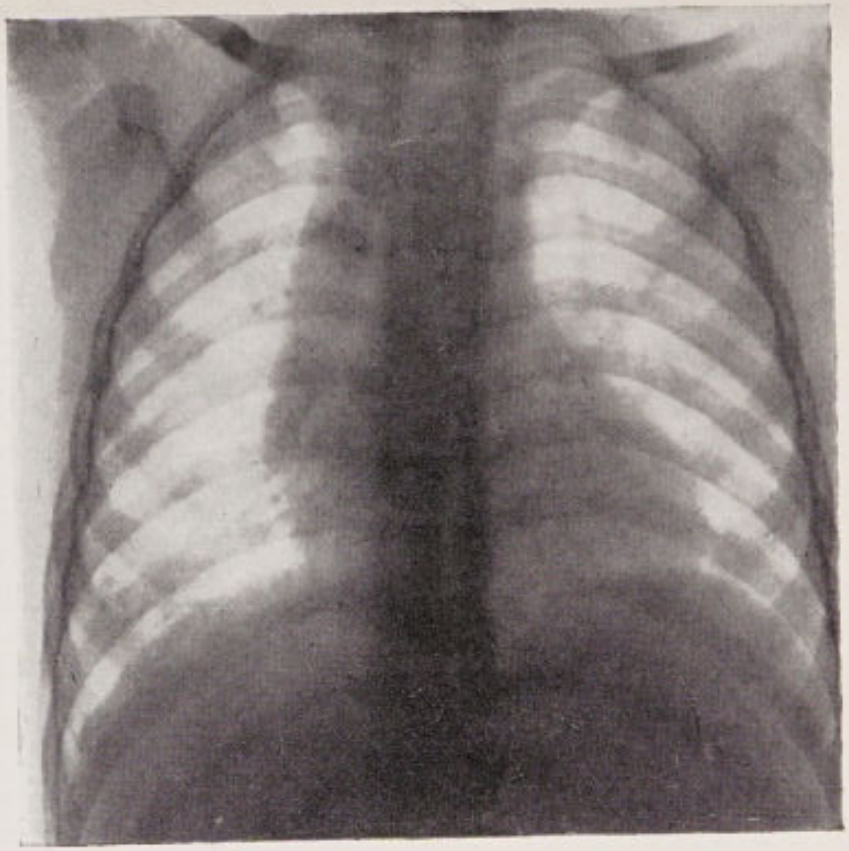


Fig. 4C.—March 30, 1939 ; aged 7 months (15 days before death). Caseous pneumonia in right mid zone ; whether due to liquefaction of primary focus or bronchogenic aspiration pneumonia cannot be decided from the radiograph ; clinically the former is more likely. Commencing spread to left upper zone.





FIGS. 5A-5B.—PRIMARY COMPLEX.

Fig. 5A.—Patricia C. Sept. 14, 1937; aged 5 months. Mantoux 1-100 positive. Primary focus at right base, small effusion in right axillary line, slightly convex ribbon shadow covers tuberculous paratracheal glands on the right side. (Mother died of pulmonary tuberculosis.)

Fig. 5B.—May 9, 1939; aged 2 years. After 11 months intern and 9 months extern treatment, calcification is seen at the right base and in the superior tracheo-bronchial and paratracheal glands.

1946. Child completely cured; dense calcification seen by X rays.

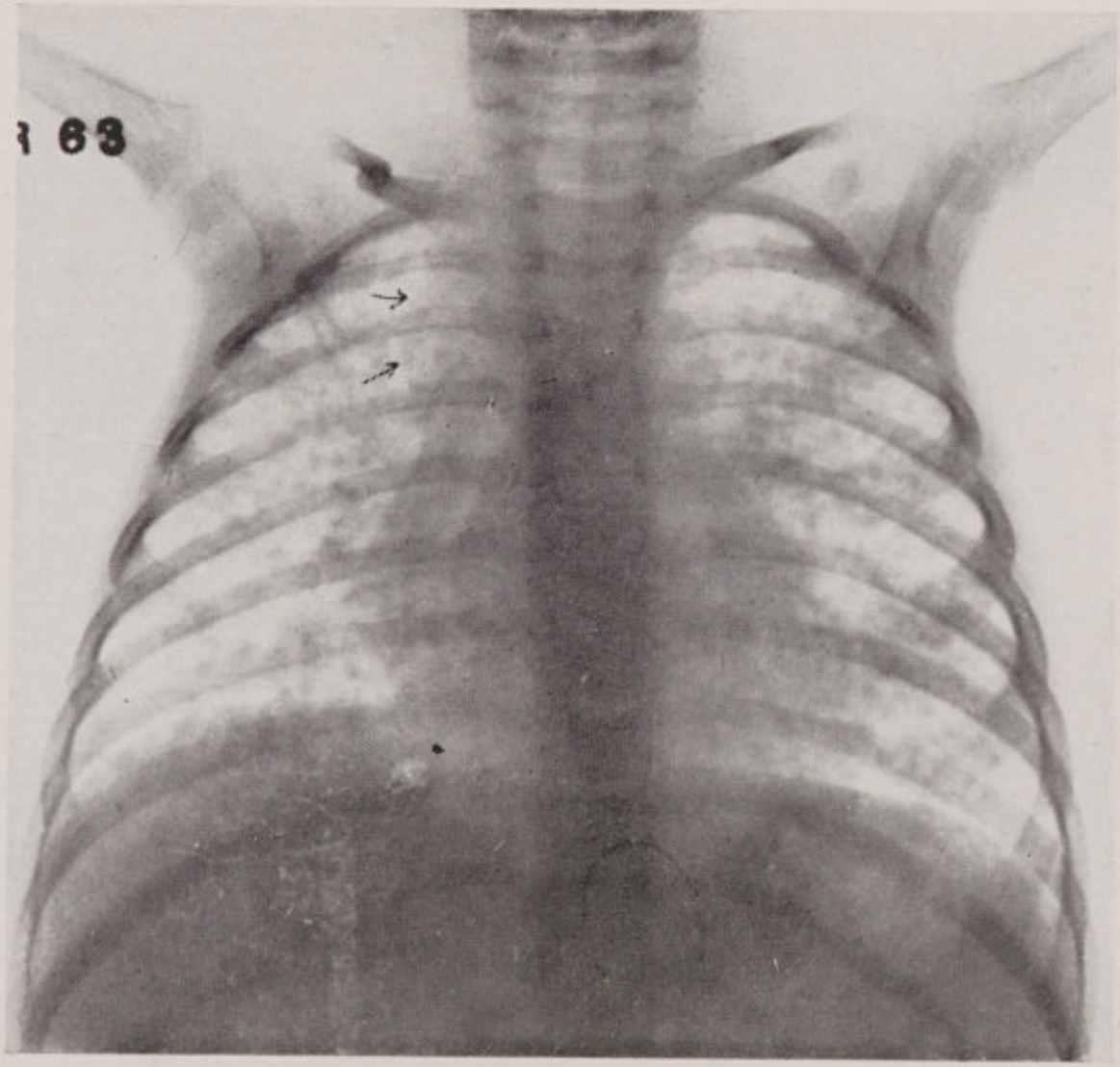


FIG. 6.—ACUTE MILIARY TUBERCULOSIS.

Theresa H. Aged 1 year and 8 months. Bilateral miliary involvement; right hilar and paratracheal glands (arrows) are enlarged. Died 2 months later.

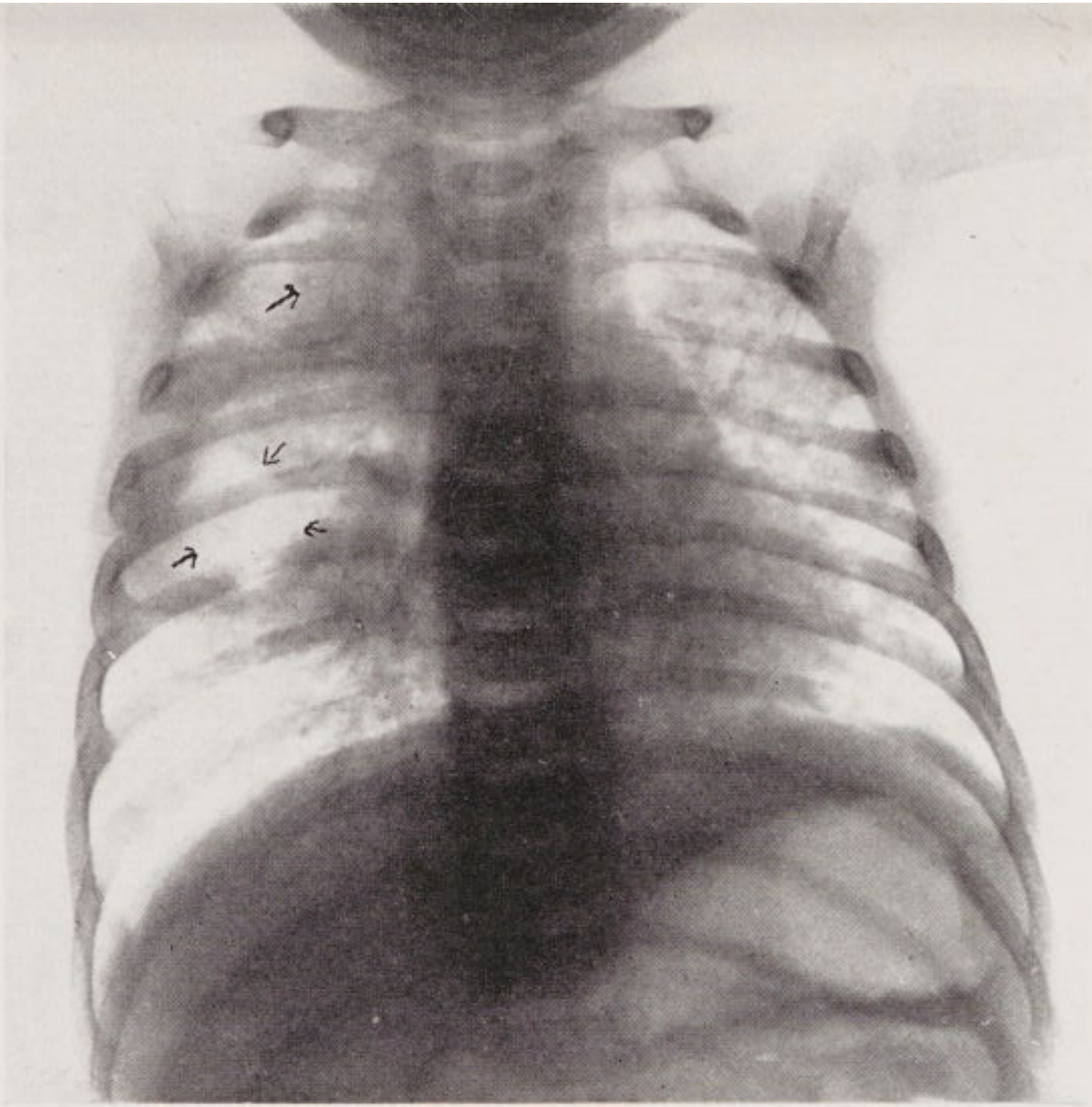
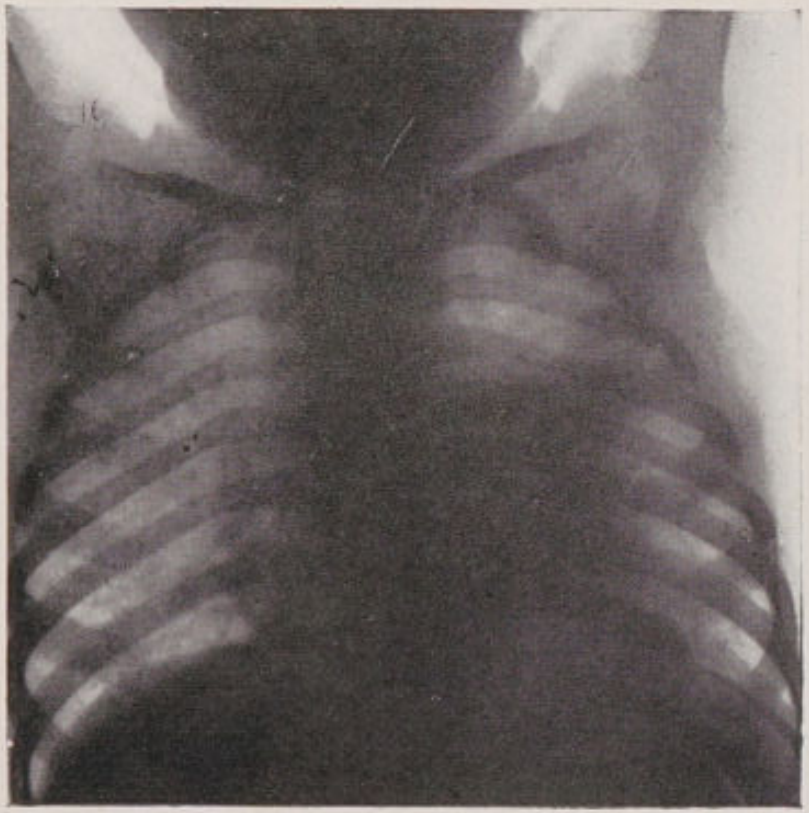
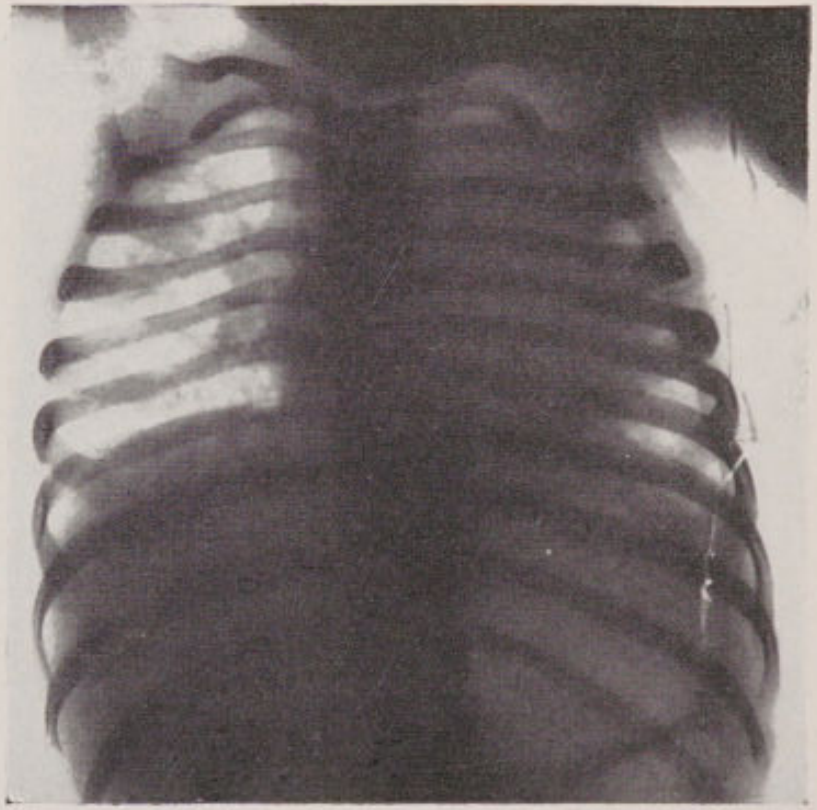


FIG. 7.—CASEOUS PNEUMONIA AND PRIMARY CAVITY.

Kitty C. Aged 4 months. Caseous pneumonia of right lung with cavitation in mid zone, and enlarged paratracheal glands. Commencing miliary spread in left lung.





FIGS. 8A-8D.—'ADULT'-TYPE PHTHISIS.

Fig. 8A.—Theresa S. Feb. 1, 1940; aged 2 years. Mantoux 1-100 positive; temperature 100°F.; S.R. 15; a diagnosis of tuberculous caseous pneumonia was made, mainly on clinical grounds. Condition gradually became worse, with some loss of weight, appetite unimpaired, and able to sit up until July, 1940.

Fig. 8B.—July 25, 1940. T. 102° F.; extension of caseous area in left lung and fluid in axilla.
Aug. 15, 1940. Mors during hæmoptysis.

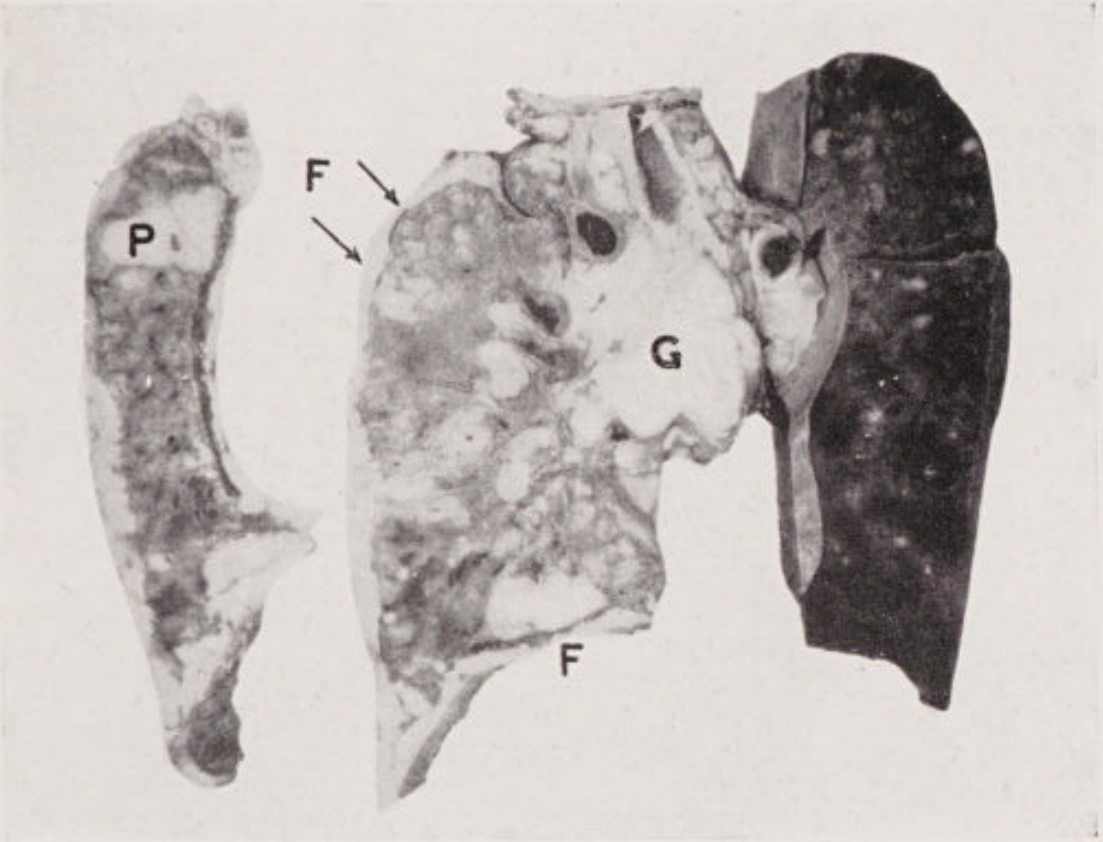


Fig. 8C.—Dr. Pagel's report on anatomical specimen: "Left section shows a Parrot-Ghon focus (P) in the sub-apical region of the ventral parts of the left lung. Right section: the left lung contains foci (F) due to broncho-aspiration, the left hilar glands (G) being greatly enlarged and caseous: there are hardly any foci in the right lung." (*By courtesy of Dr. Walter Pagel.*)

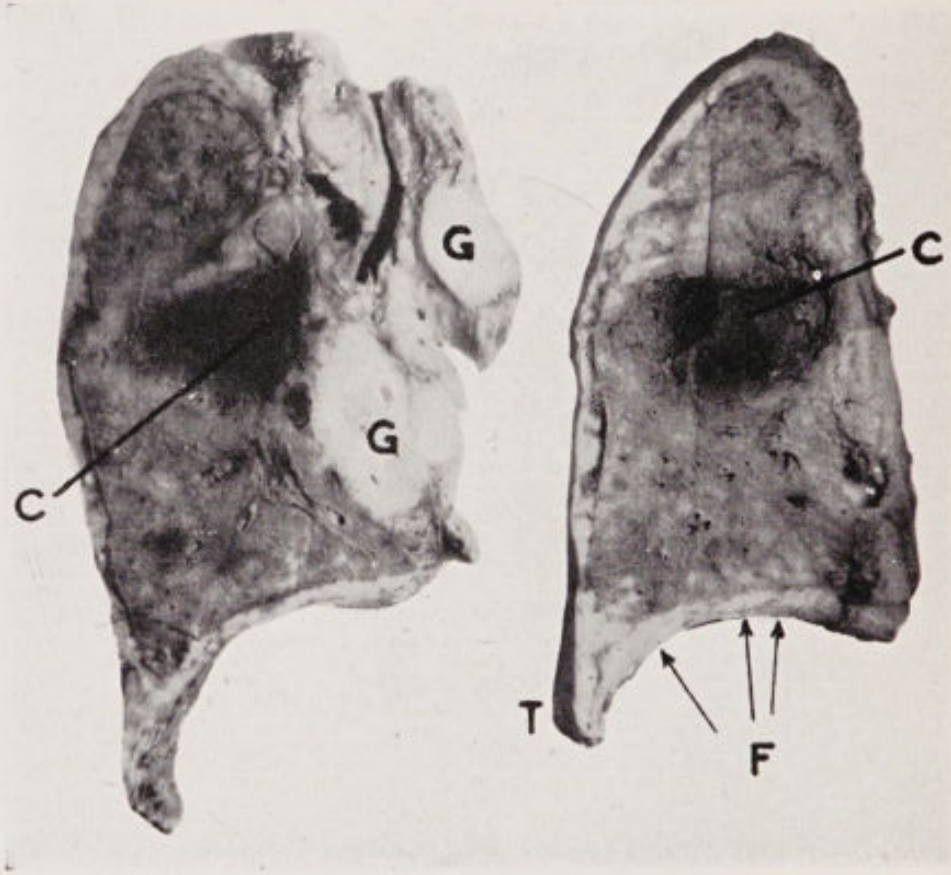
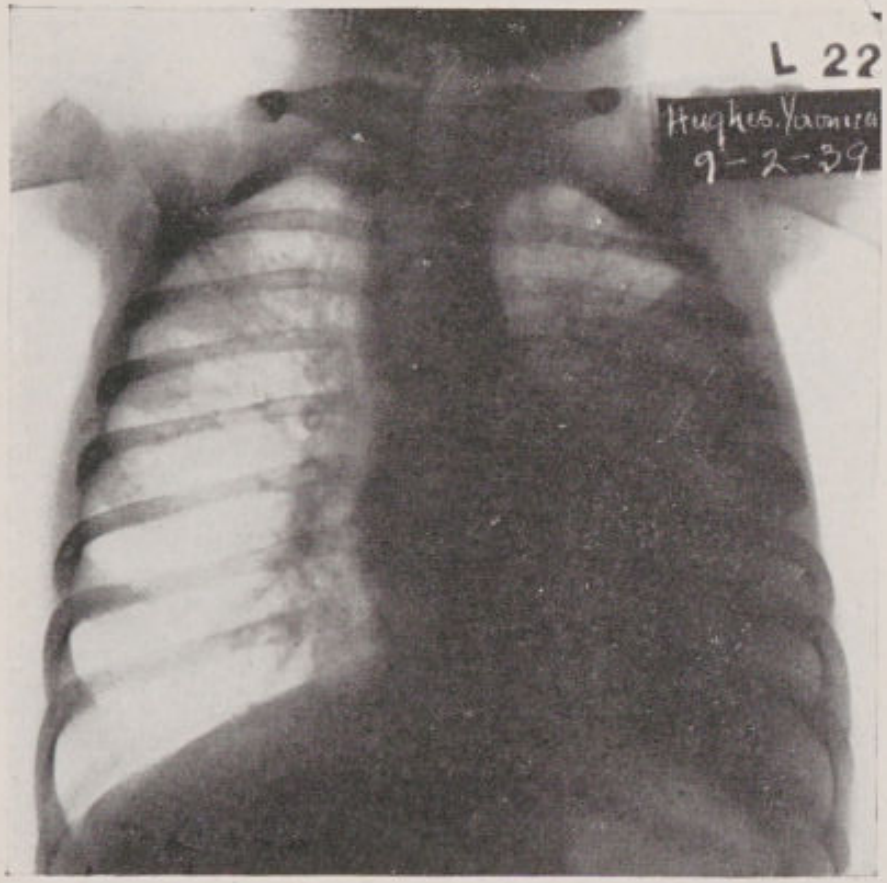
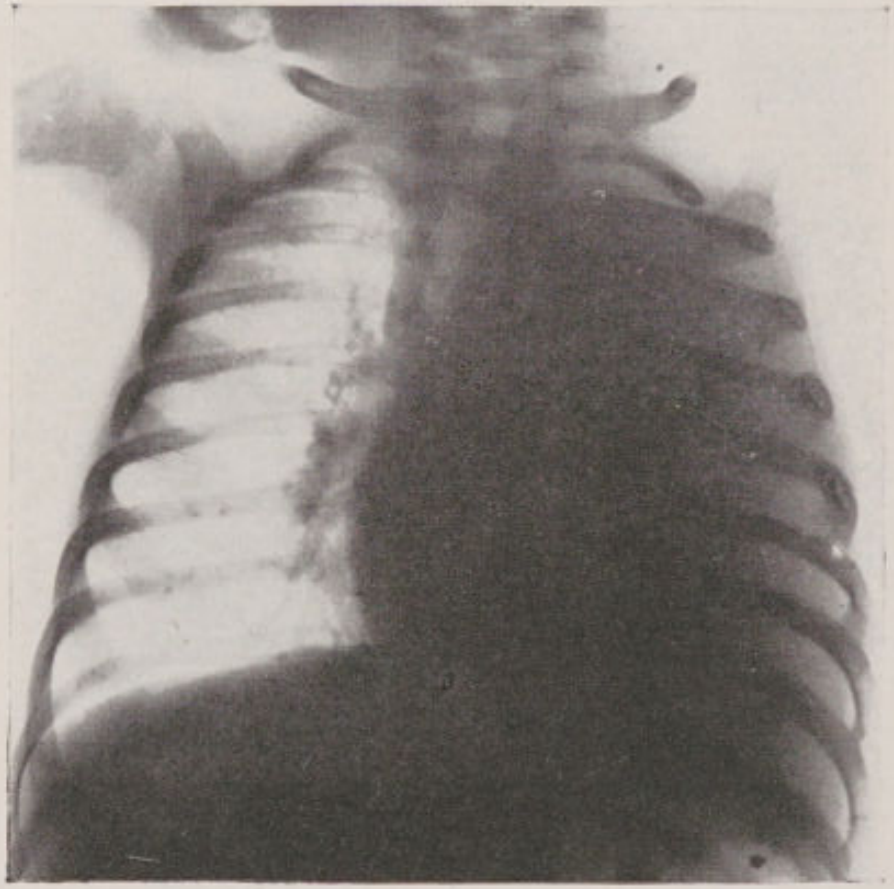


Fig. 8D.—"Here are shown two slices of the dorsal parts of the left lung, showing a cavity filled with blood clot (C) in the mid-zone, and in the mediastinal part another caseous intra-pulmonary gland (G). No gross perforation of a gland into trachea or bronchus was found. This is a picture of rapid pulmonary phthisis, rather of the adult type, following immediately bronchogenic aspiration from a primary complex." (*By courtesy of Dr. Walter Pagel.*)





FIGS. 9A, 9B.—UNILATERAL PHTHISIS.

Fig. 9A.—Veronica H. Feb. 9, 1939; aged 7 months. *See Example*, p. 107). Shadow over greater part of left lung.

Fig. 9B.—Three weeks later: extension of disease. Autopsy showed tuberculous pneumonia with multiple small cavities.

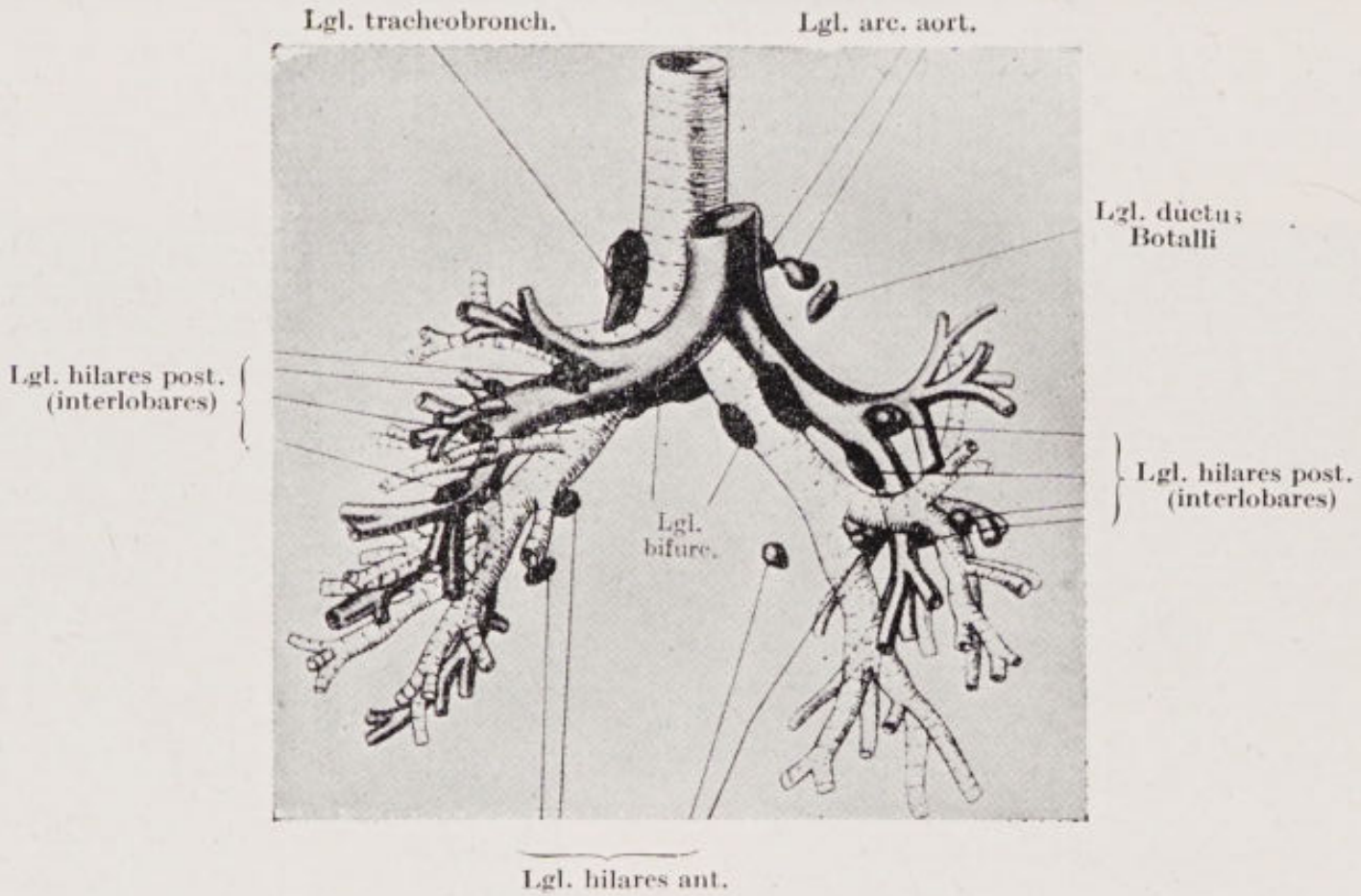


FIG. 10.—THE BRONCHO-PULMONARY TREE WITH LYMPH-GLANDS.

(From Engel and Pirquet's 'Handbuch der Kindertuberkulose'—Engel's 'Die Klinik des Primaerkomplexes.')

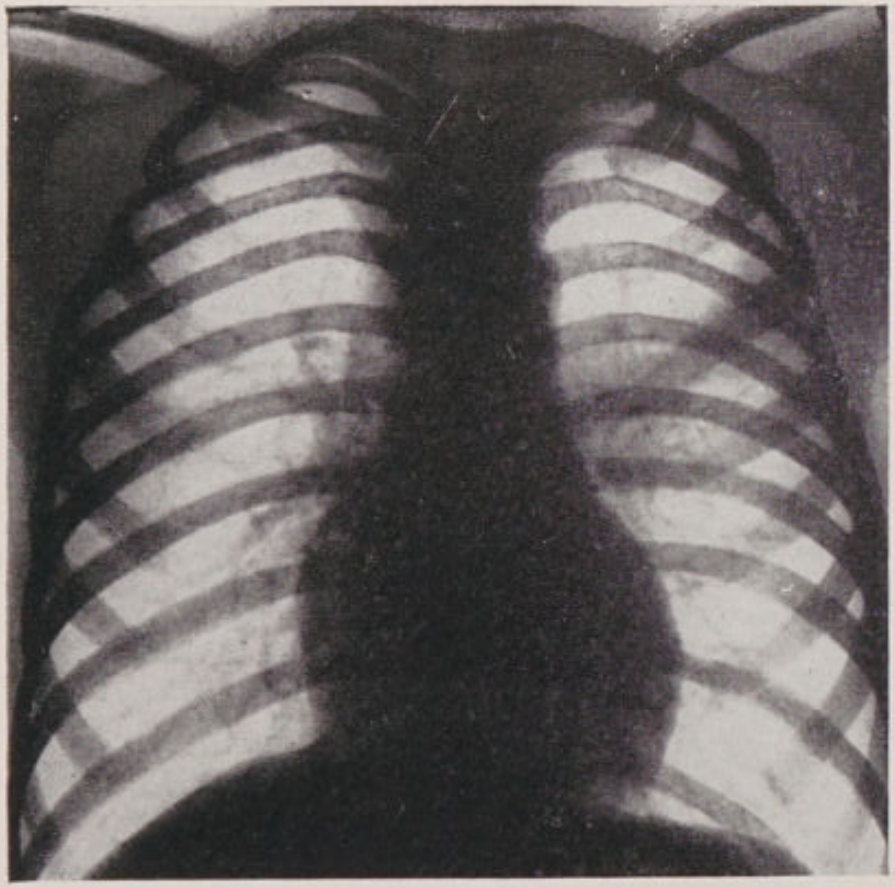


FIG. 11.—PRIMARY COMPLEX.

Tommy H. Aged 8 years. Encephalitis was suspected on clinical grounds. Tuberculin test positive. Primary complex with resolving infiltration is seen in left mid zone. Complete recovery.

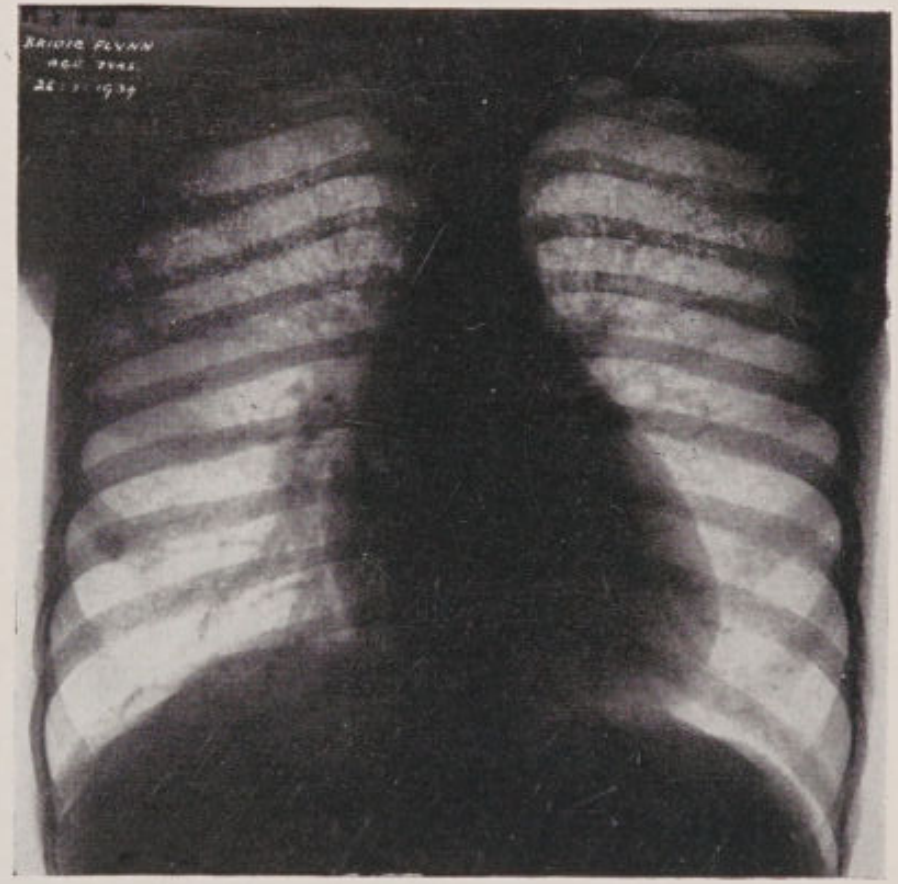
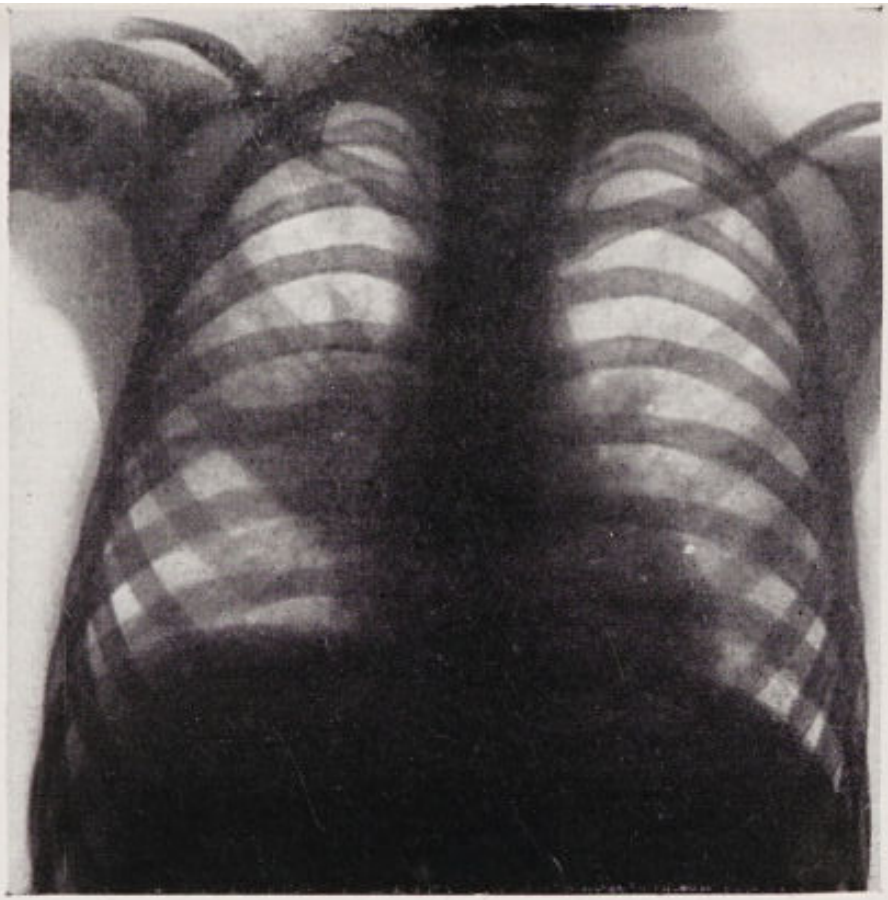


FIG. 12.—PARROT-GHON FOCUS.

Bridie F. Aged 7 years. Partial calcification of primary focus, which is seen at intersection of 5th and 7th ribs, and in gland at right hilum.



FIGS. 13A-13C.—HILAR PERIADENITIS FOLLOWED BY ASPIRATION PNEUMONIC PHTHISIS.

Fig. 13A.—Edward B. May 25, 1938; aged 5 years. Complained of headache; Hamburger positive; primary focus not seen; shadow in right hilar region, with thickening of interlobar fissure, is due to enlarged glands and probably atelectasis of right mid-lobe. Rest at home followed by marked improvement. After 3 months child suddenly became very ill.

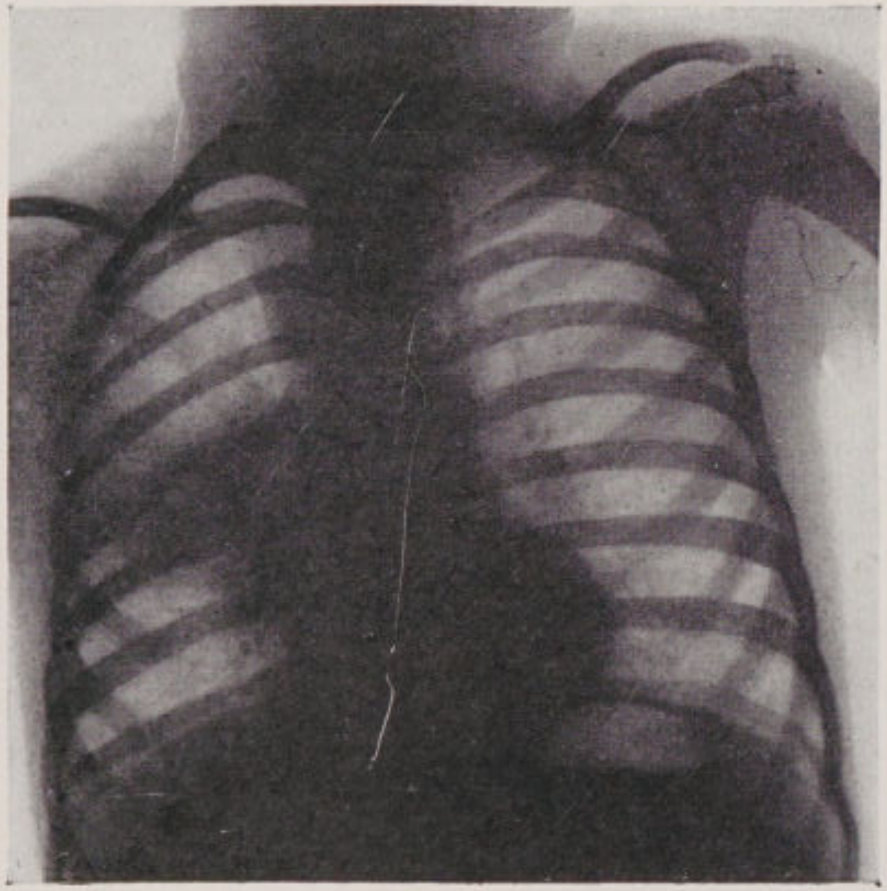


Fig. 13B. — Aug. 31, 1938. Admitted to hospital, with clinical picture and physical signs of broncho-pneumonia of right lung. Radiological appearances suggest caseation over original site with extension in neighbourhood. Treated by bed-rest and careful nursing, after 6 weeks child made an uninterrupted recovery. Condition most probably that of aspiration lobar pneumonic phthisis, although a super-imposed non-tuberculous pneumonia cannot be excluded.

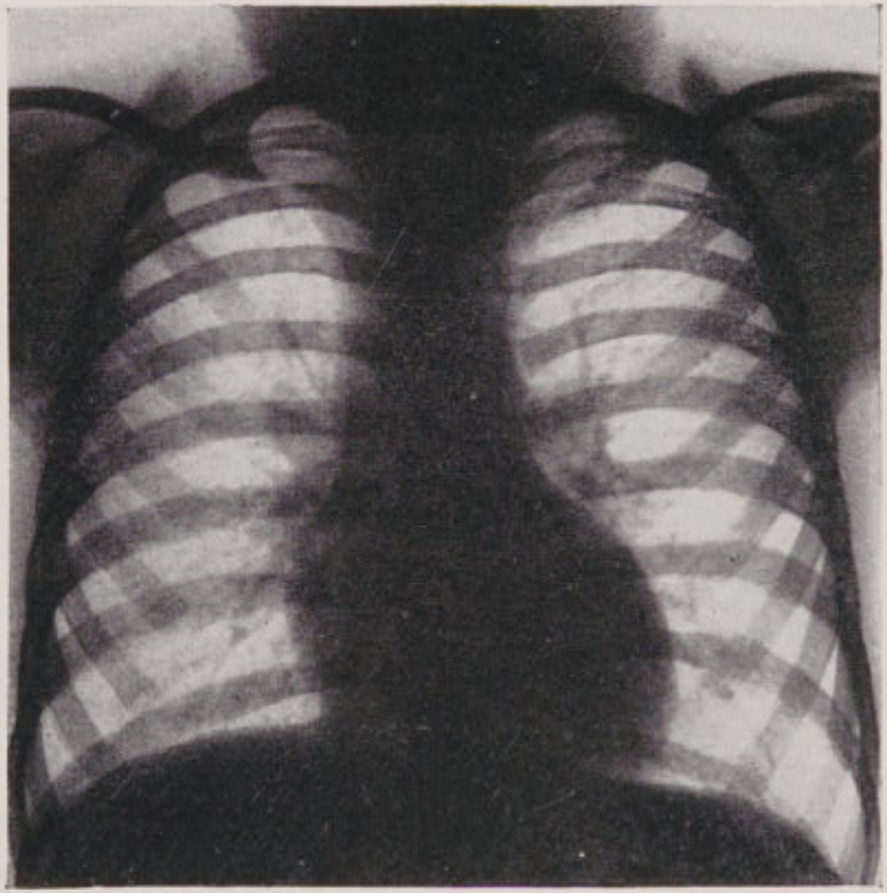
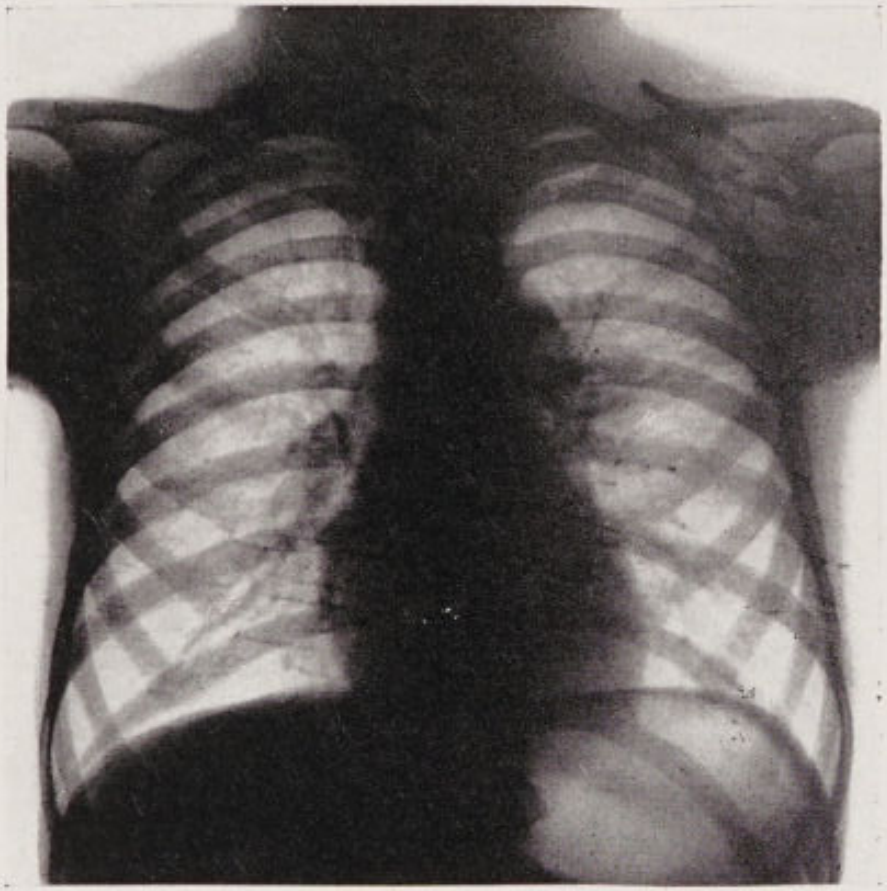


Fig. 13C.—June 6, 1940. Lungs clear, still rather heavy hilar markings, but no obvious calcification.
Sept. 9, 1941. In very good health, attending school.



FIGS. 14A-14D.—PARTIAL ATELECTASIS.

Fig. 14A.—William O'T. Sept. 24, 1937; aged 7 years. Complained of abdominal pain; Hamburger positive. Shadow seen in region of left para-aortic glands; primary focus suspected slightly outside and below the glands.

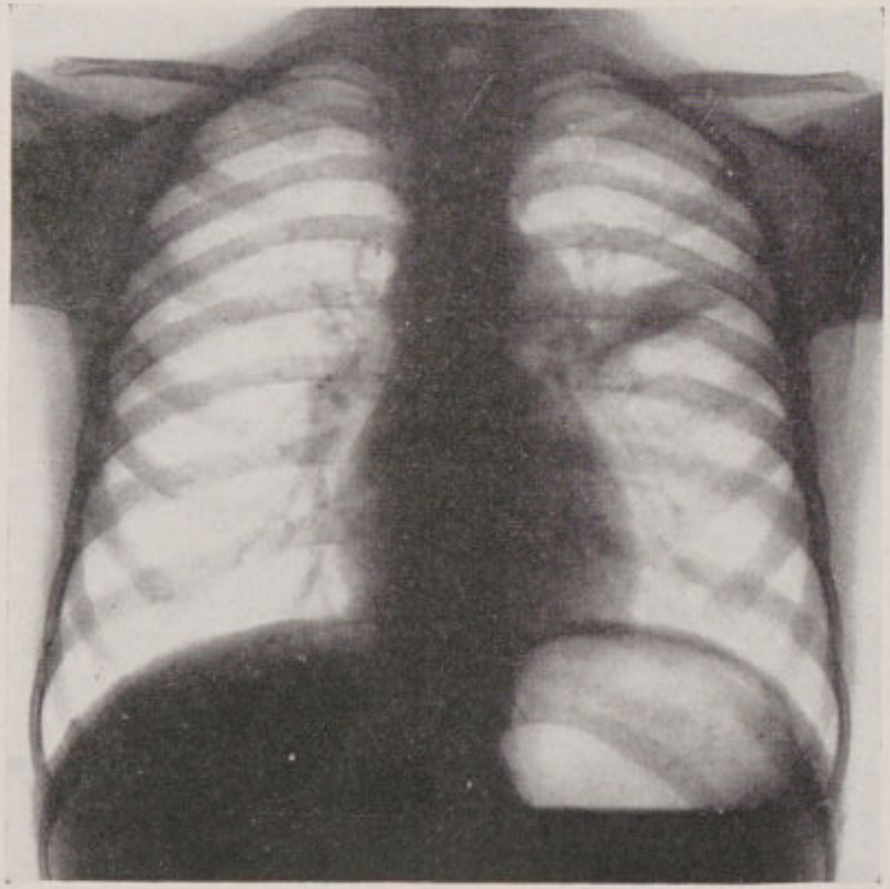


Fig. 14B.—Nov. 29, 1937. Partial atelectasis of left upper lobe due to pressure of glands on bronchus supplying the part.

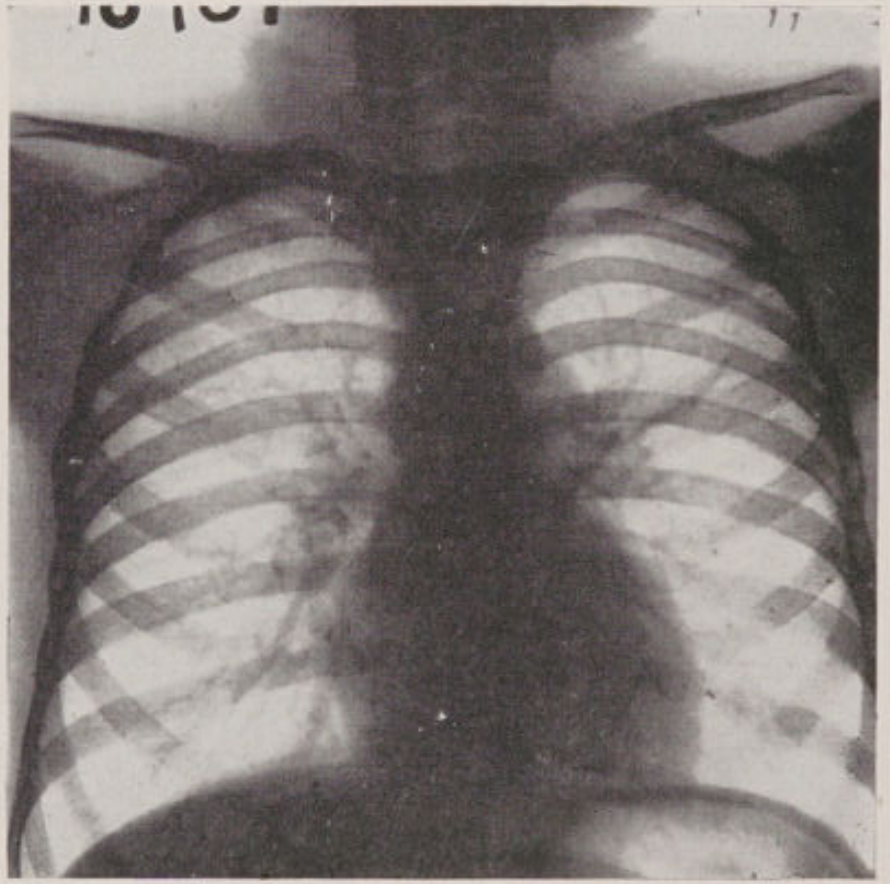


Fig. 14C.—Nov. 7, 1938. Almost complete re-aeration of the portion of lung, due to gland shrinkage.

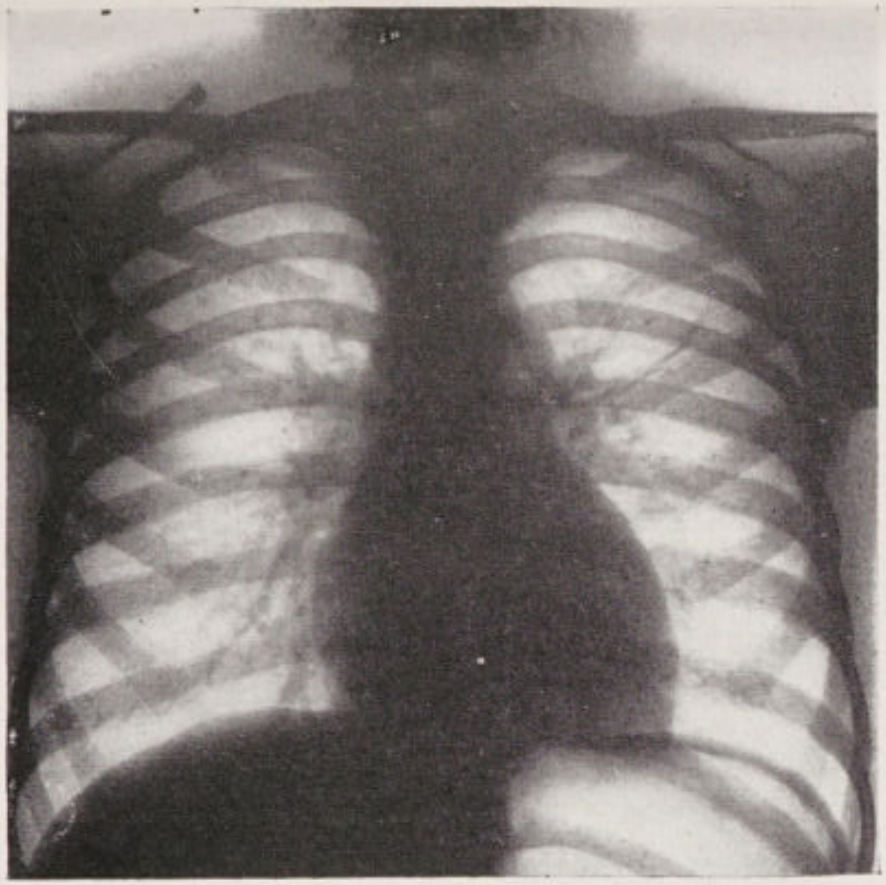


Fig. 14C.—June 5, 1940. Calcification commencing in gland.

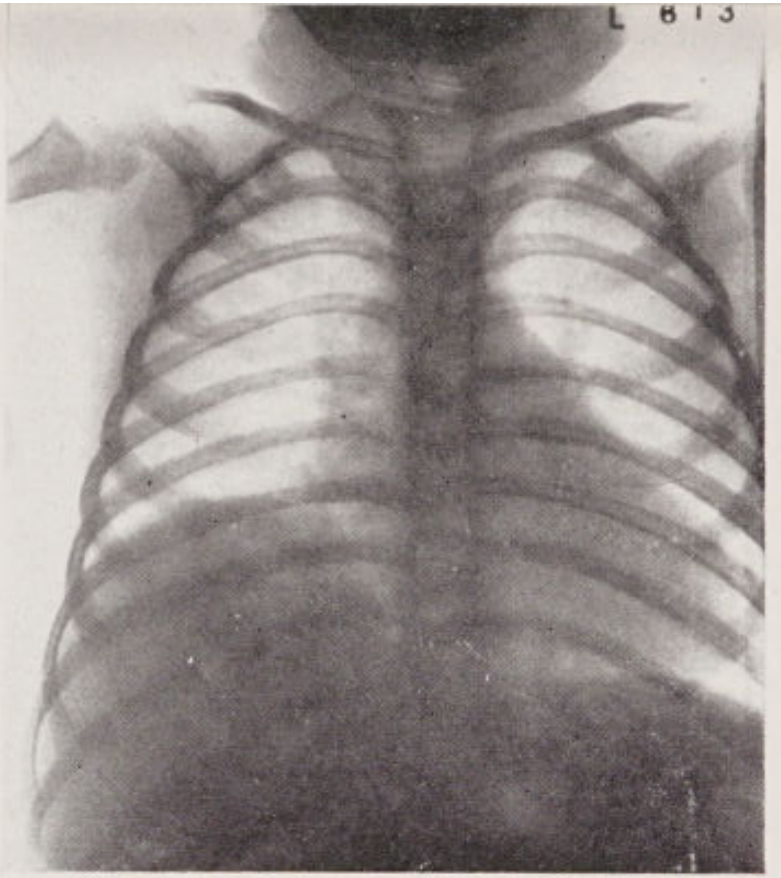


FIG. 15.—PARALYSIS OF DIAPHRAGM.

Michael H. Aged 2½ years.

Jan. 26, 1934. Aged 8 months. Hamburger negative.

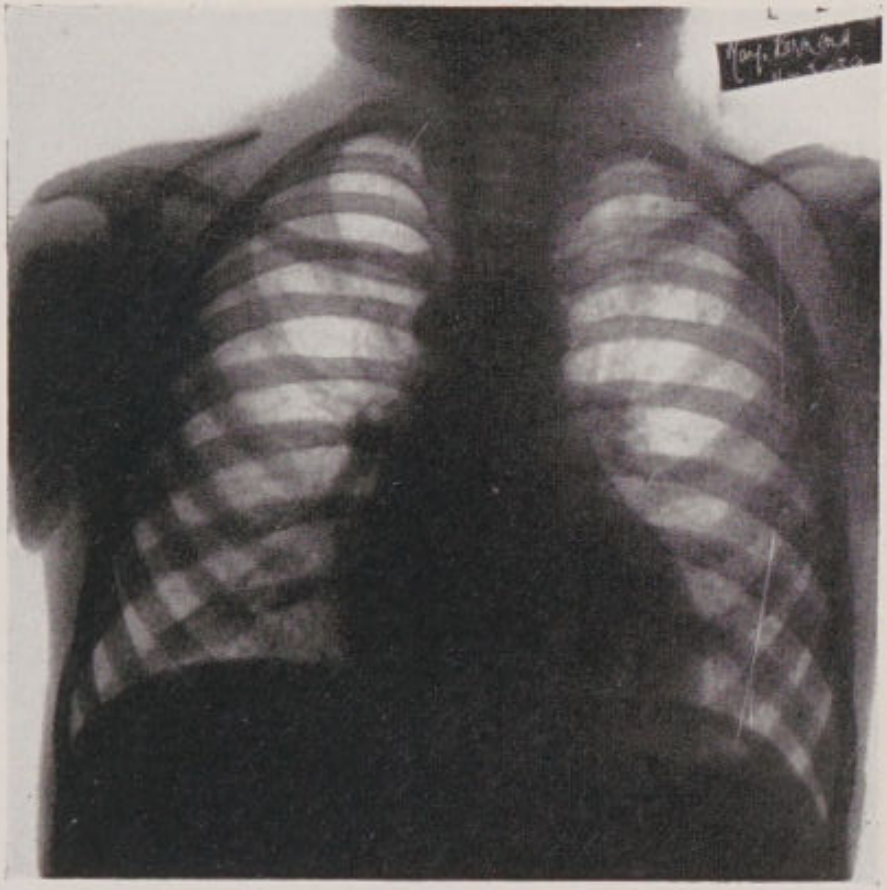
July 31, 1934. Aged 1¼ years. Hamburger positive, enlarged right hilar gland, no lung focus visible.

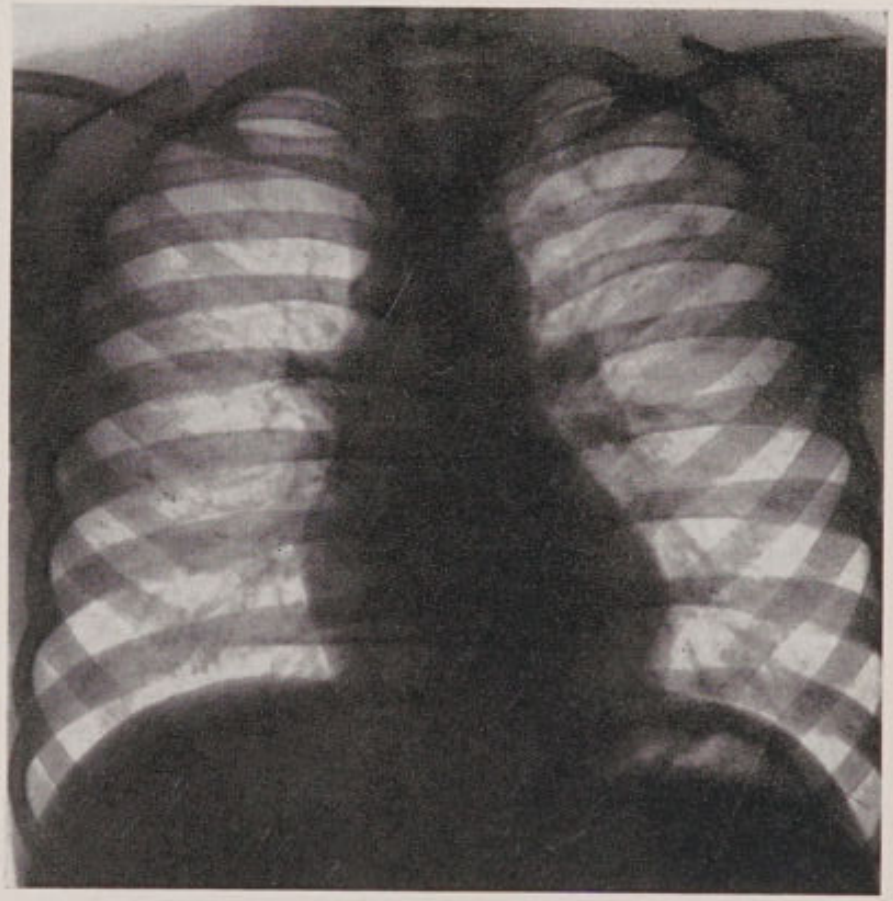
October 19, 1935. Admitted to another hospital with "bronchopneumonia."

Nov. 21, 1935. Aged 2½ years. Radiograph (*Fig.* 15): marked enlargement of right hilar glands and paralysis of right dome of diaphragm (elevation with loss of respiratory movement on screening); the gland pressure caused paralysis of the right phrenic nerve.

March 1, 1936. Aged 3 years. Dome of diaphragm descending.

Oct. 18, 1937. Aged 4½ years. Diaphragm descended and moving normally, right hilar glands subsided, no calcified residue seen.





FIGS. 16A-16B.—SIMON FOCUS, FOLLOWED BY ASSMANN FOCI.

Fig. 16A.—Bernard M. Treated in 1934, aged 1 year, for primary complex of right lung. Mother died of phthisis.

Sept. 11, 1939. Aged 6 years. Brought on account of cough; Father now has phthisis. *Fig.* 16A shows calcification of right hilar and paratracheal glands (primary tuberculosis); there is a Simon focus in the left apex above the clavicle (Type II tuberculosis); as the left apex was not completely clear, sent to a sanatorium for 6 months; the apex cleared, leaving calcified Simon focus only.

Fig. 16B.—April 25, 1941; aged 8 years. There are now several infraclavicular foci of the reinfection type (Assmann foci) in the left upper lobe (commencing tertiary phthisis). Sanatorium treatment, with successful collapse of left upper lobe.

1947. Well, attending school.

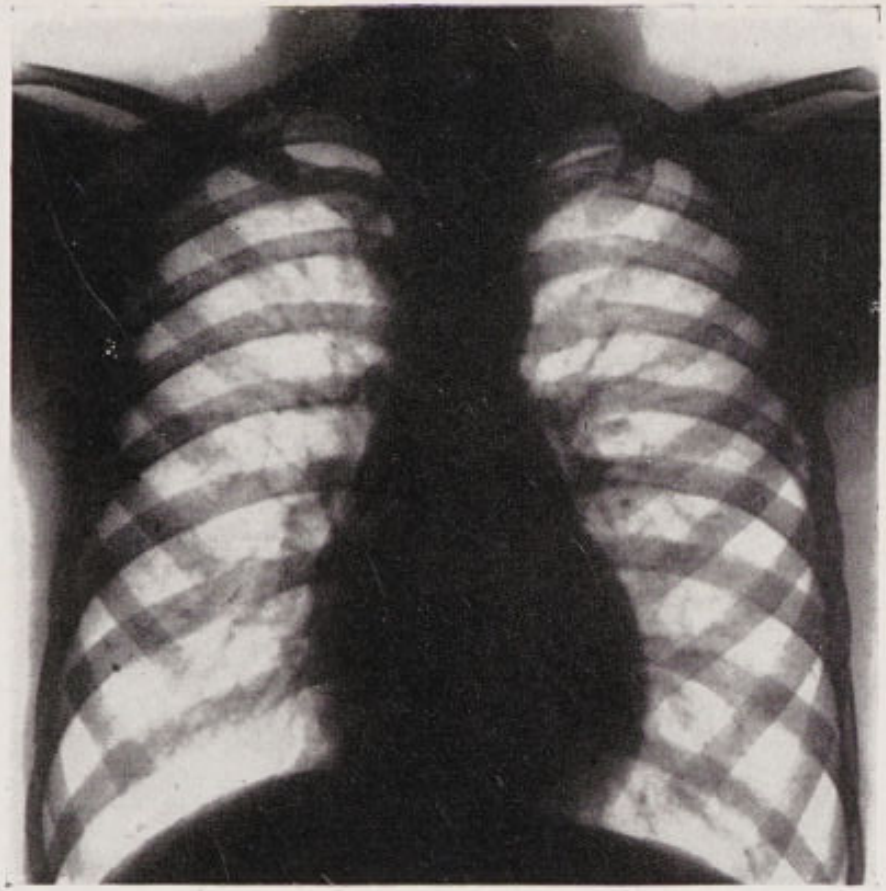
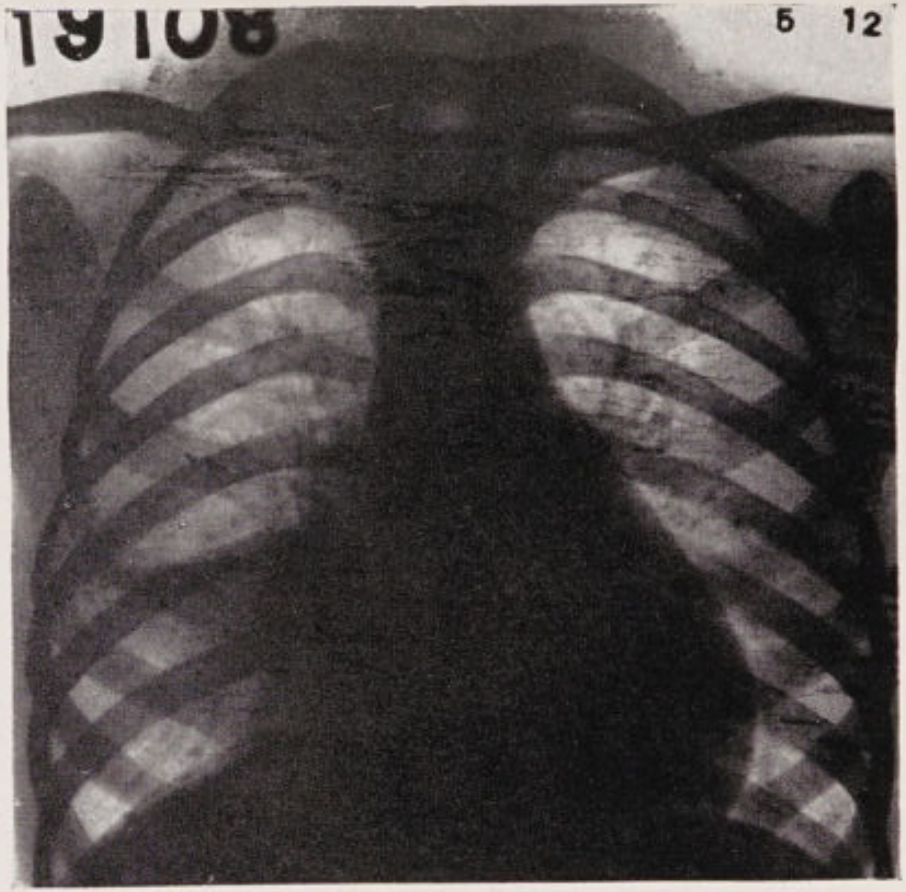


FIG. 17.—PRIMARY APICAL FOCUS AND GLAND.

William M. Aged 9 years. Father died of phthisis when William was 1½ years old. Hamburger positive ; the focus is lying immediately behind the right clavicle, and is recognized as a primary and not a Simon or Assmann focus by the calcified regional superior tracheo-bronchial gland.



FIGS. 18A-18D.—ATELECTASIS OF RIGHT MIDDLE LOBE.

Fig. 18A.—Annie W. Dec. 5, 1938 ; aged 8 years. Child complained of general malaise, anorexia, and fatigue ; Hamburger positive ; A.P. radiograph shows wedge-shaped shadow with well-defined upper border, in right lower zone, and enlarged glands in right hilum. It is impossible from this film to exclude interlobar pleurisy, but glands suggest atelectasis. Treated by rest at home.

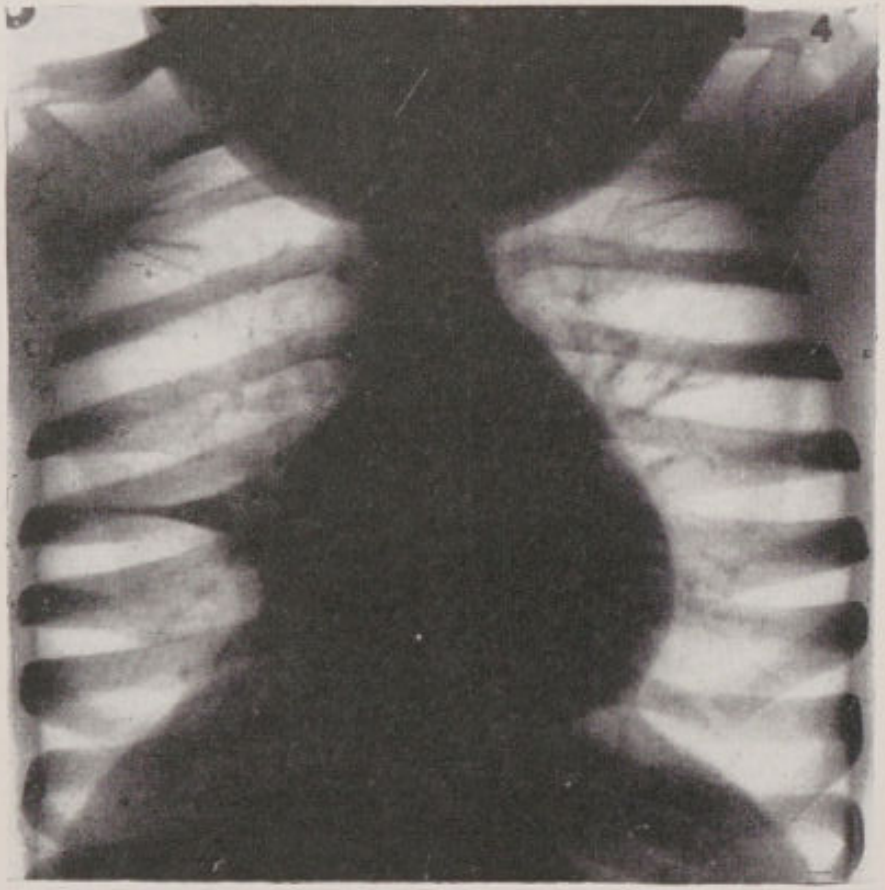


Fig. 18B.—April 24, 1939. Lordotic position shows thin wedge-shaped shadow clearly defined.

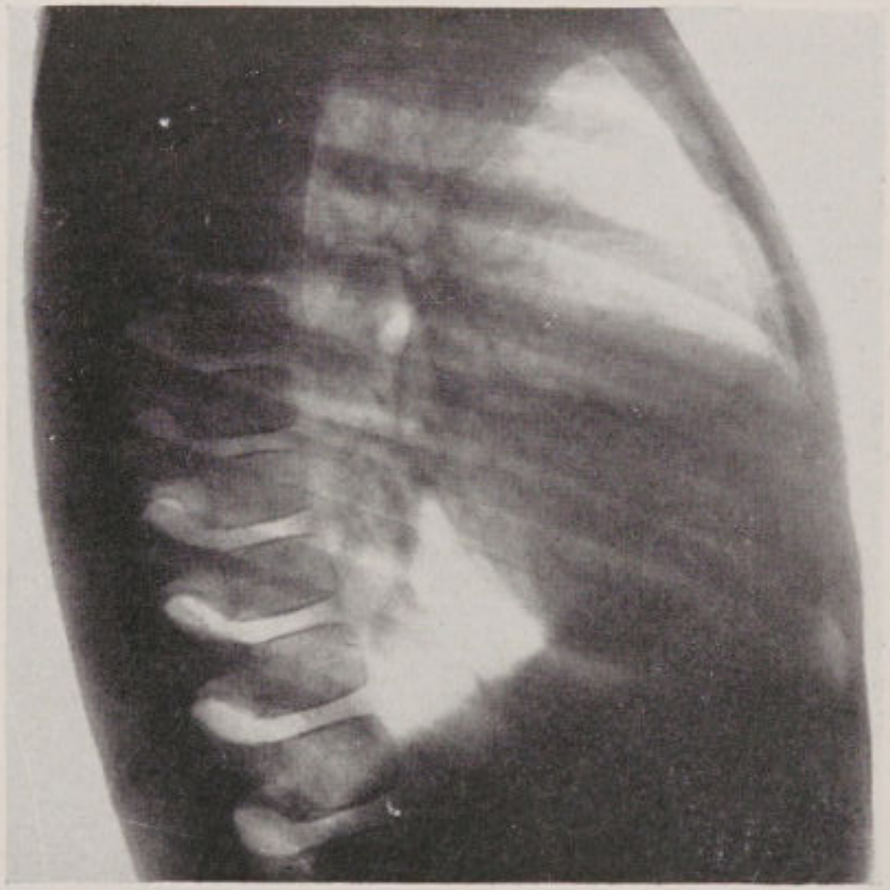


Fig. 18C.—April 24, 1939. Lateral view shows shadow clearing.

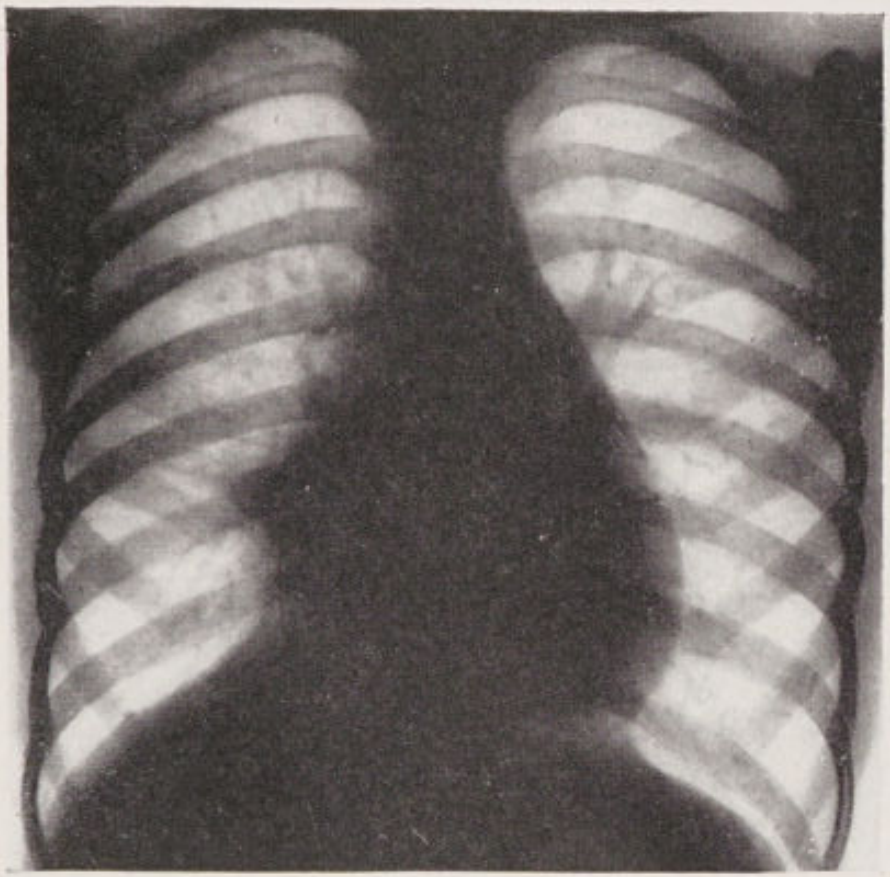


Fig. 18D.—April 28, 1939. A.P. view shows shadow receding One year later lobe re-aerated. Well in September, 1941.

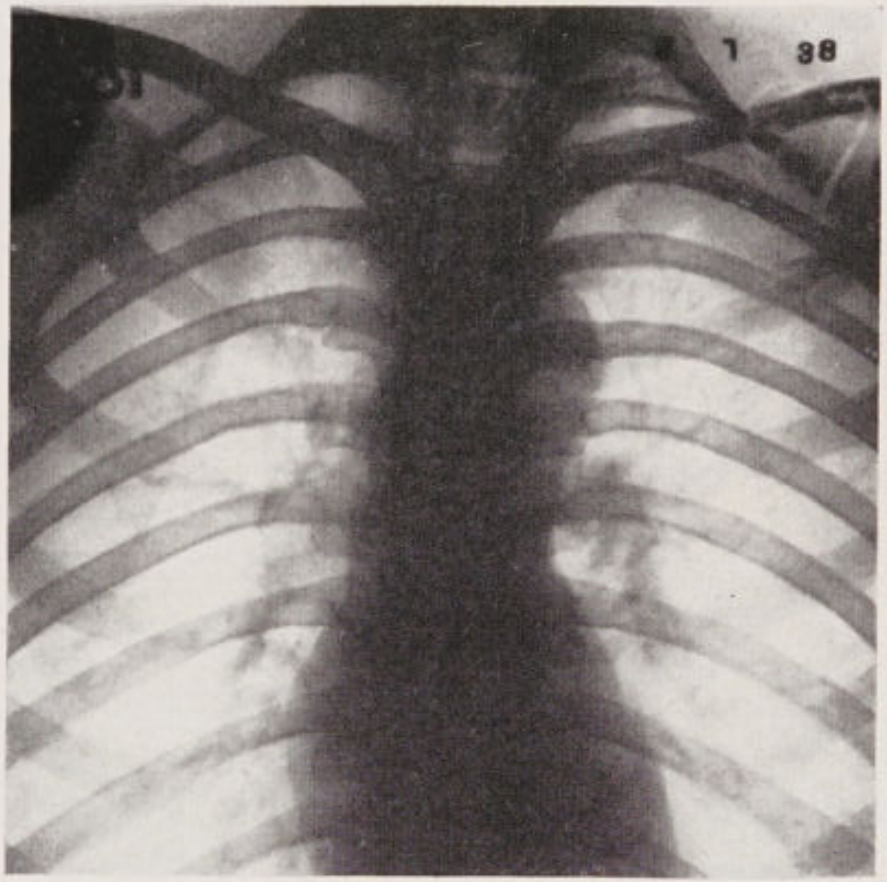
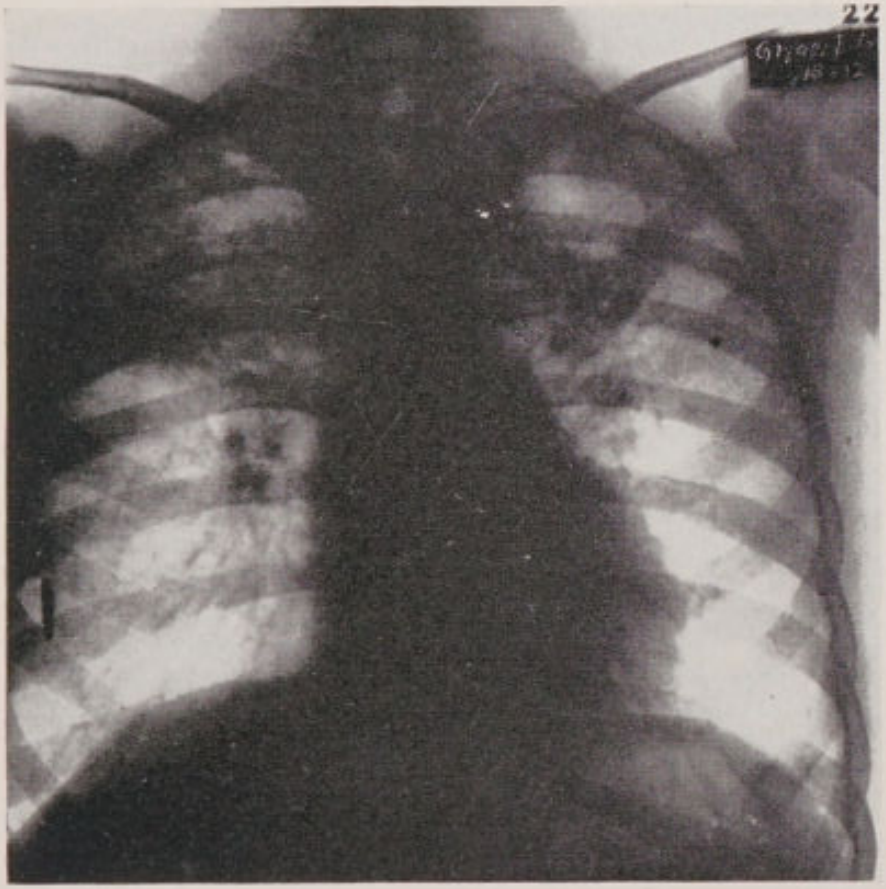
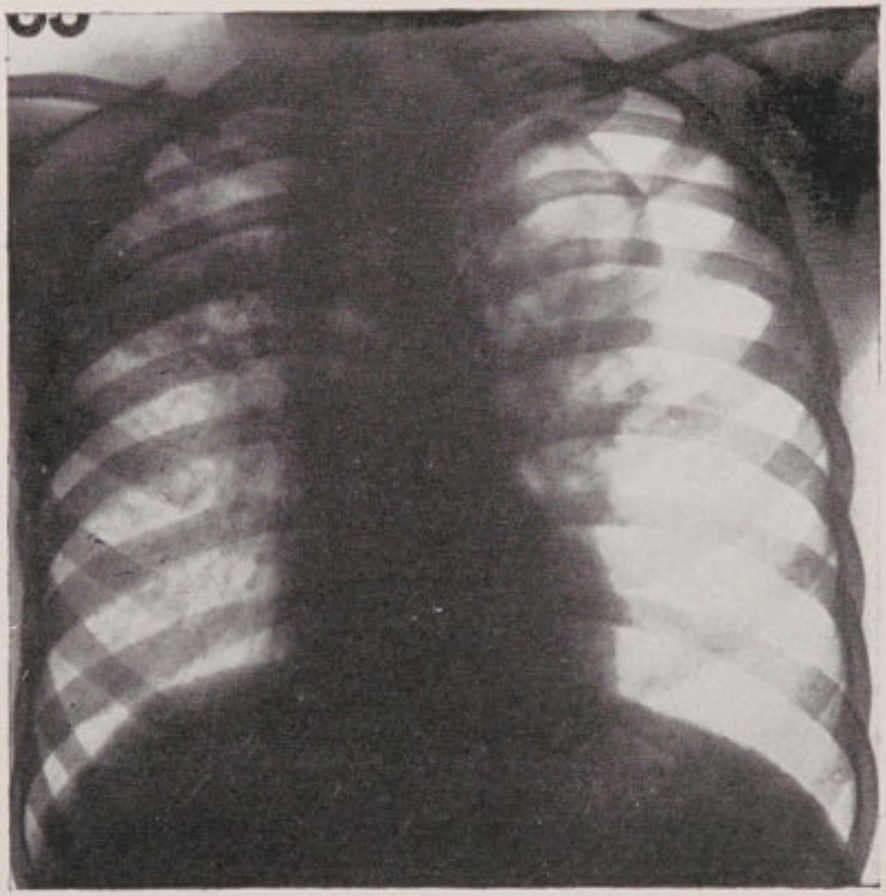


FIG. 19.—ASSMANN FOCUS IN A YOUNG ADULT.

Typical Assmann focus: a small circumscribed perifocal infiltration in the 1st right interspace.





FIGS. 20A, 20B.—BRONCHOGENIC PHTHISIS.

Fig. 20A.—Edward G. Dec. 15, 1938; aged 10 years. (*See Example*, p. 114.) Bilateral phthisis with involvement of both upper lobes; large cavity in left infraclavicular region.

Comparison between this film and *Figs.* 21 and 48 will emphasize the difficulty of deciding by radiological appearances as to the type of phthisis (infiltrative or nodular) seen in children.

Fig. 20B.—Jan. 7, 1939. Partial collapse after attempted artificial pneumothorax; cavity not closed.

Sept. 1, 1941.—Lesions healing by fibrosis, but cavity remains open; thoracoplasty under consideration.

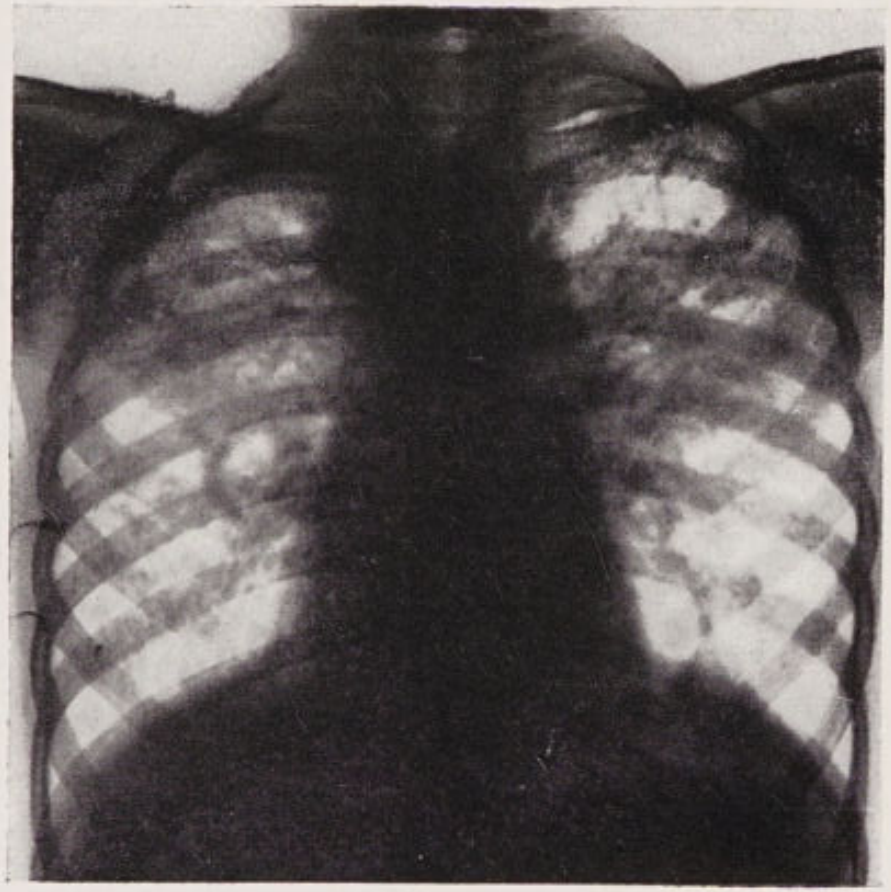


FIG. 21.—BILATERAL PHTHISIS, WITH CALCIFIED APICAL FOCI.

Katherine D. Aged 9 years. Both lungs show extensive downward spreading phthisis, with cavitation. Behind the left clavicle there is a group of partially calcified nodules, evidently hæmic in origin; these are a stepping stone from the primary complex (not seen in this film) to ulcerative phthisis.

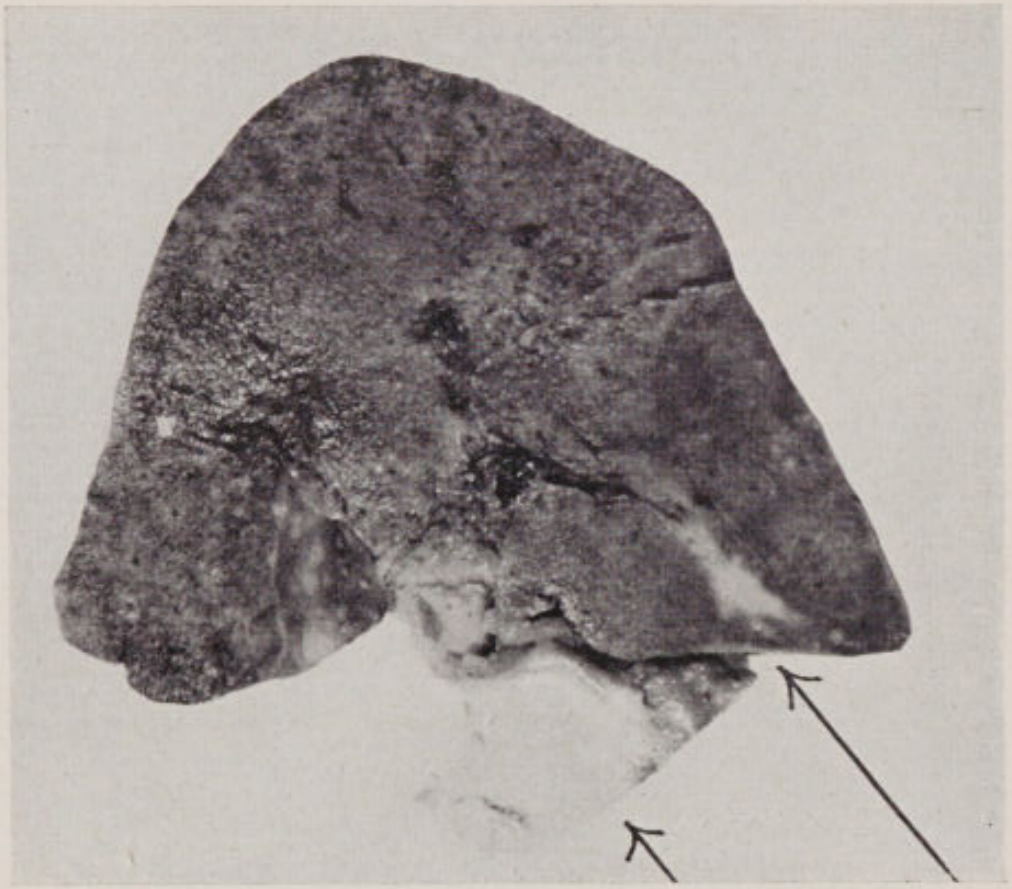


FIG. 22.—LIVER IN CONGENITAL TUBERCULOSIS

James K. Aged 6 weeks. (*See Example*, p. 44.) Liver viewed from dorsal aspect. Caseous Ghon focus (long arrow) in lower part of right lobe of liver; large caseous portal gland (short arrow); liver studded with miliary tubercles.

(*By courtesy of Dr. Walter Pagel.*)

some cases (*Fig.* 5), but it is uncommon in infants, because the degree of caseation which would lead to visible calcification often proves fatal.

3. *Mediastinal Glands.*—If the focus is small, or is first X-rayed in the healing stages, it may not be observed ; in fact, very frequently all that is seen is gland tuberculosis. This massive glandular enlargement is very typical of infant tuberculosis. Transitory enlargement of the glands, showing blurry edges and less dense shadow, occurs in non-specific conditions, such as bronchopneumonia, acute pneumonia, and whooping-cough, and also in bronchitis ; here a negative tuberculin test and rapid subsidence of the glands will decide the diagnosis. Tuberculous glands which are encapsulated and show a firm outline are not difficult to diagnose when the tuberculin test is known to be positive ; the prognosis is fairly good. All large convex or tumour-form opacities are suggestive of enlarged caseous tuberculous glands when seen in the hilar or paratracheal regions (*Fig.* 5A). Non-encapsulated glands show a blurry out line of peri-adenitis ; here the course of the disease is much more severe (*Fig.* 4). Sometimes, however, the glands do not appear as discrete entities, especially amongst the paratracheal groups ; the swollen glands displace the blood-vessels, and these will produce a ' ribbon ' or ' chimney-stack ' shadow on one or both sides of the trachea (*Fig.* 3). Post-mortem examination of many cases has confirmed the latter finding. The error of taking the film during the phase of full expiration will give a false impression of this shadow ; it must be distinguished by experience, and if necessary by repetition of the film ; often the convex outline of the gland can be seen, with deep penetration, through the shadow cast by the vessels. Several films may be necessary to confirm the gland shadow ; if it persists and the tuberculin test is positive, the diagnosis is certain. An enlarged thymus gland casts a typical broad shadow on either side of the trachea, it fills only the upper half of the thorax, and has no connexion with the heart shadow or mediastinum ; it is extremely rare.

4. *Atelectasis.*—The soft bronchi of infants are easily compressed by enlarged tuberculous glands, which may result in atelectasis of a lobe, or part of a lobe ; even a whole lung may be involved (*Figs.* 30, 31). This is more commonly seen between the ages of 1 and 3 years. The shadow is smooth and uniform, resembling that produced by lodgement of a foreign body in a main bronchus. If the atelectasis is in the lower zone,

the heart is drawn towards the affected side, thus differing from pleural effusion; if in the upper zone, the trachea is deviated to the affected side. The condition is not always easy to differentiate from a primary infiltration, except that it remains for longer, generally for months; final re-entry of air may be gradual or sudden, and commencing calcification in gland or focus may be seen appearing through the atelectatic shadow (*Fig.* 31B).

Jacobson's Phenomenon: On screening, there is a pendulum movement of the mediastinal shadow away from the unaffected side during inspiration, and away from the affected side during expiration. This is typical of atelectasis.

5. *Miliary Tuberculosis.*—Very common in untreated infants. From upper mediastinal glands showing peri-adenitis and no encapsulation, and also from encapsulated glands to a less extent, there is danger of a massive blood-stream spread. In pictures of infants suffering from miliary disease of the lungs, enlarged paratracheal glands are usually to be seen, and the glands will be found at autopsy (*Fig.* 6). In the radiological picture of miliary tuberculosis, all stages may be observed, varying from hyperæmia of the apices to millet-seed foci and later snowstorm appearances. Early diagnosis is very difficult and is often only suspected from the appearance of hyperæmia and enlarged upper mediastinal glands. In some cases the diagnosis is made only at autopsy, as the condition is not always recognizable radiologically.

6. *Caseous Pneumonia.*—This type of phthisis is not uncommon at this age period. It arises either as a break-down of the primary focus, or by rupture of a caseous gland into a bronchus causing aspiration pneumonia. When the primary focus has previously been under observation, the change to caseation can be recognized radiologically, as well as clinically. If the case is seen for the first time, the radiological diagnosis between caseous pneumonia, atelectasis, and primary infiltration is not always possible. The former is associated with a heavier and denser shadow than is the primary infiltration, and also with a less uniform and defined shadow than that produced by atelectasis. Diagnosis can only be made in consultation with the clinician. With caseation there is a rise of temperature. The percutan test may be negative in advanced cases, due to terminal lowered allergy. Cavitation is due to central liquefaction (*Fig.* 7).

7. *Phthisis.*—In infants almost every form of acute pulmonary tuberculosis may be found, but a diagnosis of the underlying

pathological condition is seldom possible from the radiological appearances. It is only at autopsy that the true nature of the lesion may be revealed (*Figs.* 8, 9 ; *see also Figs.* 23, 49). Cavities must be looked for ; these are naturally very serious for an infant, although they may clear spontaneously and rapidly ; they are thin-walled and if single due to liquefaction of a primary focus.

Finally, the infant who shows no enlargement of mediastinal glands, no infiltration in the lung field or calcified node, and who has a negative tuberculin reaction to two strengths (up to Mantoux 1–100) may be pronounced non-tuberculous. The X-ray and tuberculin test must be repeated six weeks after admission to hospital, as the incubation period from last contact will then be passed.

THE CHILD OF 6 YEARS

Normal.—At this age the child may be taken in the upright position ; in order to get the spine straight and the child steady, it is well to have him sitting astride a seat facing a low back on which he can rest his arms ; it is difficult to get small children to stand evenly on both feet. The diaphragm has now descended, but not fully, and the thorax is commencing to elongate, thus leaving the hilum more visible and projecting from behind the heart border. The bifurcation of the trachea is at the level of the 4th or 5th thoracic vertebra. The right main bronchus is shorter, wider, and more vertically directed than is the left, and makes a more obtuse angle with the trachea ; this accounts for the more frequent involvement of the right lung in primary tuberculosis, as indicated in statistics amongst children. The right hilum is at the level of the 5th rib behind, and the left hilum at the level of the 6th rib behind.

The hilum needs careful study (*Fig.* 10, and *Frontispiece*). The arrangement of the structures as they enter the lung from above downwards on the two sides is as follows :—

Right.	*Left.*
Eparterial bronchus	Pulmonary artery
Pulmonary artery	Bronchus
Hyparterial bronchus	Pulmonary veins
Pulmonary veins	

On both sides the veins are in front, the artery in the middle, and the bronchus behind. Some lymphatic glands lie in the

hilum, packed between the compressible pipes of the above-mentioned systems; this is important to remember when pressure symptoms due to enlargement of mediastinal glands arise, as they do between the ages of 0 and 5 years. The root of the right lung lies behind the superior vena cava and the right atrium, and below the azygos vein; the left root passes beneath the aortic arch and in front of the descending aorta. The phrenic nerve passes in front of the root. Assmann, Duken, and Engel have shown, through experimental filling of the bronchial vessels, that the shadow cast on a normal radiograph at the hilum is that of blood-vessels and not of bronchial tubes or of glands.

Abnormal.—At six years, the most frequent tuberculous abnormalities are seen in the region of the hilum; the glands in this locality are not so markedly enlarged as they are in the infant, however, and the paratracheal groups are less frequently involved. At this age period, one must look for the following changes: primary infiltrations or healed primary foci in the lung field, involvement of any of the tracheo-bronchial gland groups, atelectasis (usually over a rather small area), and indefinite shadows in the region of the hilum. It must be remembered that a report "roots abnormally dense", in the presence of a negative tuberculin test, signifies non-specific enlargement of the root glands, and is not due to bronchial gland tuberculosis.

1. *Primary Focus.*—A primary Ghon focus may be recognized in the lung field by its perifocal infiltration; the latter being more localized at this age than in the infant. As the infiltration clears, there is seen a small opacity, with edges which are fluffy in the earlier stages but appear denser and more circumscribed as calcification proceeds (*Figs.* 11, 12). In situation the focus may be anywhere in the lung; at this age it is seen usually in the best aerated parts, often appearing in the mid-zone of the lung. Many cases heal without visible radiological residue. Increased striation is of no particular significance in the diagnosis of these cases; it means increased blood to the part and has nothing to do with the bronchial or lymphatic drainage of tuberculous foci in children. Primary cavities are more rare at this age than in the infant, in fact, they are very rare; if present, they are usually seen in the hilar region, where a primary focus has caseated and liquefied.

2. *Glands.*—Enlarged hilar glands are often seen at this age without any primary focus being visible (*Fig.* 28). On the

right they are clearer than on the left, for on the left the lower tracheo-bronchial glands are covered by the heart border, and can best be seen, if at all, in a lateral view. According to Hamburger, the appearance of enlarged hilar glands is indicative of tuberculous changes when the hilar shadow protrudes beyond the field of the heart shadow into the lung fields. Non-specific enlargement often gives a less dense shadow, but diagnosis must depend on the tuberculin test.

3. *Hilum.*—This is an age when shadows seen in the hilum present difficulty in interpretation (*Figs.* 13, 14). If the primary focus lies in the middle or posterior part of the lung and near to its mediastinal surface, it may be overlooked, as it may be obscured by the hilar gland shadows. Besides a primary perifocal shadow, there may also be atelectasis, interlobar pleurisy, or a caseous pneumonia developing over the primary focus or elsewhere by bronchial aspiration. With caseous pneumonia, the shadow will become denser and later central cavitation may be seen. Diagnosis cannot be made in a hurry, and consultation between radiologist and clinician is essential.

In 'occult' tuberculosis, i.e., positive test with negative radiograph, repeat the X-ray examination at intervals, and search for obscure involvement of the mediastinal glands by employing lateral and oblique, in addition to anterior-posterior, views.

4. *Interlobar Pleurisy.*—This is said to show a triangular shadow with base to hilum. Lateral views are helpful, and it is usual to film the child in the lordotic position and then the interlobar line will be more apparent. But this will not help towards diagnosis of the underlying condition. Many authorities begin to doubt the presence of fluid as exploratory puncture yields negative results (*see* p. 78). The consensus of opinion at the present time is that these shadows are more often due to a partial atelectasis or to a primary infiltration than to fluid.

5. *Pleural Effusion.*—*See* pp. 76, 79 and *Figs.* 29, 39, 40.

6. *Atelectasis.*—A triangular shadow with base to hilum suggests atelectasis rather than interlobar pleurisy. At 6 an atelectasis is usually much less extensive than at 1 or 2 years and tends to be a partial collapse of a portion of a lobe (*Figs.* 14, 18, 30, 31).

7. *Paralysis of the Diaphragm.*—This occurs occasionally when enlarged glands press on the phrenic nerve at the hilum. The condition may be recognized by elevation of the dome of the

diaphragm on the affected side, which on screening shows little respiratory excursion. Resolution of the gland and shrinkage by calcification generally terminates the paralysis. Later, the calcified gland may be seen; in some cases there is no radiological residue. This condition is rare, but may be seen during the 2–4 years age period, the time when hilar glands are especially liable to gross enlargement (*Fig.* 15).

8. *Apical Lesions.*—The supraclavicular 'Simon' focus is a round focus, often solitary, sometimes in the form of a discrete dissemination, arising as a blood-stream metastasis from a primary focus; the latter may or may not be seen also in the film. The 'Simon' focus is found mostly by chance during the routine examination of positive tuberculin reactors; it is usually calcified when first recognized. It must be distinguished from the infraclavicular or 'Assmann' focus; the latter are bigger, have less tendency to calcification, and are extremely rare at 6 years, but cannot altogether be ruled out (*Figs.* 16, 21).

9. *Phthisis, Miliary Tuberculosis, and Caseous Pneumonia.*—Bronchogenic phthisis is rare but is found between 1 to 12 years (*Figs.* 8, 16, 20, 21, 49).

It may spread from a primary focus, Assmann focus, or by aspiration from a gland.

Miliary tuberculosis of the lung is at its lowest incidence between 6 and 10 years.

THE CHILD OF 12 YEARS

Normal.—At this age the shape of the chest is approaching the conical adult type, with almost complete descent of the diaphragm. The bifurcation of the trachea takes place at the level of the 5th and 6th thoracic vertebræ. The ribs have become more curved, and the hilum is emerging from behind the heart shadow as the latter becomes more elongated and less broad. The right main bronchus passes either outside or just along the heart border, and the left main bronchus is found lying just inside the heart shadow.

Abnormal.—

1. *Fresh Primary Focus.*—This occurs at all ages, but as age advances more healed cases are seen. Nevertheless a certain number of fresh infections are seen at 12 years, in some the focus and gland (*Fig.* 25), and in others glandular enlargement only, are seen. Sometimes the shadow of the infiltration round the primary focus is overlapped or merged in the hilar shadow.

Pleural effusion may follow the primary complex at this age. The primary focus and glands shrink after 3 to 6 months and commencing calcification may be seen in healing cases after 12 months.

2. *Healed Primary Complex.*—Amongst positive reactors at 12 years, the healed primary complex will be found comparatively often. Calcification is the only visible radiological residue of a healed primary infection; healing by fibrosis of primary focus or glands cannot be seen radiologically, which accounts for many inactive cases with positive test and no definite radiological abnormality. The criterion for the interpretation of a calcareous gland, according to Hardman, is an opacity of extreme density, sharply defined, irregular in outline, having the appearance of granular formation. These calcified glands must not be confused with blood-vessels or bronchi in section; the latter are small, smooth, regular, and round. A calcified focus is easily determined by its marked opacity; it is generally small, but may in exceptional cases be the size of a half-crown. It shrinks in time. The author has observed complete absorption of calcium salts; a child (aged 1 year) was treated for a large primary complex which 4 years later appeared well calcified; the calcified focus and gland gradually diminished in size; 6 years later (aged 11) radiologically the complex had disappeared.

3. *Hilar Shadows.*—These obscure shadows have been referred to in the discussion on the child of 6 years. They may be caused by enlargement of the hilar glands with peri-adenitis, or they may be due to a primary focus if the shadow of the perifocal infiltration overlaps the glandular shadow, or they may be due to partial atelectasis of the adjacent lung tissue (*Fig.* 18), or to interlobar pleurisy. Differential diagnosis between the above alternatives is sometimes impossible.

4. *Infraclavicular Focus.*—The Assmann focus may occur at 8 years, although it is usually not seen before puberty. A sharp look-out must be kept on the infraclavicular region. In the child these foci are nearly always hæmic in origin and some spread by aspiration from the hæmic Simon focus—that is to say, they are secondary to a primary complex which may be calcified, partially healed, or not very definitely apparent. The focus is recognized by a small round area of infiltration seen below the clavicle, or in the apex of the lower lobe or in the middle lobe. The Assmann focus is distinguished from a subapical primary focus in that it has no glandular response

whatever. The focus may be seen as a small discrete shadow, indicating fibrosis (*Fig.* 19), or it may be surrounded by an infiltration which has a tendency towards rapid liquefaction and cavitation. It is very important to recognize these infraclavicular foci at an early stage in children ; they are of far more serious prognosis than are primary foci, for if neglected they may develop rapidly into phthisis. They should be watched, rested, and treated by collapse therapy immediately should cavitation appear, for delay will render such treatment impossible if localized pleurisy produces adhesions.

5. *Miliary Tuberculosis.*—Miliary tuberculosis may be seen at this as well as at any other age. After infancy, the incidence is highest at about 14 years of age, and especially in girls.

6. *Phthisis.*—The radiological appearance of phthisis in children is of an infiltrative rather than of a fibrotic type ; the course is usually much more rapid than in the adult, and fibrosis is rare. Spread nearly always occurs from an infraclavicular focus (Assmann focus) and is from above downwards, with daughter foci on the opposite side. Cavities appear as in adults (*Figs.* 20, 21).

7. *Caseous Pneumonia*, at the site of the primary focus, may occur at this age, but is not seen so frequently as in young children. Aspiration pneumonia, due to rupture of a gland into a bronchus, is rare.

CHAPTER VI

CONGENITAL TUBERCULOSIS

General Remarks.—Young infants can be infected with tubercle bacilli before, during, or after birth. It is important to distinguish between the more common condition of inhalation infection which is due to contact with a phthisical mother just after birth, and the very rare condition of intra-uterine or intra-partum infection. Germinal infection in human beings has never been proven, but cases of congenital blood-stream infection have been established beyond doubt. The first case of congenital tuberculosis was described by Jacobi in 1861. A comprehensive survey was published by Beitzke in 1935; he examined 100 cases reported in the literature, and of these he passed 61 as true congenital tuberculosis. Since then further authentic cases have been reported. The conditions which are necessary to the proof of the congenital nature of the individual case are: (1) proof of tuberculosis in the mother; (2) proof of tuberculosis in the infant; (3) exclusion of the possibility of post-uterine infection by contact. Entry of the bacillus into the body of the fœtus can take place in two ways: either from the maternal blood-stream, or from the amniotic fluid.

Placental Infection.—The more likely route of entry is by the blood-stream; infection in this manner is dependent on the presence of tubercle bacilli in the maternal circulation. As a result of this maternal bacillæmia, one or more tubercles are formed in the placenta; such placental tubercles are not easily recognized macroscopically, and must be sought with great thoroughness. In serial examination of the placentas of tuberculous mothers, German workers have found as many as 27 per cent to 63 per cent to be infected, but in some instances as many as two thousand sections had to be cut before the tubercles were demonstrated. Not every placenta containing tubercles, however, leads to infection of the fœtus; for instance, no infection will reach the fœtus if, through tuberculous changes, occlusion of the placental vessels cuts off the fœtal blood-supply from that part of the placenta. When, on the other hand, tubercle bacilli penetrate into the fœtal side of the placental

circulation, they may reach the fœtal liver by the umbilical vein. After primary lodgement has occurred in the fœtal liver, one of the hepatic glands becomes involved, thus forming a primary complex. Most of the maternal blood-supply to the fœtus passes through the fœtal liver, and only a small stream reaches the heart direct. Primary lodgement of the tubercle bacillus is in the inferior region of the liver where the umbilical vein enters; thus the enlarged gland will be found in the group around the porta hepatica. In some cases not only the liver but also the lungs are found to be infected. The explanation in such cases is that in fœtal life the pulmonary circulation is largely cut out by the patency of the foramen ovale; the minute stream, however, which nourishes the atelectatic lungs flows so slowly that it favours lodgement of the bacillus; when the lungs expand at the first respiration after birth, the blood from the placental circulation flows freely until the cord is cut; thus there can arise a miliary infection of the lungs. Tubercle bacilli are also carried in the fœtal circulation to the spleen, and to a lesser extent to other abdominal organs; thus the disease becomes widely disseminated. The usual picture, therefore, in the type of congenital tuberculosis due to placental infection, is that of a primary complex in the liver (recognized by the great enlargement of the hepatic gland), with varying degrees of miliary spread into other organs, particularly the spleen and lungs. Some proportion of these infections may occur during parturition; this will happen if a tubercle in the placenta is torn during the separation of the placenta from the uterine wall, and bacilli are released in quantities into the umbilical vein; this is known as *intra-partum* infection.

Amniotic Infection.—Cases of congenital tuberculosis due to amniotic infection are considerably more rare even than are the placental hæmic type. They can occur only if a placental tubercle lies close to the amniotic sac, and if it should discharge its caseous contents into the amniotic fluid. Entry of tubercle bacilli to the fœtus from infected amniotic fluid can occur before birth: (1) by ingestion, with primary lodgement in the intestinal wall, or (2) by aspiration, with primary lodgement in the lung. Only two cases of infection by the ingestion of amniotic fluid have been described in the literature.

Theories.—Loewenstein, in recent years, has tried to prove the importance and frequent occurrence of congenital tuberculosis by demonstrating the presence of tubercle bacilli in the

blood in the umbilical vein of infants. Calmette and others advanced the theory of the filterable virus; the suggestion was that the virus entered the fœtal body during intra-uterine life and remained latent for years, producing symptoms of disease in later childhood. This theory, which is contrary to all experience of tuberculosis during the childhood period, was supported by the slender evidence of a few doubtful cases. We must regard all this work as purely hypothetical and still unproven.

Degree of Maternal Infection.—There seems to be no law on this point; reported cases show a varying degree of severity in the maternal infection. The majority of mothers with congenitally infected offspring show severe phthisis, but cases are also reported where the mother was only slightly infected, and recovered, although the infant died. With regard to involvement of maternal organs of generation, the question remains open; such infection is present in some cases, absent in others. With regard to time, it seems that the infection occurs late in pregnancy; if the fœtus is infected early, abortion follows. It is thought that those cases which come to term, with survival of the infant for a few weeks, are infected during the last two months of pregnancy. Should a congenitally infected infant remain in contact with his phthisical mother after birth, it is possible that he will acquire pre-allergic sub-primary exogenous foci in the lungs, which will complicate the picture. The large portal gland will, however, place the site of primary infection in the liver, and thus confirm the diagnosis of congenital tuberculosis.

Clinical.—Infants who have been infected congenitally by the tubercle bacillus die at any date after birth up to two months of age. Death from infection acquired after birth occurs seldom less than six weeks after primary implantation. Therefore, if an infant dies from tuberculosis under the age of two months, congenital infection should be considered. Diagnosis during life is extremely difficult, for the disease has no characteristic signs or symptoms. There may be a rise of temperature, but this will occur in other conditions; weight may be gained for a time, before a final drop; cough may be present, if the lungs are involved, but this can also occur in post-natal tuberculosis; the most important sign is swelling of the lymph-glands in the upper abdomen, which may sometimes be felt; towards the end the liver and spleen may be palpable; dyspnœa, œdema, and cyanosis may appear as terminal symptoms, but are not always present. The only pathognomonic sign is a positive skin test,

which appears before the third week of life. The tuberculin reaction, however, may later become negative owing to swamping of the system and resultant lowered allergy.

Two examples of congenital tuberculosis will now be given ; the first is a case of placental infection, hæmic spread from placenta to fœtal liver (Price, 1937) ; the second a case of infection of the fœtal lungs by aspiration of infected amniotic fluid (Pagel and Hall, 1946).

Example 1.—James K., male infant aged 38 days, was admitted to St. Ultan's Hospital. Weight 4 lb. 4 oz., temperature 101° F. During the following week the temperature was between 97° and 99° F ; the infant gained 8 oz. No enlargement of liver or spleen, and no cyanosis or dyspnœa. On the seventh day the temperature rose to 102° F, remained at 100° F for the next three days, and the weight increased by 3 oz. ; there were no definite physical signs in the lungs or abdomen. On the eleventh day he collapsed, temperature 97° F, and he died aged 49 days. A radiograph taken immediately after death showed nothing in the lungs except some loss of translucency in both upper lobes, suggestive of bronchopneumonia.

Maternal History : The mother, who was suffering from pulmonary tuberculosis had given birth to 10 children (of whom 6 survived) and had had three miscarriages. She gave birth to this baby in her home on July 27, 1936. She did not breast-feed or handle the baby herself, but he remained with her in the four-roomed house for five weeks. In January, 1937, she was examined gynæcologically and had no pelvic tuberculosis ; she died in a sanatorium soon after that date. The father was free from tuberculosis.

Autopsy (by courtesy of Dr. Pagel) on the infant showed the liver and spleen studded with disseminated foci, some small and others larger. The portal lymph-gland was enlarged to the size of a small plum, caseous and liquefying. At the hilum of the spleen were a few glands the size of a grain of wheat ; the lungs showed hæmatogenous dissemination, with a few solitary caseous foci ; the bifurcation gland contained a tiny caseous focus. In this case the congenital origin of the disease was proved by the size of the portal gland, which far exceeded that of any other gland. The solitary pulmonary foci could have been mistaken for exogenous lesions, but they were recognized as blood-borne by : (*a*) the condition of the regional lymph-glands ; and (*b*) the fact that all organs showed transitional stages between miliary disseminations and large foci (*Fig.* 22).

Example 2.—This case was presented as an addition to the very few instances reported in the literature of the ' aspiration-type ' of congenital tuberculosis. A female infant died aged 17 days ; the mother failed to show any evidence of tuberculosis apart from an uncharacteristic pyrexia after delivery. Autopsy on the infant showed universal spread of submiliary foci throughout both lungs and confluent caseation in the hilar lymphatic glands ; the abdominal organs were practically free. Acid-fast bacilli were found to fill dilated bronchioli and alveolar ducts, and the histological pictures conveyed a suggestion of aborted alveolization of the lungs, possibly due to the tuberculous infection of the fœtus.

CHAPTER VII

PRIMARY TUBERCULOSIS OF THE LUNG MEDIASTINAL GLAND TUBERCULOSIS PLEURISY

PRIMARY TUBERCULOSIS OF THE LUNG

General Remarks.—The primary stage of tuberculosis of the lungs comprises by far the largest group of tuberculous manifestations which occur during childhood. The primary infection is in itself a benign condition. But nevertheless the fact that the child has developed such an infection is of the utmost importance, and such cases must be regarded as potential victims of fatal forms of the disease. Every case of tuberculosis has to pass through the primary stage; many primary tuberculous infections heal during the first stage and progress no further; after a space of years such a healed primary focus confers a definite degree of immunity towards a later infection. Many children pass through the primary infection without symptoms. A certain small proportion of untreated cases of primary tuberculosis do not heal and proceed to the development of tuberculosis of the lung, bone, meninges, or other organs; these secondary and tertiary manifestations in children are most damaging and often prove fatal. Every endeavour, therefore, should be made to recognize the occurrence of the primary infection, and to treat it immediately; a well-treated first infection will invariably lessen, if not altogether eliminate, the risk of further extension of the disease.

Every country has statistics to show percentage of children who at various age periods have passed or are passing through this phase. A proportion of these children with positive reactions are in need of treatment; but these numbers must necessarily vary according to the groups from which they are drawn, whether amongst healthy school children or amongst sick children attending hospital. Price (1940) reports amongst 2135 children aged 3–14 years in the extern department of a general city hospital that 132 (6 per cent) were found by routine

skin tests to be suffering from active primary tuberculosis of the lungs with radiological evidence of focus or glands. Price (1938), amongst 200 tuberculin-positive children aged 3 to 14 years in the extern department of the Royal City of Dublin Hospital, found the following: 22 per cent healed primary complex; 31 per cent active primary complex; 21 per cent occult primary, requiring supervision only; 5 per cent erythema nodosum (associated with primary infection); 4 per cent abdominal tuberculosis; 3 per cent phthisis; the remaining 14 per cent showed extrapulmonary lesions. Thus, omitting the healed cases, 57 per cent of all positive reactors required treatment, and 21 per cent supervision.

The influence of treatment on the prognosis of primary tuberculosis can best be judged by figures, and these are scanty. In the days of Ghon the mortality-rate from tuberculosis amongst infants was considered to be 90 per cent. Rosenberg and Kereszturi report 348 tuberculous infants (28 per cent showing radiologically visible lung lesions), of whom, after treatment, 9 years later 9·1 per cent had died. Collis reports a series of 120 primarily infected children who were under observation for 3½ years, of whom only 1 died of tuberculosis. For more recent figures the reader is referred to *Tables V, VI* (pp. 12, 13). From Norway are reported (*see Table XI*) 4 tuberculosis deaths within 10 years amongst 109 children with benign primary tuberculosis.

Table XI.—Fate of In-patients 3 to 10 Years after Treatment for Tuberculosis (*Salomonsen and Traetteberg, Oslo*, 1935).

Age at Onset	Total Cases	Deaths from Tuberculosis	Deaths from other Causes	Living with Active T.B.	Living and Well
0– 1 yrs.	17	—	1	—	16
1– 2 yrs.	8	—	—	—	8
2– 5 yrs.	27	1	1	—	25
5–10 yrs.	40	1	1	1	37
10–14 yrs.	17	2	—	—	15

The Primary Infection.—The primary infection occurs when the tissues of the child come into contact with the tubercle bacillus for the first time; the reaction of the individual body at this first encounter is different to any later reactions which may occur after subsequent infections. The importance of this first encounter cannot be over-estimated, for on it depends the

future fate of the tubercle bacillus in the body of the young individual. The question which is in the balance at this time is whether the defence capabilities will be successful in localizing the invaders with resultant complete healing, or whether they will fail to restrict the invasion with resultant dissemination either immediately or by reactivation at some future date. It has already been explained that entry to the lungs takes place by inhalation of moist droplets from coughed sputum or spoken particles from a phthisical contact, or by dust from floors contaminated by tubercle-laden dried sputum.

The Primary Focus.—When a tubercle bacillus is inhaled it reaches the end of a small bronchiole and lodges in the wall of an air space at the periphery of the lung. At the site of lodgement is formed the primary focus (*Figs.* 8C, 47A). The earliest primary focus as yet described was reported by Pagel and Price (1943) in a child who died aged 9 weeks; this focus formed an alveolitis with central caseation, with tubercle bacilli and mononuclear cell infiltration, not yet encapsulated nor followed by involvement of the regional gland. Another primary focus at a slightly later stage, but still without capsule, in a child who died aged 11 weeks, is demonstrated in *Figs.* 55A–C.

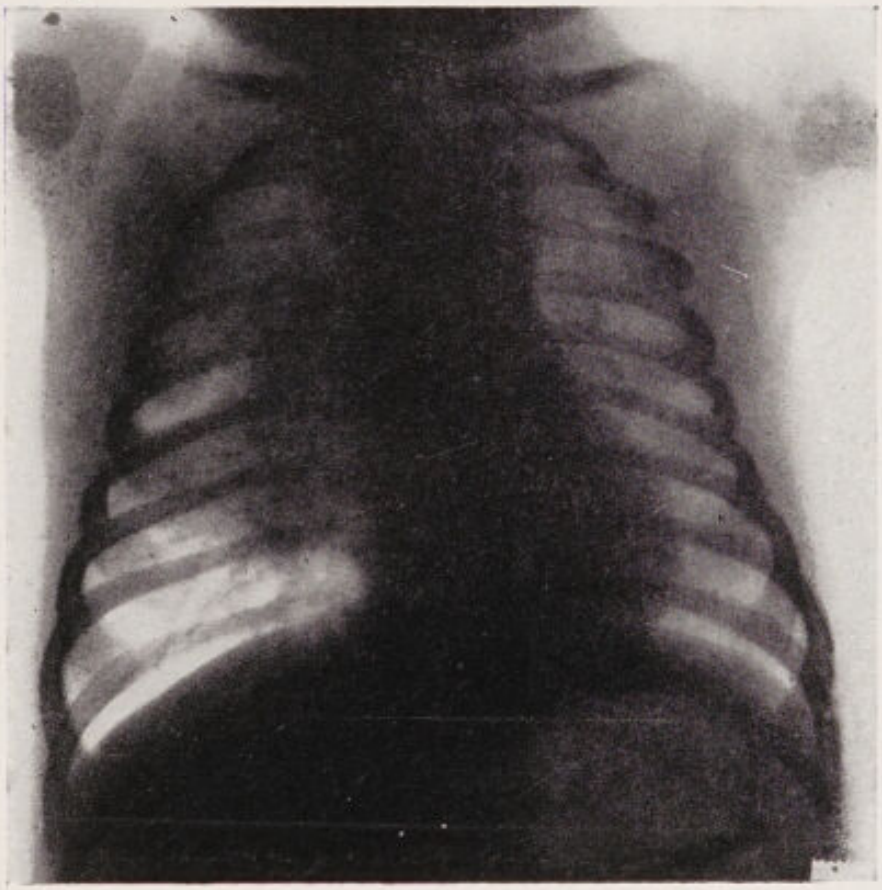
The Primary Infiltration and Complex.—Around the *primary focus* there occurs an exudation (*primary infiltration*), allergic in nature, which varies in area from the size of a pea to involvement of a whole lobe. Drainage from the primary focus takes place by the *lymphatic vessels* which lie around the bronchioles to the corresponding lymph-gland at the hilum; thus the *gland* becomes involved in the tuberculous process. This three-fold infection of lung focus, lymphatics, and gland is known as the *primary complex* (*Figs.* 11, 25, 55A–C). The primary complex impresses the pathologist as a lesion consisting of at least two circumscribed foci, lung and gland, which are caseous or calcified. The clinician, however, often sees the infiltration in the radiograph as a diffuse shadow, out of which the typical primary complex may emerge later, when the infiltration has resolved. This so-called *primary infiltration* is an exudation (non-caseous tissue reaction) around the primary focus; it is apparently due to diffusion of toxins from the focus into its neighbourhood, which elicits a hypersensitive exudation in the form of a perifocal toxic œdema or gelatinous infiltration (*Figs.* 23, 24). Clinically, a wrong impression of a primary infiltration may be created by atelectasis or by pleurisy. In many cases the infiltration is

not seen radiologically if it casts a very light shadow (*Figs.* 46, 47), nor can it be detected by physical examination; these cases are called "occult". On the other hand, there may be a very extensive infiltration which covers a whole lobe, sometimes more than one lobe; in infants it is particularly widespread (*Fig.* 2). The focus and gland may at first be invisible, if covered by a diffuse infiltration; the latter may present a sharp shadow or it may be indefinite, depending on its limitations; if it has contact with an interlobar fissure, it is clearly defined and difficult to distinguish from a resolving pleural effusion. At times, the infiltration forms two discrete shadows—a smaller one around the primary or Parrot-Ghon focus, and a larger and denser one around the hilar gland; a clear area of lung is seen between the two; this is known as the stage of bipolarity, and occurs as an early phase when the infiltration is just beginning to clear; focus and gland, each surrounded by a small infiltration, are seen distinct from one another (*Fig.* 26).

Incubation Period.—An interval of incubation occurs between the implantation of the tubercle bacillus (infection) and the development of allergy as manifested by a negative tuberculin skin response becoming positive. This period lasts from 4 to 8 weeks, in most cases 6 weeks (Wallgren).

Initial Fever.—At the end of the incubation period, the skin test gives a positive reaction; the appearance of allergy in a number of cases is accompanied by a rise of temperature, which varies from 99° F. in the evenings to high fever, reaching 104° F.; the febrile period lasts for 1, 2, or 3 weeks; usually 10 days. Wallgren has studied the initial fever of primary tuberculosis in cases accompanied by erythema nodosum, when the development of allergy and the appearance of the eruption coincide; he finds that initial fever is a reliable indication of a primary complex, which is established usually 6 weeks after exposure to infection.

Example.—Sylvester W., aged 3 months. Sister suffering from pulmonary tuberculosis. Admitted to hospital Nov. 19, 1938. Percutaneous test and chest radiograph both negative (*Fig.* 4A). Ran a temperature (*Fig.* 27) after 2 weeks and on Dec. 13, 1938, percutaneous test positive, and radiograph showed shadow in right hilar region, primary focus not seen (*Fig.* 4B). General condition remained poor, weight gain ½ lb. in 5 months. Ischio-rectal abscess and occasional vomiting. March 30, 1939: X-ray (*Fig.* 4C)—caseous pneumonia observed over the site of primary complex. On April 12, 1939, he developed measles and died three days later, aged 8 months, in a fever hospital.



FIGS. 23A, 23B.—PRIMARY COMPLEX FOLLOWED BY ASPIRATION PNEUMONIA.

Fig. 23A.—Joe McG. March 16, 1939; aged 3 months. Radiograph shows primary infiltration in right mid zone, with extensive periadenitis in right hilum. The actual primary focus is not seen.

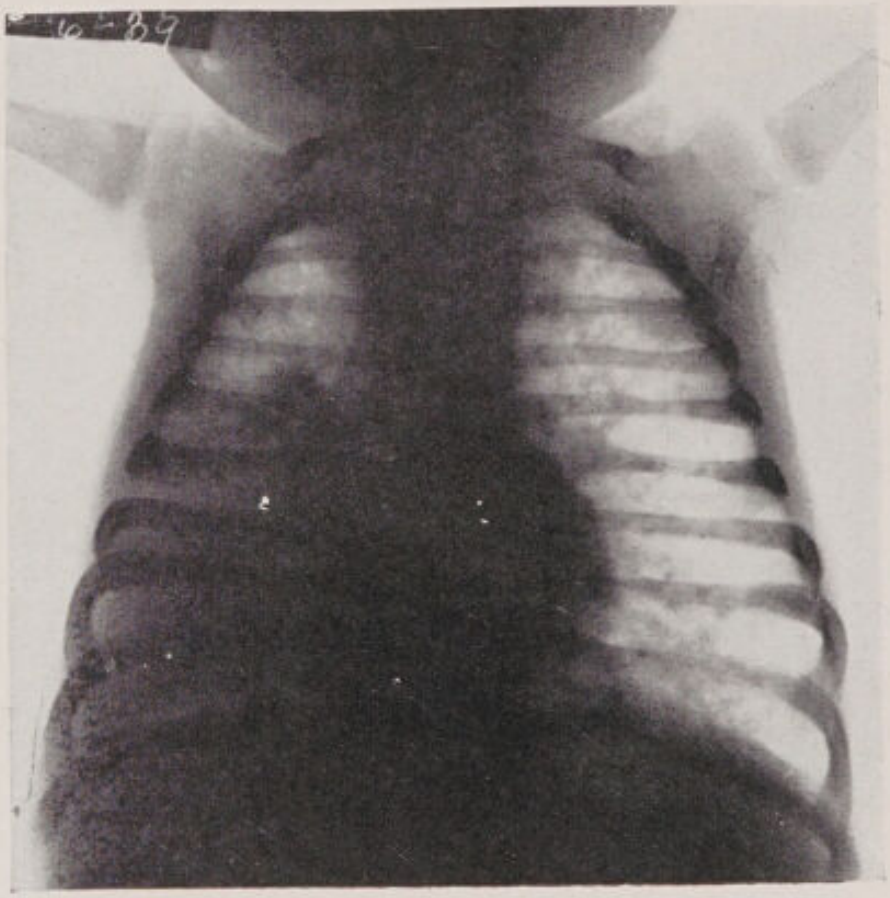


Fig. 23B.—June 1, 1939. Extension of disease, caseous pneumonia in lower and mid zones of right lung, and spread to left lung.

Clinical History.—

March 16, 1939. Aged 3 months. Admitted with positive Hamburger; contact phthisical young adult in the house. Condition became gradually worse, temperature 102-104° F., dull percussion note with rales over right lung, cervical glands involved.

June 1, 1939. Aged 6 months. Temperature 102-104° F.; adventitious sounds over both lungs, dullness over right.

June 28, 1939. Mors. Report on anatomical specimen by Dr. Pagel: "Typical Ghon focus in anterior aspects of right mid lobe with gross liquefaction. Massive caseation of lymph glands on right side. Coarse aspiration in right lower and finer aspiration in left upper and lower lobes. This is the product of eruption of a gland into a bronchus; the perforation is near the bifurcation on the right side."

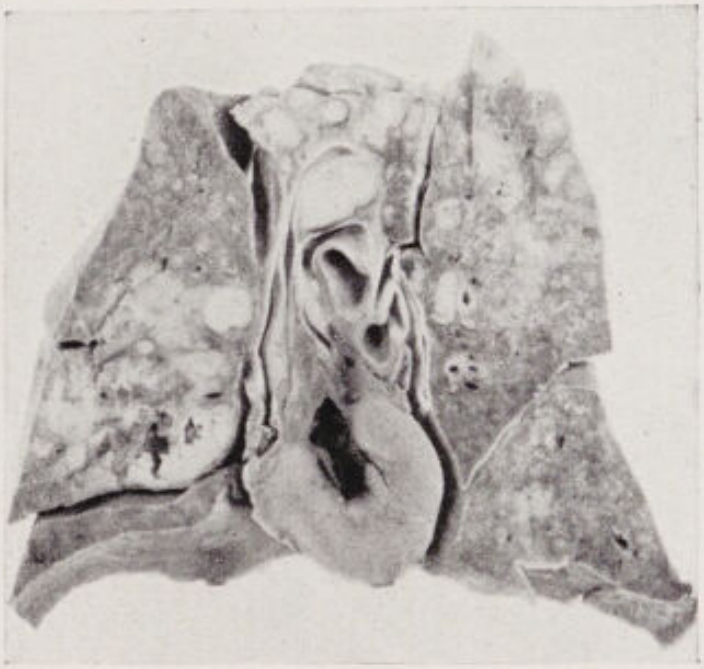


FIG. 24.—ANATOMICAL SPECIMEN FROM CASE SHOWN IN FIGS. 23A, 23B.

Primary infiltration with liquefaction, middle lobe. Bronchogenic spread on the left due to aspiration.
(*By courtesy of Dr. Walter Pagel.*)

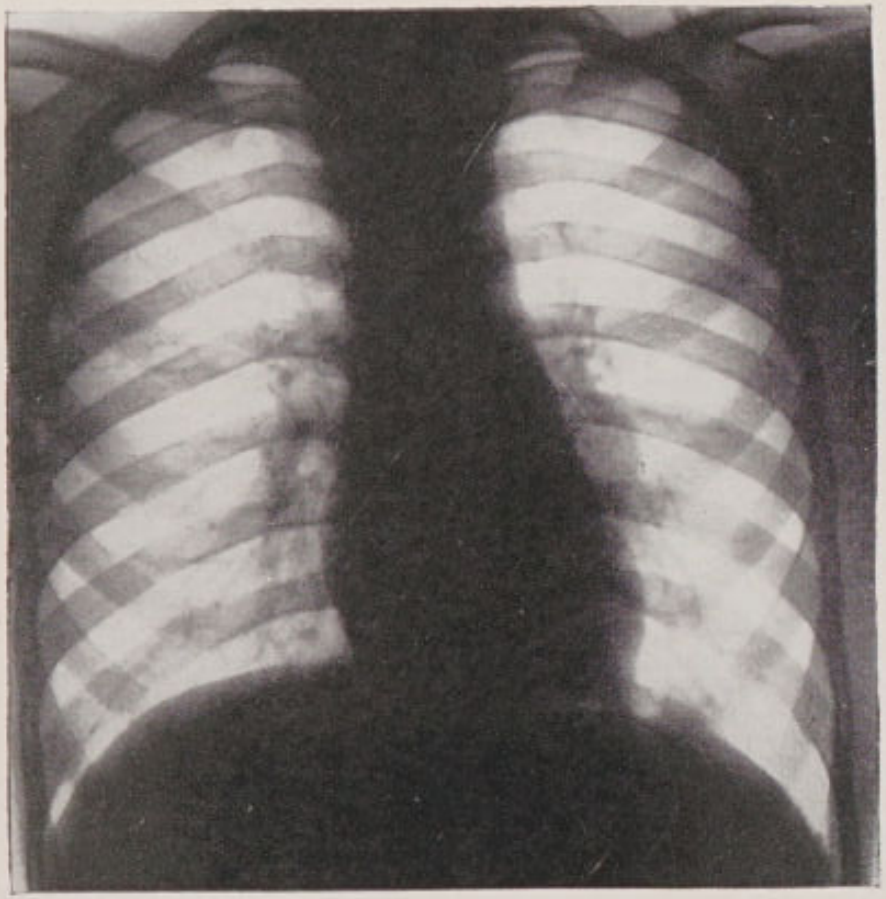


FIG. 25.—PRIMARY COMPLEX.

Michael B. Aged 10 years. Ghon focus seen on the diaphragm near the cardio-phrenic angle, and enlarged gland in lower hilum, on the left. Tuberculin test positive.

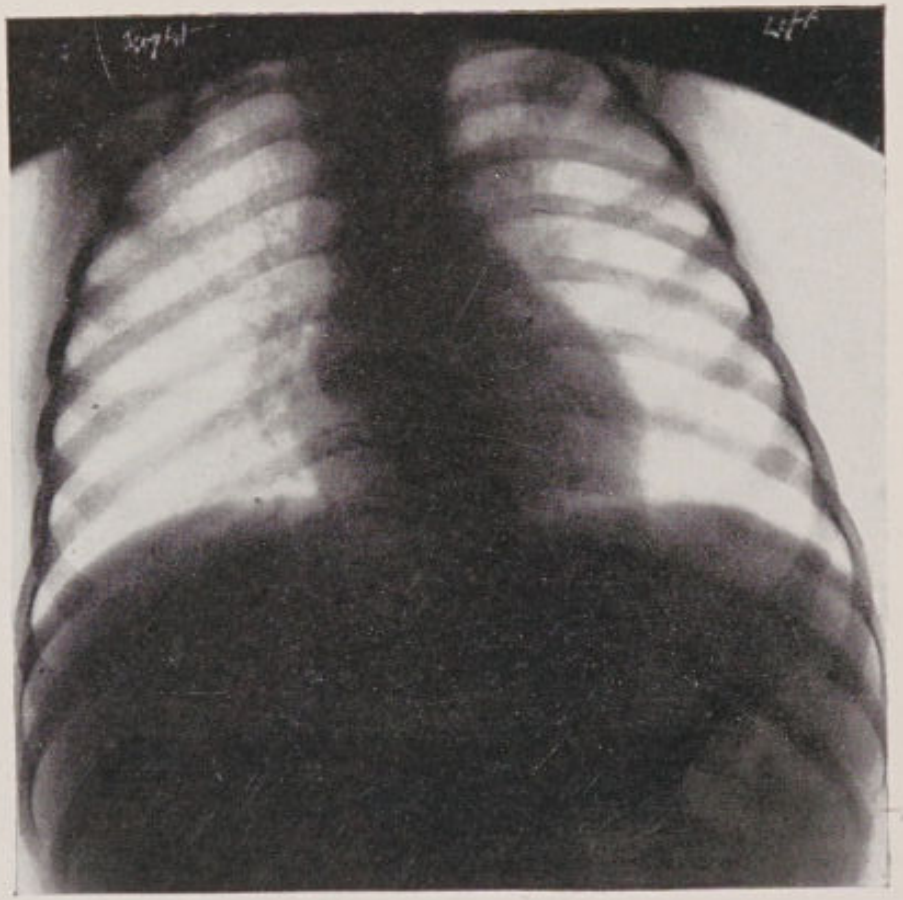


FIG. 26.—PRIMARY COMPLEX. STAGE OF BIPOLARITY.

James R. Aged 3 years. The primary infiltration round the focus is resolving, leaving focus and gland as separate entities (dumb-bell shadow), i.e., stage of bipolarity.

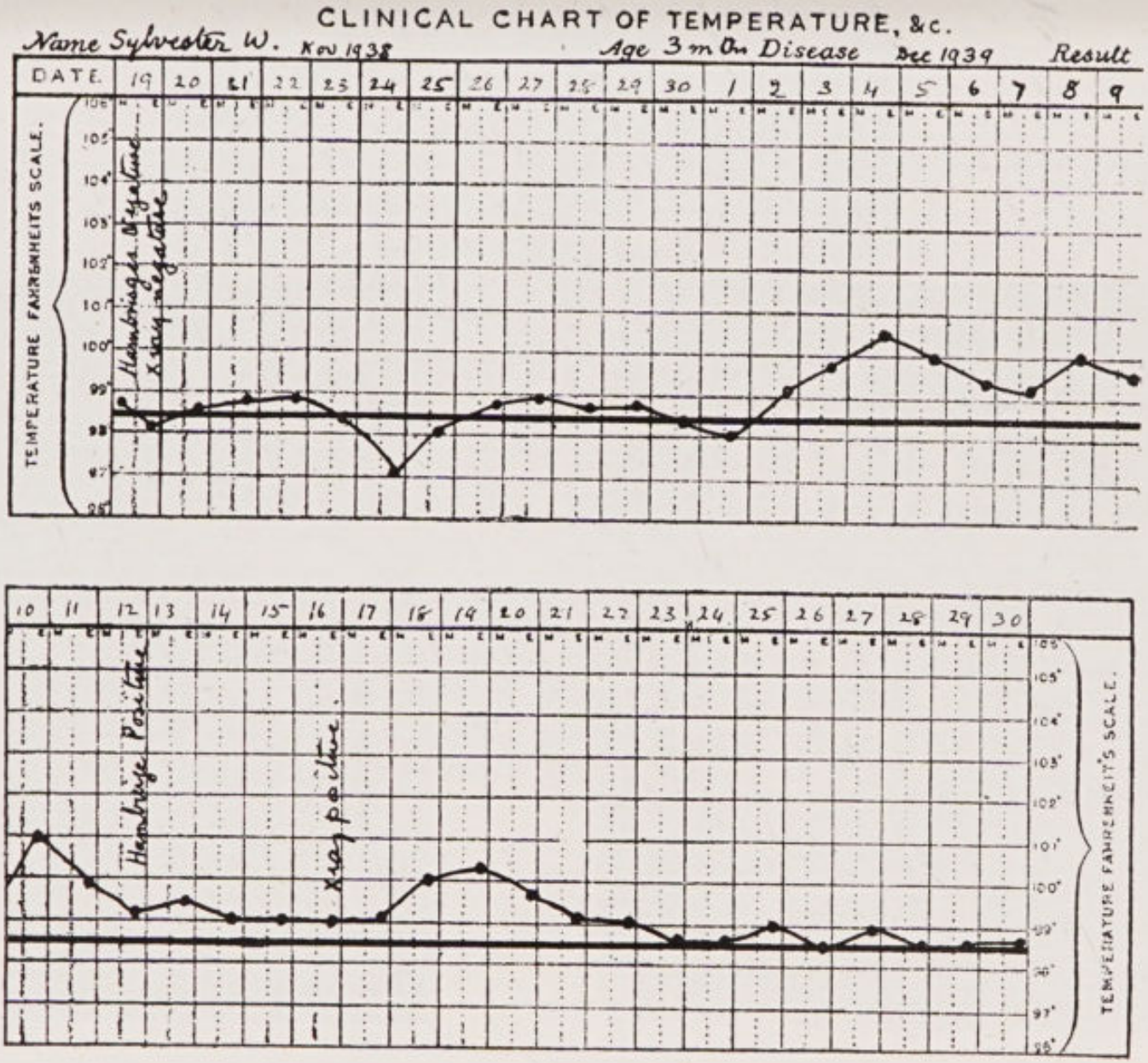
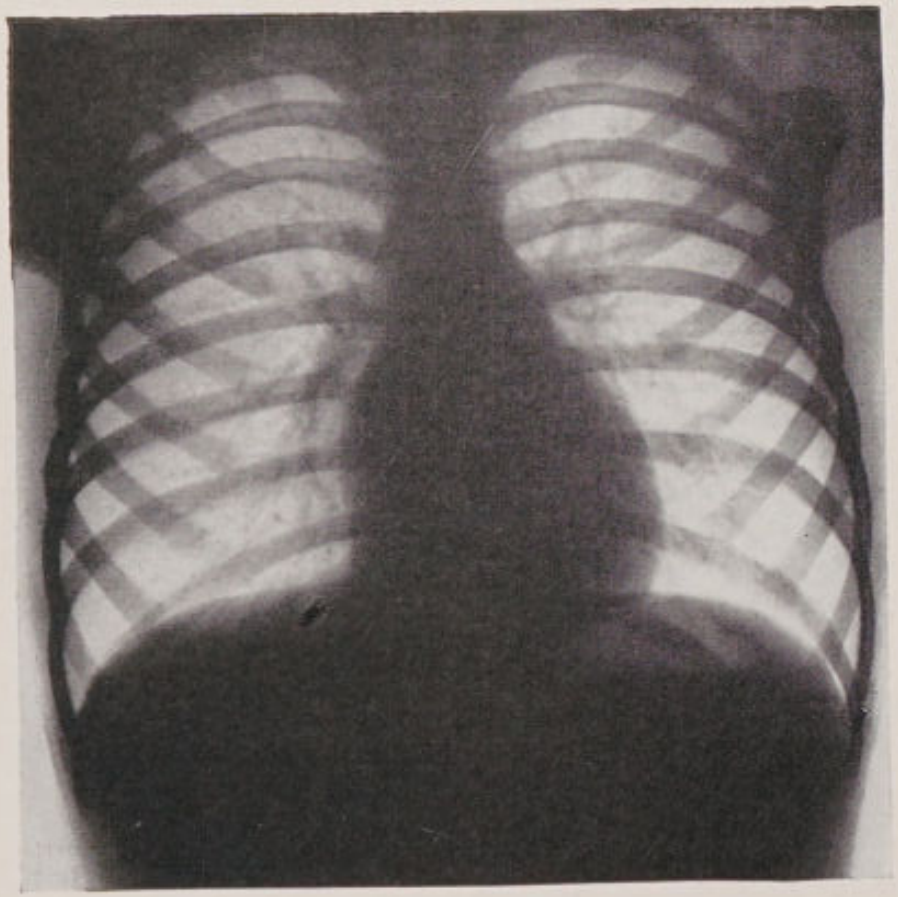
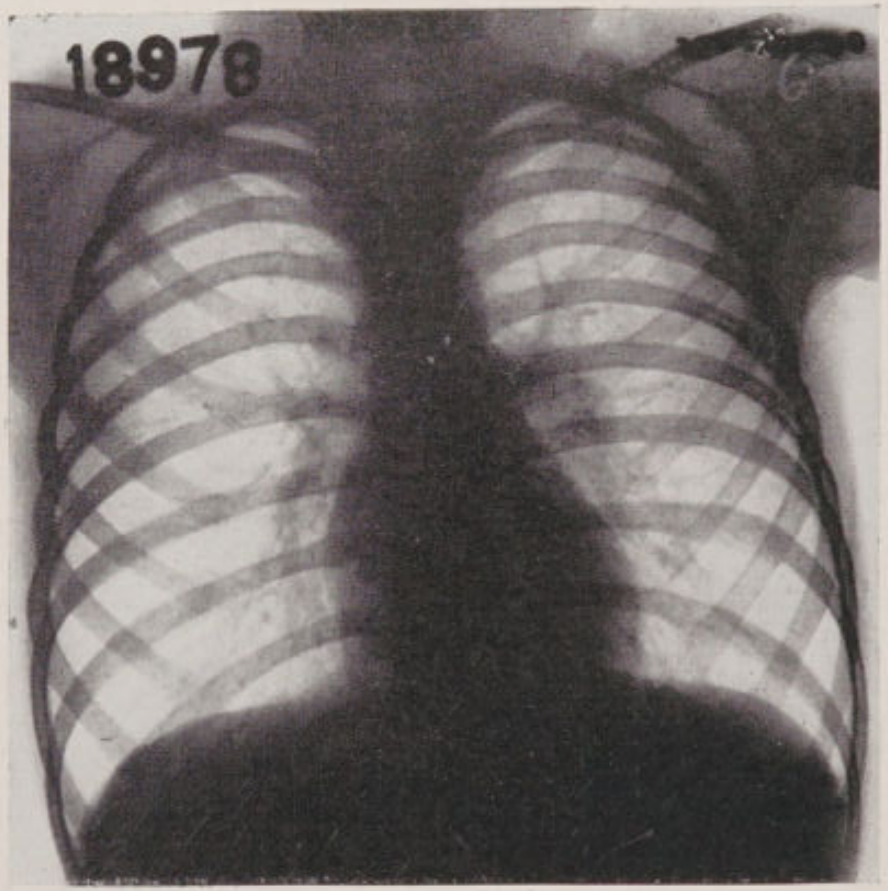


Fig. 27.—Chart showing initial fever in primary tuberculosis.

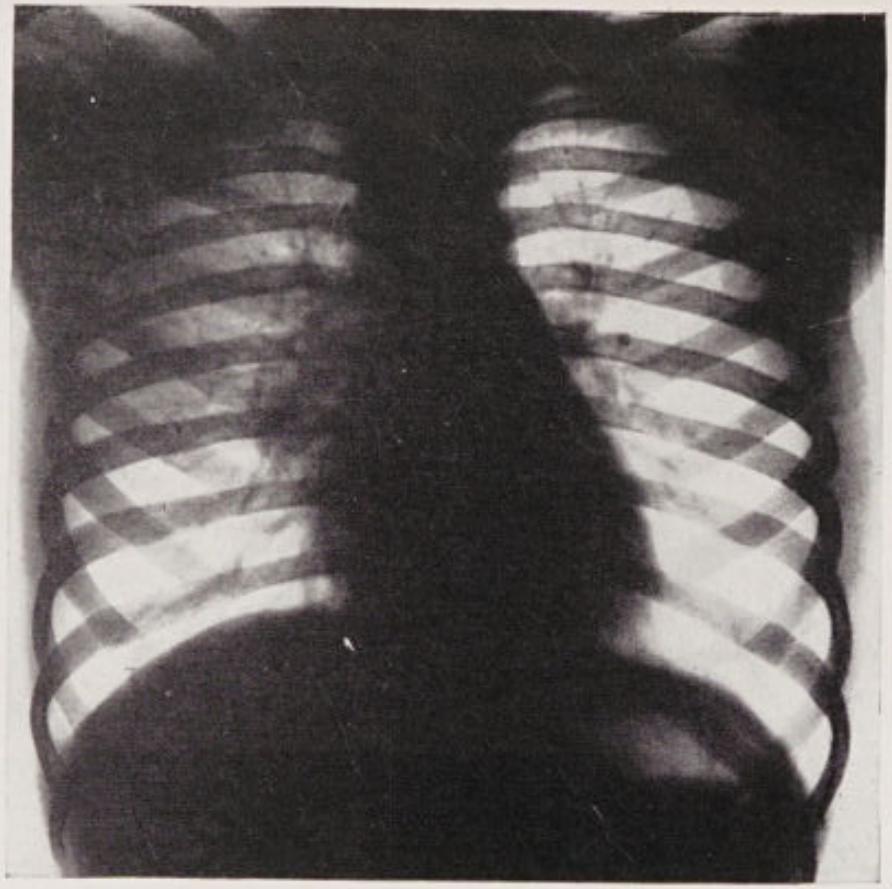




FIGS. 28A, 28B.—ERYTHEMA NODOSUM AND HILAR GLAND.

Fig. 28A.—Peter R. Feb. 7, 1939 ; aged 6 years. (*See Example*, p. 5).) Tuberculin test negative. Chest radiograph normal.

Fig. 28B.—June 15, 1939. Erythema nodosum. Tuberculin test positive ; radiograph shows enlarged gland in left hilum, primary focus not seen.



FIGS. 29A-29C.—ERYTHEMA NODOSUM WITH HILAR GLAND ENLARGEMENT.

Fig. 29A.—Berna K. Jan. 9, 1939; aged 8 years. (*See Example*, p. 51.) Right hilar gland enlarged, primary focus not seen. (The shadow lateral to hilum is due to scapula.)

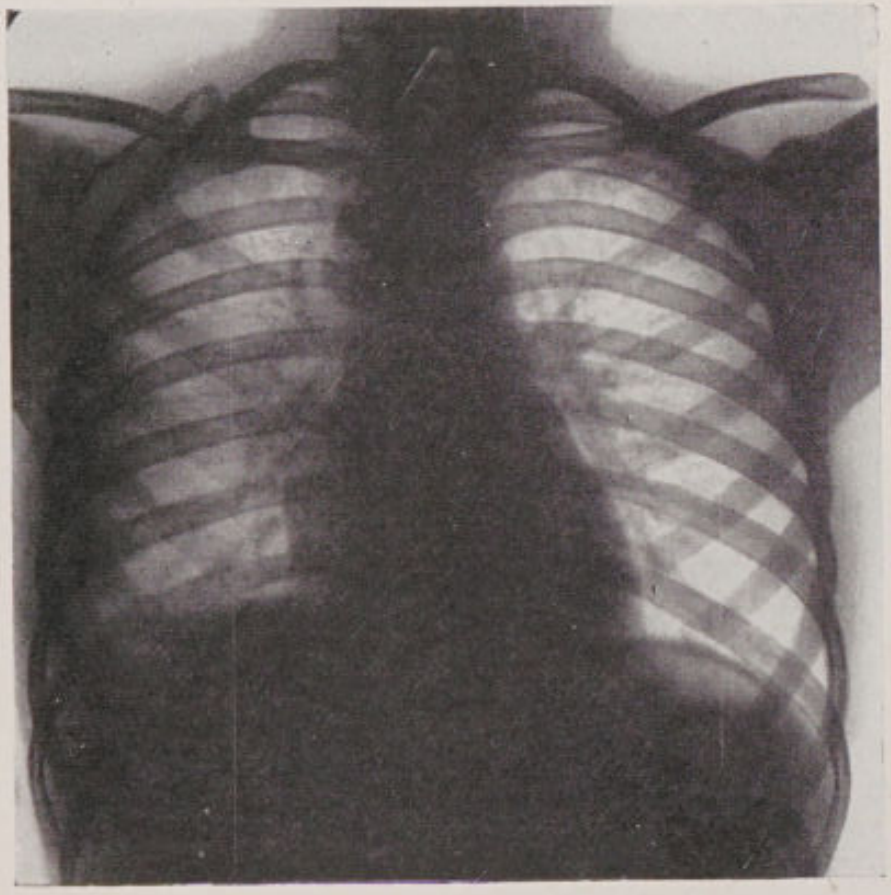


Fig. 29B.—April 13, 1939. Tumour-form shadow due to enlarged paratracheal gland. Pleural effusion in right costo-phrenic angle and axilla.

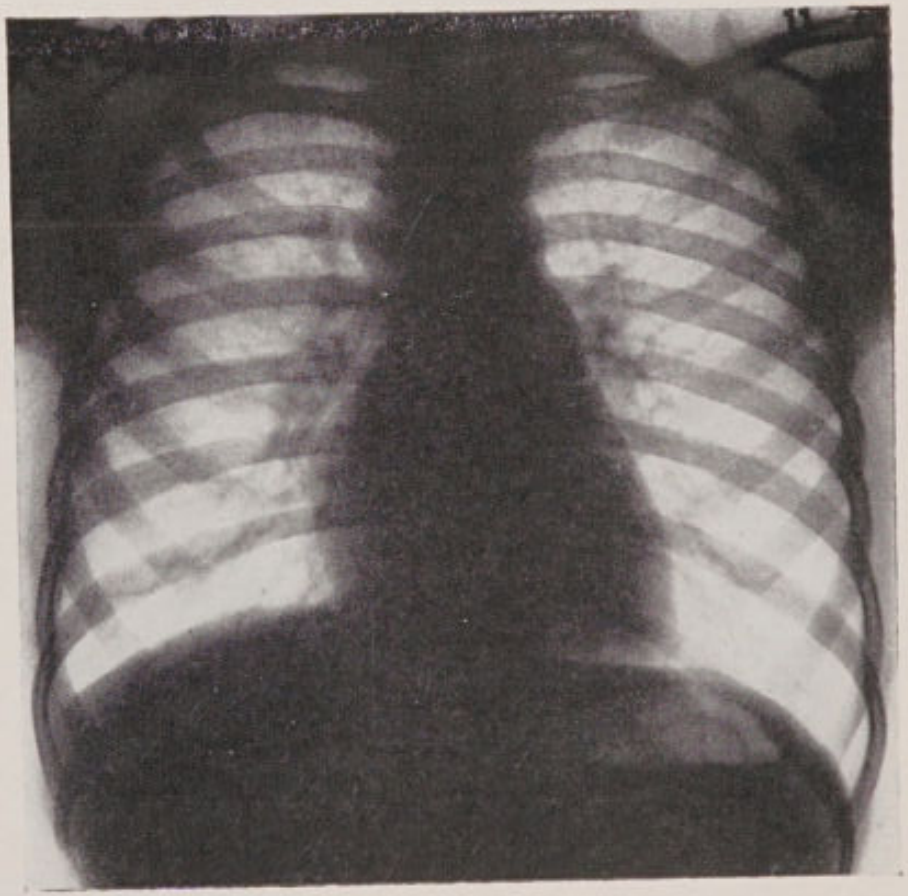
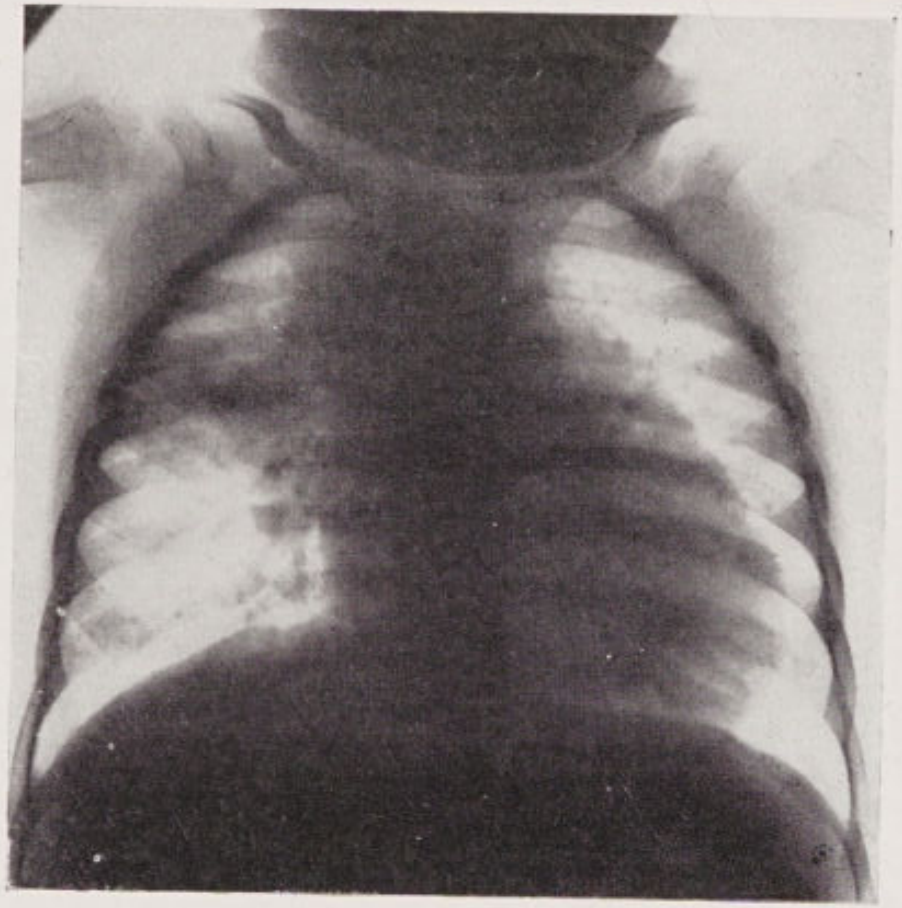


Fig. 29C.—July 11, 1939. Effusion cleared and glands organizing; right paratracheal gland still enlarged. Complete resolution later.



FIGS. 30A-30C.—ATELECTASIS.

Fig. 30A.—Brigid W. Sept. 10, 1936; aged 1½ years. (*See Example*, p. 52.) Partial atelectasis of right upper lobe, with enlarged right paratracheal and upper hilar glands.

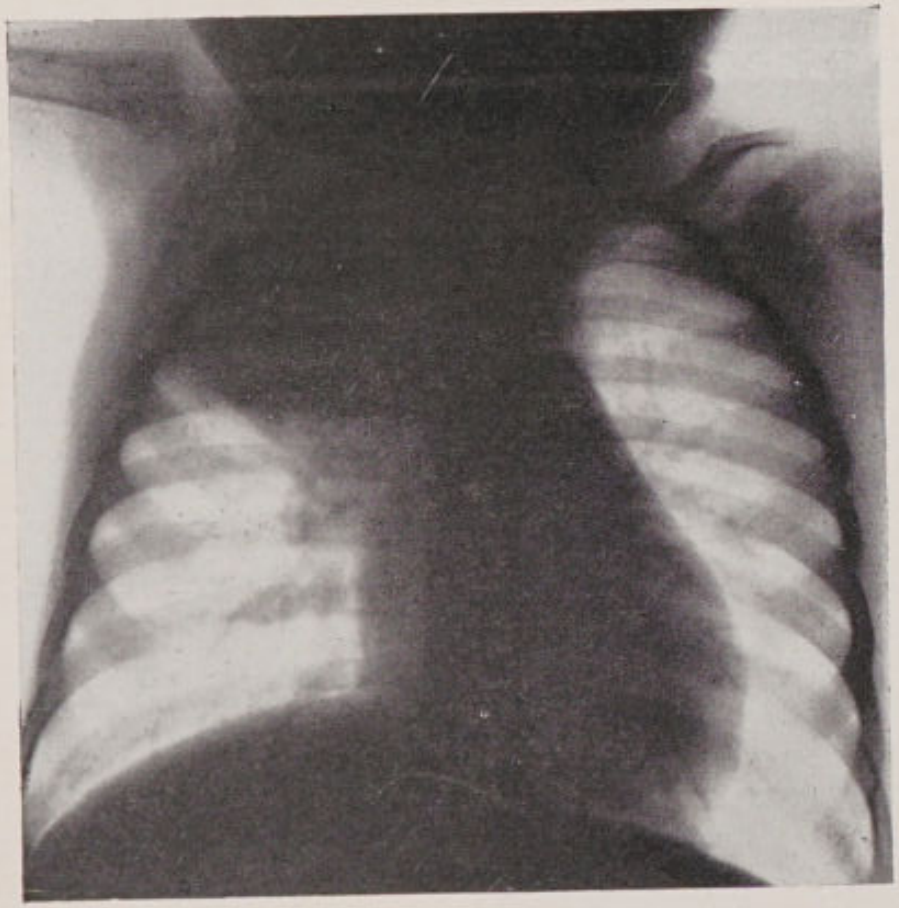


Fig. 30B.—May 4, 1937. Atelectasis of right upper lobe; the oblique position of lesser interlobar septum indicates reduction in capacity of upper lobe.

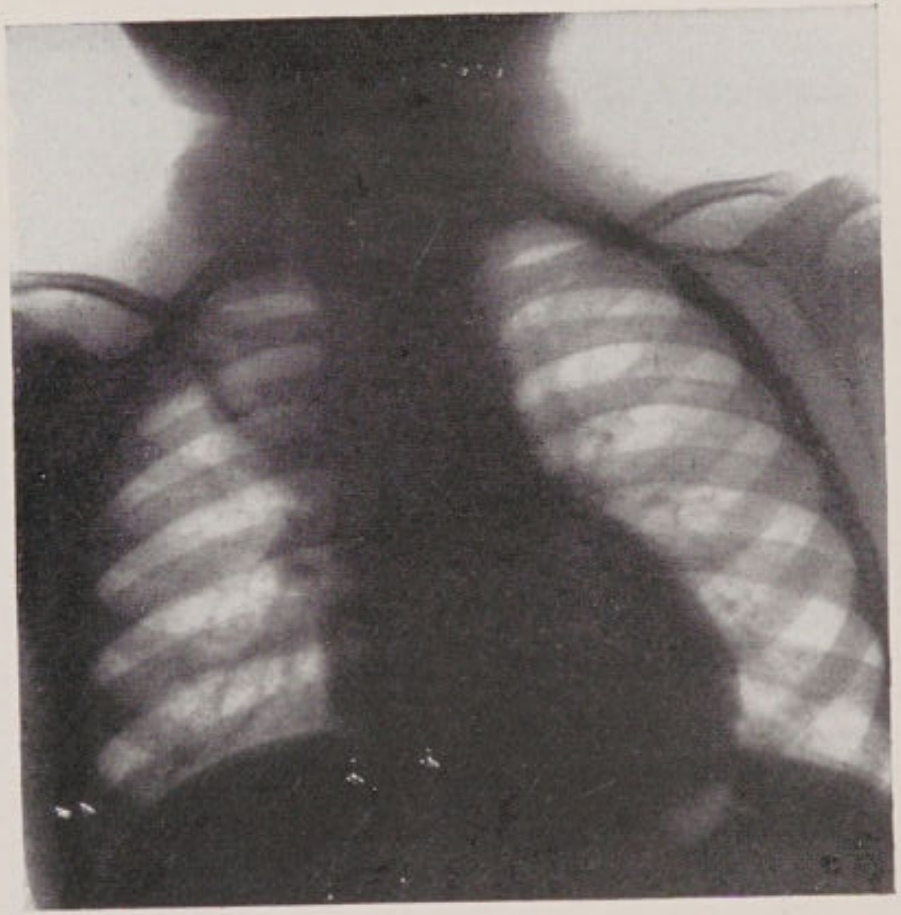
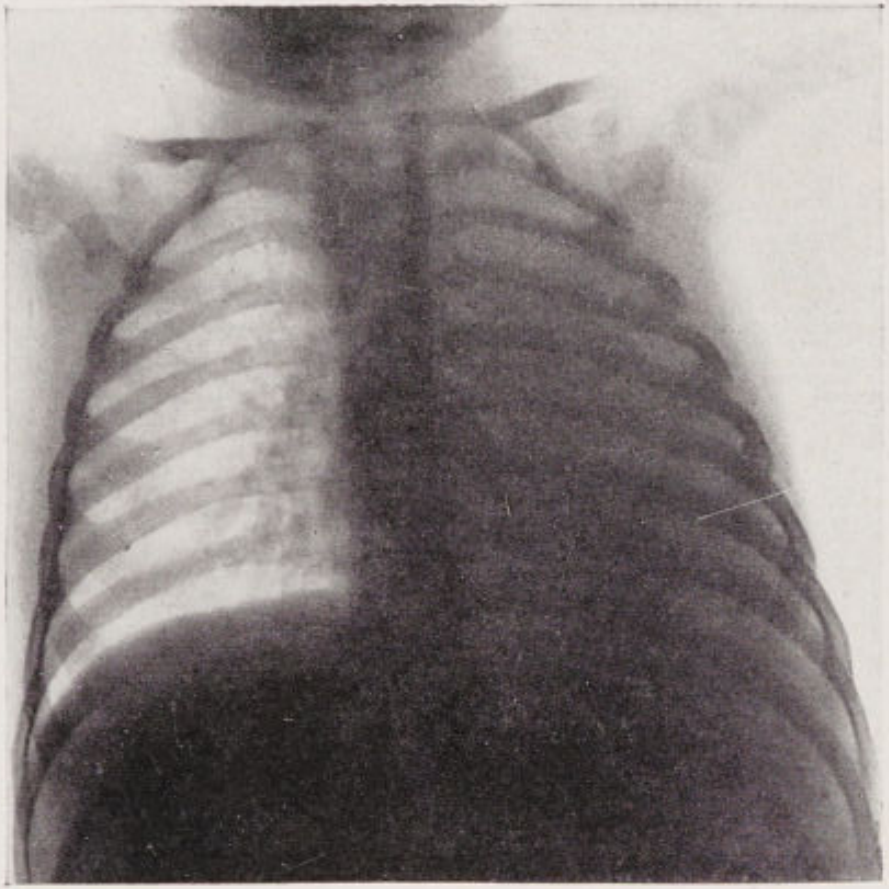


Fig. 30C.—April 11, 1940. Aged 5 years. Partial re-aeration of atelectatic area, and retraction due to fibrosis; some calcification in right tracheo-bronchial glands. Condition same one year later, calcification proceeding.



FIGS. 31A-31C.—ATELECTASIS.

Fig. 31A.—Patricia C. June 1, 1937; aged 16 months. (*See Example*, p. 52.) Atelectasis of whole left lung; the heart is drawn somewhat to the affected side.

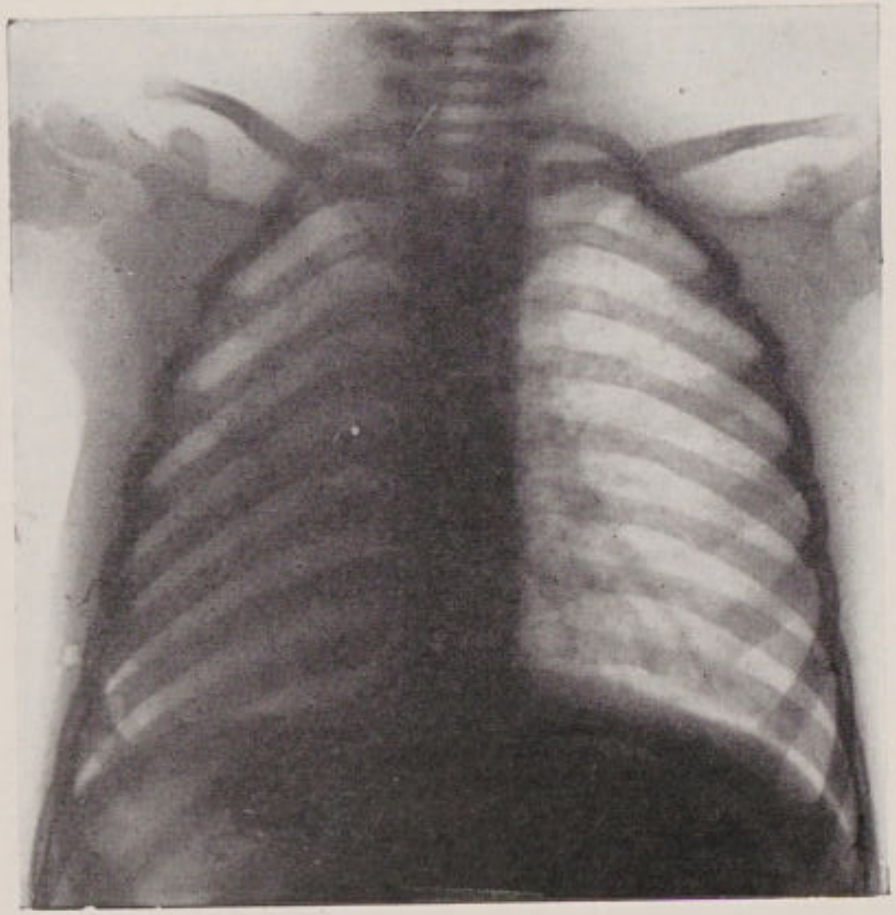


Fig. 31B.—March 8, 1939. Partial re-aeration of left lung. A large area of calcification is seen through the shadow, presumably a primary focus in lower part of left upper lobe. (This film is reversed.)

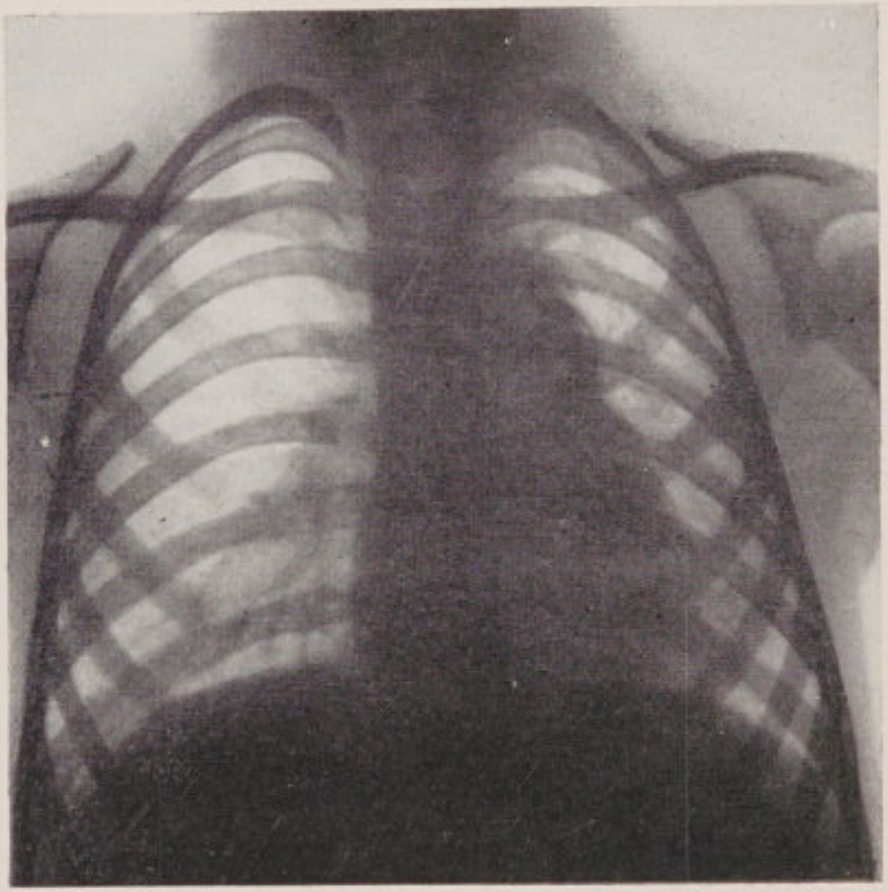


Fig. 31C.—Sept. 24, 1940. Aged 4½ years. Re-entry of air almost complete. The calcified area appears to be a primary focus with its regional para-aortic gland (also calcifying) lying between it and the vertebral column. A second primary focus is now recognized by its calcium deposit in left lower lobe; this may be a sub-primary focus. Alternatively the large calcified mass seen on the left heart border (which has a smooth encapsulated appearance) may be a para-aortic gland; in this event one would expect the primary focus to be in the left apex.

Multiple Primary Foci.—Multiple primary foci can occur in the lungs, but are not at all common (*Fig.* 31C).

Post-Primary Foci.—Post-primary foci are those which occur at any time after the primary complex is established, and include re-infection foci of the Assmann type. Foci are called 'sub-primary' if they occur immediately after the primary focus and before allergy is established. But the general rule is for *post-primary foci* to occur during the allergic state, and then they have no glandular response.

Erythema Nodosum.—Erythema nodosum is an inflammatory condition involving the cutis and subcutis. On successive days crops of bluish-red nodes appear, chiefly situated on the shins and dorsum of the foot; rarely they appear also on the arms, calves, and occasionally all over the body. The eruption consists of few or many nodes which are firm, hot, and painful to the touch; in many cases there is accompanying fever, ranging from normal to 104° F., malaise, and pains in the joints of the lower limbs, or occasionally in all joints. The nodes fade gradually in a week or two.

These nodes must be regarded as an allergic response to toxins. The toxins may be due to several causes, but by far the most common is that produced by the tubercle bacillus during its first implantation in the human subject. Occasionally these nodes are associated with rheumatic conditions or with sulphonamide poisoning; tubercle bacilli, however, are responsible for the majority of cases although the bacillus has never been demonstrated in the nodes themselves. Rheumatic erythema nodosum has been known to recur annually. Collis (1933) found that in London 27 of 38 cases of erythema nodosum were tuberculous, 7 streptococcal, and 4 indefinite. Wallgren (1935) in Sweden found that 96 per cent of 800 cases gave a positive tuberculin reaction; also amongst 300 children suffering from erythema nodosum he found radiological evidence of intrathoracic tuberculosis in 90 per cent of the 1–5 age group, 85 per cent of the 5–10 group, and 56 per cent of the 10–15 age group. Price (1945) in Dublin found that out of 36 cases of erythema nodosum which all reacted positive to tuberculin, 35 were suffering from primary tuberculosis and 1 from acute rheumatism. Wallgren was the first to point out the true nature of erythema nodosum; he showed that the skin eruption makes its appearance simultaneously with the first manifestation of primary tuberculosis, coincides with initial fever when present, and is

connected with the allergy which is developed at that time. This clinical observation indicates that erythema nodosum is an important diagnostic sign in the detection of a number of cases of primary tuberculosis at their inception. The incidence of erythema nodosum varies in different countries, being common in Scandinavia, less common amongst dark-complexioned peoples. Girls are affected in much greater numbers than boys. Daniels (1944) in England reports 5 cases (1·7 per cent) of erythema nodosum amongst 285 nurses during Mantoux conversion. Price (1945) in Ireland found amongst 31 girls aged 14–18, who developed primary tuberculosis whilst under observation, that 4 (12·9 per cent) had coinciding erythema nodosum ; and also found that 18 out of 35 cases of erythema nodosum had radiological evidence of primary or root-gland tuberculosis ; 3 of these were nurses whose films had been negative a short time previously. Although usually associated with pulmonary primary infection, erythema nodosum has also been observed during the development of primary tuberculosis of the cervical glands.

Local treatment is unavailing. General treatment for primary tuberculosis should be instituted immediately. Even when the chest radiograph is clear, one month's bed-rest should be given, followed by three months' part-time rest ; in severe cases with radiologically demonstrable lesions, both these periods should be extended. There should be subsequent supervision for a further year, for pleurisy has been known to develop 14 months after an attack of erythema nodosum. There is no specific treatment for erythema nodosum. Price reports a small series of results with sulphatherapy : 15 out of 36 cases got sulphonamide (10 g.) within 3 days of the appearance of the nodes, 1·5 g. per day ; 21 got no sulpha treatment ; similar rest-therapy and after-care was carried out in both groups. Of the sulpha-treated cases none had any subsequent trouble, with the exception of one pleural effusion ; the nodes faded rapidly and fever where present subsided. Of the 21 controls, 9 cases had subsequent trouble, viz., 4 pleural effusions, 2 prolonged illness associated with the eruption, 2 had to undergo sanatorium treatment before the primary lesions healed, and 1 developed skin tuberculides.

Example.—Peter R., aged 6 years. Chest radiograph and tuberculin test negative on Nov. 8, 1938, and on Feb. 1, 1939 (*Fig.* 28A). Erythema nodosum accompanied by fever appeared in June, 1939. Tuberculin test became positive. Radiograph June 15, 1939, (*Fig.* 28B) shows enlargement of gland at left hilum, primary focus not visible in film.

Example.—Berna K., aged 8 years. Jan. 9, 1939, skin test positive, erythema nodosum and fever. Radiograph shows heavy shadow in right hilum, no focus seen in lung field (*Fig.* 29A). April 14, 1939, radiograph shows small pleural effusion, some activity in each hilum, and tumour-form shadow over lower paratracheal glands on right (*Fig.* 29B). July 11, 1939, radiograph shows pleural effusion gone, hilar glands reducing, and paratracheal gland still enlarged (*Fig.* 29C). Later complete cure.

The eruption may occur in the form of a mild epidemic when a case of open tuberculosis comes into close contact with a number of child or adolescent negative tuberculin reactors, as in a school class (Landau) or factory (Drum).

Example.—Drum reported 5 cases of erythema nodosum which occurred within a short interval of time amongst young women aged 16 to 22 years in a factory in a country town in Ireland. These cases all reacted positively to Mantoux 1–1000, but no definite focus was found on physical examination. The tuberculous infection was traced to an expert who had come from across the water for a short period as overseer in the factory, and who was subsequently found to be suffering from open phthisis.

Post-Primary Erythema Nodosum.—Post-primary erythema nodosum has been described by Wallgren. This occurs in cases which are already known as positive reactors suffering from primary pulmonary tuberculosis. After the reduction of allergy by some inter-current infection such as pharyngitis in a patient, the eruption appears when allergy returns; thus the tuberculo-protein is able to influence the hypersensitivity of the individual as though it were a fresh infection. A certain proportion of these cases have already had a previous eruption of erythema nodosum associated with the initial fever. They are rare.

Example.—Bernard L. (*See* p. 159.)

Atelectasis.—During the course of primary tuberculosis, a condition of atelectasis arises in any area of lung to which the entry of air is impeded; this may occur in two ways. A bronchus may be plugged by caseous material from a gland, or the lumen of a bronchus may be occluded by external compression on its walls by a primary focus or an enlarged gland; in either case there will be resultant cessation of aeration in the portion of lung which is supplied by that bronchus (*Figs.* 14, 18). In young children especially are the bronchi readily compressible. Atelectasis is a condition which is itself benign, but it is said that bronchiectatic changes in the affected

portion of lung may result. In course of time, with dispersal of the plug, or shrinkage of the pressing gland, the bronchus once more becomes patent, and air re-enters the alveoli. Restitution may take 2 months to 2 years before it is complete; it is especially prolonged in the case of gland pressure. Atelectasis appears at varying intervals after the establishment of the primary complex; the shadow may be seen radiologically even after the primary focus has healed; at times calcification of gland and focus may be seen emerging through the atelectatic shadow, thus confirming the diagnosis of occlusion of a bronchus from without. The author finds that re-aeration of the lung in these cases takes place after rest therapy, without bronchoscopic interference; lipiodol investigation is not recommended unless there is some distinct advantage to be gained by the introduction of such a substance into the air-spaces of a primarily infected unhealed lung.

Example.—Bridget W., aged 1½ years. Sept. 10, 1936: admitted to hospital. Tuberculin test positive. Child in fair condition, Radiograph shows partial atelectasis of right upper lobe (*Fig.* 30A). May 4, 1937: radiograph (*Fig.* 30B); sudden severe cardiac distress. with loud murmur over whole base of the heart; pulmonary stenosis, apparently due to gland pressure; remained very ill for three months. Oct. 1, 1938: left hospital in good health, pulmonary murmur less marked. Jan. 17, 1939: cardiac murmur still audible. In very good health. April 11, 1940: radiograph shows atelectasis still present but calcification commencing in the right paratracheal gland (*Fig.* 30C). Child very well, attending school.

Diagnosis of Atelectasis.—On percussion the note is more dull than that of an infiltration, but less so than in pneumonia; on auscultation the breath-sounds are diminished and occasionally a few rales are heard. Radiological appearances are discussed on pp. 33, 37. Bronchoscopy may give information as to the nature of the occlusion during the atelectatic period, whether by plugging or by pressure. In case of plugging a portion of caseous material may be extracted during bronchoscopy. Subsequent lipiodol radiography will help to demonstrate bronchiectatic changes, after the primary lesion has healed.

Example.—Patricia C., aged 16 months. Admitted to hospital June 1, 1937. Mother dying of phthisis. Tuberculin test positive, and atelectasis of entire left lung. General health fairly good, S.R. 17. Temperature normal. Left lung showed dullness on percussion and diminished breath-sounds on auscultation. July 1,

1937 : radiograph the same as on June 1, 1937 (*Fig.* 31A) and shadow persisted for months. Bronchoscopy revealed left main bronchus occluded by pressure from without, but failed to relieve it. She was kept off her feet in hospital for twelve months ; then a calcified area was seen to be appearing through the shadow in the upper zone outside the left hilum. During resolution she developed two small conjunctival phlyctens in the left eye. March 8, 1939 : radiograph showed partial re-aeration of the periphery of the lung and a large area of calcification beneath the atelectatic shadow (*Fig.* 31B). After 18 months' treatment she was discharged from hospital ; physical signs had disappeared but the shadow had still not cleared. General health excellent. Sept. 24, 1940 : two calcified areas are seen in lung field, probably both primary foci (*Fig.* 31C). June 1, 1945 : health good.

Epituberculosis.—The word ' epituberculosis ' has crept into the terminology of childhood tuberculosis. No two persons are agreed as to its exact meaning. The term generally conveys to the mind the idea of a condition which runs a benign course and which is associated with primary tuberculous infection of the lung ; it is recognized radiologically by a shadow which covers a lobe or part of a lobe, a shadow which remains unchanged for months, eventually disappearing without tissue destruction. Clinically there are found dullness and bronchial breath-sounds over the affected area ; there are few, if any, signs of disturbed health.

The word was originally coined by Eliasberg and Neuland in 1920, to describe a lung infiltration seen by them in certain cases of tuberculous infection in very young children ; these patients showed a heavy shadow in the radiograph covering the right upper lobe ; there was no acute onset to the disease, and the course was benign, almost without symptoms ; there were no tubercle bacilli in the sputum. The authors make no mention of atelectasis in their articles, but they describe bronchiectasis as a possible sequel. In their second paper (1921) they differentiated more fully between epituberculosis and early caseous pneumonia, the latter having an acute onset, high fever, and rapid fatal termination.

Pathologists since that time have produced evidence as to the nature of this so-called ' epituberculosis '. The material for their consideration has been scanty, owing to rarity of deaths. Recent pathological views may be summarized as follows :—

a. Rössle in 5 cases found collapse, caused by glands or primary focus.

b. De Bruin found atelectasis and bronchiectasis in 1 case.

c. MacGregor and Alexander found alveolar infiltration with epithelioid and giant cells.

d. Pagel, and Pagel and Fish, report 3 cases and summarize very clearly the existing evidence ; Pagel definitely distinguishes between atelectasis due to pressure and the condition of non-caseating tissue reaction, which is subject to healing by absorption, with epithelioid and giant cells in the exudate, but no tubercle bacilli nor caseation and tissue destruction. He finds it is due to aspiration of dead or scanty living bacilli into allergic lung tissue. The aspiration often comes from caseous plugs in the bronchus. Therefore the non-caseating tissue reaction may be combined with atelectasis due to plugging of a bronchus.

By the findings under (*c*) and (*d*) it is clearly shown that there is a pathological condition deserving of a special name.

On the clinical side, varying views have been expressed. Spence found tubercle bacilli in repeated lung puncture of a case of clinical 'epituberculosis'. Other observers have failed to do so. Burton Wood finds the chief factor to be atelectasis due to bronchial compression, with a secondary factor in alveolar exudation ; he suggests that the term should be now abandoned. Clinically we must assume by the knowledge of primary tuberculosis which has been acquired since 1921, that at least two conditions exist which will produce a shadow over the lung during the course of primary tuberculosis in the child : (1) A primary infiltration ; (2) Atelectasis.

It would be desirable for the clinician to be able to differentiate further between pure atelectasis and atelectasis combined with non-caseating tuberculous tissue reaction. This, however, is clinically impossible. Primary infiltrations tend to disappear in a short time, but a shadow which persists for months must raise a very strong suspicion of obstruction to air-entry, especially as these cases are seen most frequently at the age when the bronchi are readily compressible, and at a stage in the disease when clinically the child is past the acute initial phase associated with primary infiltration. Pagel has shown beyond doubt that the obstruction of the bronchus can be located at the site of the primary focus as well as at the hilum.

The word 'epituberculosis' conveys to some people the idea of the allergic state as a whole which accompanies in varying degrees of severity the primary tuberculous infection of the

lung in childhood. This is manifested by such phenomena as non-specific infiltration of the lung, erythema nodosum, the conjunctival phlycten, pleural effusion. But these features are not mentioned in the original definition of Eliasberg and Neuland.

The term 'epituberculosis' is, therefore, in many respects unsatisfactory, and it would be advisable for clinician and radiologist to cease to apply this term in a loose and general manner to several kinds of pathological conditions which show a lung shadow in the radiograph during the stage of primary tuberculosis.

Primary Cavities.—Occasionally the primary focus fails to heal, and in the centre of the caseous area liquefaction may occur; this liquefaction leads to a cavity which is thin-walled and may be large or small. Small primary cavities, as observed radiologically, may get smaller or disappear suddenly, in the course of two or three months, leaving behind the shadow of the primary infiltration in which they had been observed, which infiltration also gradually disappears in the course of time; large cavities are less likely to heal. Such cavities were reported amongst Viennese infants during the starvation years 1918-20 by Ghon. They have been observed in Dublin infants during two recent periods, 1942 and 1946: small cavities closed on the administration of large doses of calcium and vitamin D, whilst Alston noted success in such cases by a short period of immobilization on a spinal frame; for these children are not very ill, and will not remain quiet. By these measures primary cavities which were small and apparently very early were successfully closed. Primary cavities also occur in young adults.

Example.—Ann B., aged 7 months on admission to hospital; she had a shadow over the left upper zone; S.R. 49 mm.; she was diagnosed as a simple primary complex, although no gland shadow was visible. Contact was her aunt, a young girl, who lived in the same house. She began to lose ground, and 5 weeks later commenced to vomit; this continued on and off for 3 more weeks; she then showed dried blood in the vomit; four days later, aged 9 months, she died. Autopsy revealed a large primary cavity in the left upper lobe, filled with blood; the regional gland was very small in comparison with the size of the liquefied primary focus, and no other glands were involved. No tuberculous foci were found elsewhere in the lungs. At no time was this cavity visible in the radiographs. Death here was due to hæmorrhage from a primary tuberculous cavity.

Fate of the Primary Complex.—

Healing	*Non-healing*
By fibrosis and calcification (visible or invisible to X rays) *a.* Immediate *b.* Delayed	1. Caseation, liquefaction, and cavitation of primary focus *a.* May heal *b.* Death from toxæmia or hæmorrhage *c.* Acute pneumonic phthisis 2. Involvement of other mediastinal glands 3. Hæmic spread from mediastinal glands (via thoracic duct) 4. Hæmic spread from primary focus or gland 5. Rupture of caseous gland into a bronchus, giving rise to aspiration caseous pneumonia

The Clinical Picture of Primary Tuberculosis of the Lungs.— Over and over again it happens that a child passes through its primary tuberculous infection without parents or physician suspecting what has taken place. In many cases, even if the condition is suspected, physical signs and radiological evidence may be lacking; in such cases the only sure proof lies in the change from a negative to a positive tuberculin test. Clinically occult cases heal without visible calcification, thus leaving no apparent residue, although pathologically a small healed focus and gland are present; if the child's resistance is good, these occult cases often give no further trouble. But it also happens in occult, as well as in recognized, cases that within three months to one year after the primary infection, there is complaint of a painful hip which shows on investigation tuberculous changes, or of a headache which persists and which develops rapidly into tuberculous meningitis. A number of cases of primary pulmonary infection will present manifestations of ill health in varying degrees; the child is not well, he is off his food, listless, tired, for no obvious reason. Again, some cases show a very marked illness picture, with high fever lasting some two or three weeks, and typhoid fever or septicæmia may be suspected. The early stages of the primary infection are more easily recognized on account of the initial fever than are the later stages when the temperature has settled; in both stages the child hardly ever presents symptoms pointing to lung involvement, but frequently complains of stomach ache, headache, fatigue.

Diagnosis.—It is not easy to differentiate between primary tuberculosis and many other childish disorders; the signs of anorexia, lassitude, anæmia, and cough are common in many complaints of childhood. The tuberculin test in these cases often reveals the most unexpected results; a sick-looking pale child will have a negative test and be suffering from thread-worms; a child with a bad cough is usually suffering from bronchitis or bronchiectasis and has no tuberculous lesion; a child complaining of stomach ache has an atelectasis or tuberculous mediastinal glands; another child faints at school and is found to have a tuberculous primary infiltration of the lung. The more one sees of these children, the less one relies on clinical evidence, but bases one's diagnosis solely on positive skin test and positive radiograph. An inactive case of primary tuberculosis in the healing stage often presents a healthy appearance. If, however, the child is in an early hypersensitive phase with perifocal infiltration, the picture will be different. This child walks languidly into the clinic, looking pale and standing badly. His mother will complain of his disinclination to play, stating that on return from school he is too tired to eat his dinner, has at all times a bad appetite, and wants to sit about in a chair; in short he does not behave as a normal healthy child should.

How do we set about the exclusion of primary tuberculosis in such a case? Physical examination may reveal no definite signs in the chest; signs if present are never pathognomonic of tuberculosis; absence of signs is no contra-indication to tuberculous infection. A tuberculin test should be performed at once, and read in two days' time; it may equally well be read a week later. If negative, a stronger test may be given and read in two days' time. Meanwhile a radiograph of the chest should be taken; but if this is not convenient, as for instance if the child lives in the country, one may wait to see if the test is positive before insisting on X rays. This whole examination can be done inside a week, and in nearly all cases a diagnosis of tuberculosis can be excluded or confirmed right away; further examination will be required in about 20 per cent of still doubtful cases. Always examine for abdominal and cervical glands in those cases where the chest radiograph is negative.

The early recognition of tuberculous infection can be simplified if a certain routine is followed: (1) Tuberculin skin test;

(2) X-ray examination ; (3) History of contact, especially familial ; (4) Physical examination ; (5) Symptoms ; (6) Sputum examination and gastric lavage ; (7) Sedimentation rate ; (8) Blood-picture.

1. Skin Test.—The finding of a negative skin test in a suspected child (that is to say, negative to Mantoux 1 in 100) must be given full weight in the interpretation of a radiograph. A positive test means that the child has now, or has had in the past, a tuberculous infection. In the majority of cases the radiograph will decide whether the disease is active or healed. Any doubtful cases, with positive test and negative X-ray findings, should be refilmed after a few weeks, and the child's weight watched ; often in these doubtful cases a culture of the gastric washings may reveal tubercle bacilli, thus indicating activity of the occult process in focus or gland. A change from negative to positive in a child's skin reaction is the most conclusive evidence we have that a primary infection is taking place ; when this change is accompanied by fever of unknown origin, the diagnosis is confirmed. Yearly or half-yearly routine testing of negative reactors is therefore of the utmost value. Cases which have been in contact with a phthisical adult should have their skin test repeated six weeks after removal from contact, in order to allow for the incubation period which proceeds the development of allergy. The tests recommended are : as a first test any reliable percutaneous ointment, equivalent to 1–1000 Mantoux ; as a second test in negative cases the Mantoux 1–100 intracutaneous test.

2. Radiographs.—An X-ray film, technically perfect and correctly interpreted, if judged in conjunction with the results of the tuberculin test, is the most reliable aid we have towards the diagnosis, prognosis, and treatment of primary tuberculosis. Screening as a preliminary to filming will discover infiltrations, large primary foci, and glandular enlargement ; but the possession of serial films is invaluable for reference. One seeks a primary infiltration, or a primary focus or gland, either active or in healing stages. It must be remembered that if the primary infiltration is very light or the whole primary complex a rather slight lesion, radiological evidence may be entirely lacking. Many obvious active lesions heal without radiologically visible residue. Negative radiological evidence must be checked and weighed by the clinician in his judgement of a tuberculin-positive case.

3. History of Contact.—A history of recent contact is of importance when judging a doubtful case, which has a positive skin test but negative X-ray picture; caution is advised in treating lightly such a case. The knowledge of a tuberculous home contact is strong supporting evidence; the number of children who suffer from primary tuberculosis is much greater in homes where there is a phthisical adult than in tubercle-free homes; the infection, furthermore, is considered by some observers to be more severe in its effects amongst the former.

4. Physical Examination.—Primary tuberculosis is remarkable for its absence of physical signs. "The more one hears in a small child's chest, the less likely it is to be tuberculous" is a truth born of practical experience. The diagnosis of primary tuberculosis of the lungs must be made on grounds other than those of physical examination; otherwise the condition will be overlooked, or, as often happens, cases of bronchitis and bronchiectasis will be condemned to a sanatorium. To say that a child has not got primary tuberculosis because the stethoscopic examination is negative, is a blunder. It is indeed unpardonable, for to wait for auscultatory evidence is to postpone treatment until cure is doubtful. At times, signs do exist, and can be elicited, but they are in no way diagnostic of primary tuberculosis. On *percussion* at times a somewhat dull note is heard over the affected area; this occurs for a brief period if the primary infiltration is heavy, but it may be missed entirely if the focus is deep-seated; this dullness remains for a much longer period in cases of atelectasis and is more definite. The primary focus in its healing stages will give no signs. *Auscultation* will reveal nothing abnormal over a light infiltration, whilst a heavy infiltration or an atelectasis may show some diminution in breath-sounds. Moist sounds are reported by some observers over a heavy infiltration.

It may thus be seen how very slight is the value of percussion and auscultation in the diagnosis of tuberculous infections in the primary stage. Any child with a bronchial cough will give more pronounced physical signs. On the other hand, one sees completely healed and calcified primary complexes of large dimensions where there is a history several years ago of "pneumonia lasting three months" or "congestion of the lungs years ago"; these histories indicate that heavy primary infiltrations produced lung changes which were puzzling and atypical at the time. As has already been emphasized, a

healing primary focus or hilar gland tuberculosis defies recognition by the most expert clinician without the aid of tuberculin test and radiology. The positive results of physical examination are misleading, but as a general rule signs are absent. The grave mortality-rate from tuberculosis in childhood can only be avoided when it is realized that the first stage cannot be recognized by the stethoscope alone.

5. Symptoms.—

Fever.—Only in the initial stage of the primary infection is fever present. Hypersensitive children may have very high initial fever with some degree of malaise, but they are surprisingly little ill in comparison with the rise of temperature. The fever lasts for a period between one and three weeks. The initial fever is often mistaken for a prolonged chill, influenza, and even typhoid fever. There is now no doubt (Wallgren) that tuberculous initial fever is the condition described fifty years ago by Landouzy, and called "*Typho-bacillose*", where after three weeks' fever a child later on developed tuberculosis of the lung.

Fatigue.—The most common symptom of primary tuberculosis is fatigue, and it is most often responsible for bringing the child under medical care. Healing of the primary complex is accompanied by a return of the child's normal desire for activity and play; for this reason it is more difficult to ensure that the healing case takes the prescribed amount of rest, whereas in the active phase there is no trouble in getting the child's co-operation in the matter of bed-rest. Loss of sleep, loss of appetite, and secondary anæmia are all present in varying degrees. Faintness and vomiting also occur.

Abdominal Pain.—Next to fatigue, abdominal pain is the symptom most often complained of by children who are passing through their primary pulmonary infection. They point to the umbilical region, but examination will fail to reveal any localized tenderness or involvement of the mesenteric glands. The dyspepsia of primary tuberculosis can be explained anatomically as the effect caused by pressure of enlarged mediastinal glands on the pulmonary plexus of the vagus nerve (Engel's *pes anserinus*). These and other pains in children are misleading and do not suggest a pulmonary lesion. A child of 5 years complained persistently of a pain in his head, and on finding that his tuberculin test was positive, he was X-rayed, and showed a primary infiltration in his right middle lobe.

Cough.—In the 4 to 14 year age period, cough is present in surprisingly few children suffering from primary tuberculosis. McPhedran found a higher percentage of coughs in tuberculin-negative than in tuberculin-positive children in a group of 300. The cough which may accompany a primary infiltration is never very definite ; indeed it is often absent. Young children under 4 years, suffering from primary tuberculosis, do not cough ; they only cough with infections involving the upper respiratory tract, either the trachea or larynx as in whooping-cough, or when enlarged mediastinal glands press on the trachea. In bronchitis they swallow the bronchial secretion, and vomit rather than cough it out ; in pneumonia it is only by continuous stimulation that infants can be got to cough. The exceptions are *bi-tonal cough* and *expiratory stridor* ; these tuberculous types of cough are most commonly met in infants and very young children, but may occur also at later ages. The bi-tonal cough is typical, due to compression of bronchi by enlarged glands ; one sound comes from the larynx and one from the bronchi. Expiratory stridor is due to compression of a bronchus or of the trachea by an enlarged gland, and produces an expiratory rattle, which resembles the wheeze of asthma. These coughs may be heard together or separately ; if heard they are diagnostic of enlarged tuberculous glands, generally in the upper mediastinum. Such cases should be propped up on pillows, to facilitate breathing ; if the gland pressure is present in a case of spinal caries, which has to lie in the recumbent position, severe choking attacks with cyanosis may occur, which are both alarming and dangerous. Other forms of cough in children usually indicate one of the acute infections, notably bronchitis. Many children sent for examination on account of chronic cough will prove to be suffering from bronchiectasis. Therefore cough as a diagnostic symptom of primary tuberculosis is often misleading, and its absence by no means a contra-indication. If, however, a child complains of cough, it is very simple to perform the tuberculin test ; in the event of a positive result, the chest should be X-rayed, and the sputum, if any, examined for tubercle bacilli, lest the condition present should be bronchogenic phthisis.

6. Sputum Examination and Gastric Lavage.—Few children produce sputum during the primary stage, and examination of any such sputum for tubercle bacilli will prove negative. From this fact we conclude that primary tuberculous cases are

not capable of infecting other children ; this conclusion is borne out by practical experience. Further examination, however, must be performed before a negative sputum can be regarded as of the slightest importance as contra-diagnostic evidence of tuberculous infection. Many of these children have no cough ; many when asked to cough will produce only saliva ; as a rule, if there is sputum present, it is swallowed ; thus the material presented for examination is seldom true bronchial secretion. Coughed material may be collected on a laryngeal mirror or the larynx swabbed in young children. Older children, over 4 years, however, will often co-operate in producing true sputum in tertiary phthisis, if sufficient trouble is taken on the part of nurse or doctor. One cannot then say that a child is tubercle-free unless a laryngeal smear or gastric washings have been examined. Examination of laryngeal or gastric contents by direct smear nearly always gives negative results ; guinea-pig inoculation gives more reliable information, but with a delay of many weeks ; culture of the centrifuged deposit is the most satisfactory method ; if saprophytes interfere with the reading, subculturing will eliminate them. Positive gastric washings are less frequently found in very early than in later stages of primary tuberculosis ; and are most often found in mediastinal gland tuberculosis. Gastric-washing examination is invaluable in doubtful and occult cases, where a diagnosis can be confirmed beyond doubt by the finding of tubercle bacilli. In practice it is not always possible to do this examination, nor does one wish to worry children unnecessarily ; and in many cases the diagnosis can be quite clear without this procedure. However, some people need to have the tubercle bacillus demonstrated before they are convinced ; and scientifically speaking the investigation can be complete only when the bacillus has been found.

The best method of obtaining stomach contents for examination is to pass a sterile stomach tube early in the morning, holding a kidney tray under the patient's mouth ; the tube is joined by a glass connection to a funnel ; 100 to 300 c.c. of sterile water are used. When the fluid has returned by syphoning to the funnel, the glass connection is removed, and the lavage fluid is received into a sterile bottle through the tube. Any vomited material in the kidney tray is added. It is best to collect from two lavages done on consecutive days. In the laboratory the fluid should sediment for 24 hours in funnel-shaped tubes with rubber

stopcock. The sediment is run into the centrifuge tube and centrifuged for 10 minutes at 1500 revolutions. Direct examination, culture on Loewenstein medium, or guinea-pig inoculation follows. Statistics show that guinea-pig inoculation will discover more positive reactors than will culture, although K. A. Jenson recommends culture smears. Evidence provided by examination of gastric washings is only of value when positive; and its chief use lies in (1) showing whether cases apparently healed are not yet free from tubercle bacilli, and (2) in the diagnosis of occult cases. To pronounce a child to be non-tuberculous because tubercle bacilli are not found in the gastric washings is erroneous if the tuberculin test is positive and the radiological appearances suggest an active intrathoracic lesion.

7. Sedimentation Rate.—An acceleration in the rate of fall of the erythrocytes in the blood-plasma gives an indication of the existence of inflammatory changes in the body during many infections. In tuberculous infections of the lung in childhood, the test has a certain value as a measure of the degree of activity present. The test is by no means specific for tuberculosis, as is the tuberculin skin test, and it has no diagnostic value. An accelerated sedimentation rate indicates that the tuberculous process is active, and that rest is required. If one is faced with a child showing a positive skin test, a normal radiograph or one showing a healed focus, and the sedimentation rate is normal, then one can be satisfied that the infection has been dealt with adequately by the defence forces of the body; in other words the condition is healed or at least inactive. Intercurrent infections such as head cold, sore throat, and infectious diseases, will cause a temporary rise in the sedimentation rate, and a view of the tuberculous condition may be wrongly interpreted under these conditions. In the treatment of the primary complex and mediastinal gland tuberculosis, the test is a useful guide; children should not be allowed out of bed until the sedimentation rate is within normal limits, and remains so after exercise. In a number of cases the test provides information which cannot be gained in so exact a manner by clinical observation of the case. The normal for a child is under 5 mm. per hour. A high reading (25–50 mm. per hour) is usually associated with a primary exudative lesion; a low reading (0–20 mm. per hour), with a caseous lesion of Type II or Type III, as well as with a healing primary complex.

Capillary Test.—The test may be performed with ordinary capillary tubes. Two ink marks are put on the tube, 1 in. and 5 in. from one end. Sodium citrate is allowed to run up to the first mark (1 in. of citrate). The thumb is pricked and blood is allowed to run up until the citrate above it has reached the second mark (4 in. of blood). The blood and citrate are run out into a watchglass, mixed, and run up the tube again, the entry of air-bubbles being avoided. The tube is set upright in plasticine and at the end of one hour the reading is taken, in millimetres. This is a convenient method for infants.

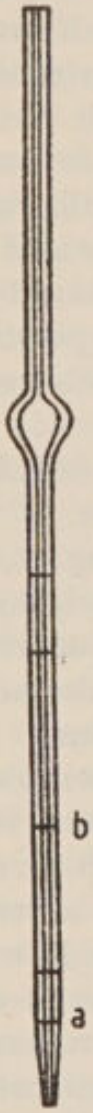


Fig. 32.—Microsedimeter (Landau's modification).

Westergren Test.—This is a very practical test, especially in older children. Payne's modification is widely used in some countries; in his test 0·4 c.c. of blood are drawn from a vein into a 1-c.c. syringe in which there is already 0·1 c.c. of a 3·8 per cent solution of sodium citrate. The blood is emptied into a small narrow vessel, mixed, and then run up a tube of 1 mm. calibre to an indicated mark. This tube is then set upright, sealed in plasticine, and after standing for one hour the fall is estimated by measuring in millimetres the column of clear plasma left below the mark. A fall of over 5 mm. per hour in children is accelerated.

Landau's Modification of the Linzenmeier-Raunert Test.—A 5 per cent sodium citrate solution is drawn up the 1-mm.-bore tube to (a) (*Fig.* 32). A large drop of blood, obtained by a prick of the child's thumb, is drawn up to (b), avoiding air-bubbles. The tube is tilted three times so that blood and solution are well mixed in the bulb. The tube is then held upright and the column of blood is allowed to fall a short distance down the tube, which is then sealed by plasticine at both ends or is placed in a special stand. The clear plasma at the top of the column is measured after one hour; 4 mm. is normal.

8. Blood-picture.—A great deal has been written about the white-cell picture in tuberculosis. In dealing with children, suffice it to say that a considerable degree of assistance can be gained from a white-cell differential count; when lymphocytes

predominate over neutrophils a healing stage is indicated. In an early stage of primary tuberculosis the hæmoglobin content may be as low as 40 per cent, rising to normal after treatment is instituted.

Differential Diagnosis.—

Pleural Effusion.—This is not always easy to distinguish from a primary infiltration. The radiology is discussed in the section on pleurisy (p. 79). Clinically, with effusion the percussion note is duller and the breath-sounds less audible. Exploratory needling is the best way to decide, for a history of contact and the tuberculin test will be positive in both cases. The pleural effusion appears at a later date in the primary infection than does the primary infiltration.

Pneumonia.—The clinical picture in acute pneumonia is more severe than that ever seen with primary infiltration ; all physical signs in the chest are more marked ; in primary infiltrations there is no increase in respiratory rate. The differential diagnosis may, however, present a difficulty in the case of small children suffering from lobar or broncho-pneumonia. A throat swab and vomited sputum should be examined for the presence of pneumococci ; the radiological appearances in pneumococcal conditions will in most cases clear up in a week or two. A negative tuberculin test (two strengths) will be very helpful in differential diagnosis ; in an older child a positive test may prove perplexing if pneumonia is superimposed on healed or active primary tuberculosis. The rapidly curative effect of sulphapyridine on the pneumonias has facilitated differential diagnosis during recent times. To exclude broncho-pneumonia is sometimes difficult by the radiological appearances, but the clinical picture is much worse than in primary tuberculosis. The following case will illustrate the procedure for differential diagnosis from unresolved pneumonia, a difficult matter.

Example.—J. M., aged 14 months (1934), admitted to hospital with extreme wasting, anæmia, anorexia, and cough. Radiographs at two weeks' interval revealed an irregular shadow over base of right lung. No history of tuberculous contact, and repeated skin tests negative. No fluid on puncture. Pneumococci, but no tubercle bacilli, found in gastric washings, and throat swab on two occasions. X-ray appearances the same two months later. After this a mixed pneumococcal vaccine was administered, and the child recovered rapidly ; in three weeks the radiograph was clear. Had reliance been placed on the negative tuberculin tests in the first instance, a cure could have been effected by the vaccine some months sooner.

Influenza.—Owing to the short initial febrile period, primary tuberculosis is often mistaken for influenza, especially as there are no specific symptoms in either condition ; in influenza there are, however, complaints of pains all over the body, and the tuberculin test may be negative ; radiographs will also be negative.

Bronchiectasis.—Tuberculin test and history of contact may be positive if the bronchiectasis is a residue of an old tuberculous lesion. Severe and chronic cough point towards bronchiectasis, and there will be ample sputum for examination. Lipiodol radiography will determine the presence of bronchiectatic changes. Many children with bronchiectasis have negative tuberculin tests. Cases unfortunately often find their way into sanatoriums, either with residual or with non-tuberculous bronchiectasis.

Example.—Patricia B., aged 5 years. Father died of phthisis three years previously. Four older children showed positive tuberculin tests and calcified Ghon foci. This child had chronic cough ; no tubercle bacilli in sputum, Hamburger negative, Mantoux 1–100 negative. Radiograph (*Fig.* 33) shows typical bronchiectasis following the line of the right hyparterial bronchus.

Empyema.—Clinically the child appears much more ill with empyema than with primary tuberculosis ; the temperature is hectic, or in long-standing cases normal ; empyema shadow is much denser in the X-ray film ; and many such small children show abdominal distension, wasting, and anorexia. The tuberculin tests in these cases are invaluable, especially in small children. Exploratory puncture is necessary. The blood-picture should be examined, a lymphocytosis favouring a tuberculous infection.

Lung Abscess.—Temperature is hectic, cough is present, and physical signs over the site are more definite ; radiological appearances are sharper in outline and more dense than in a primary infiltration. Tuberculin test, contact history, sputum or gastric-washing examination, exhaustive radiographs in various positions—all these, with the help of a blood-film, will be necessary before the case can be considered as a whole and a diagnosis made.

MEDIASTINAL GLAND TUBERCULOSIS

Anatomy.—A knowledge of the anatomy of the pulmonary lymphatic system and of the mediastinal glands is essential to the understanding of mediastinal gland tuberculosis in children.

The *Pleural Lymphatic System* consists of visceral and parietal plexuses; the visceral drain into the superficial pulmonary efferents, and the parietal into the posterior mediastinal glands. Communication between these two systems exists; flow of lymph from the superficial pleural to the deep lung plexus is prevented by a system of valves which lie in the smaller connecting vessels.

The *Pulmonary Lymphatic System* has a superficial and a deep set of plexuses. The superficial lie beneath the pulmonary pleura and terminate in some lymph-glands in the hilum. In childhood tuberculosis most important are the *deep pulmonary lymphatics*. These arise in the region of the bronchioles, where they are closely and thickly packed. The air-cells have no lymphatics. From the bronchioles these deep vessels accompany the branches of the bronchi and the pulmonary vessels, and eventually empty into the tracheo-bronchial lymph-glands at the hilum. Anastomosis between superficial and deep pulmonary lymphatics is slight. All the lymphatics in the lung drain into the glands at the hilum; drainage is always towards the hilum, owing to a complete and regular system of valves in the lymph-vessels, which prevents backward flow towards the the lung periphery.

The *Mediastinal Glands* (*see Frontispiece*, and *Figs.* 10, 34) consist of three main groups which lie in the bifurcation of the trachea, at the lung root, and around the trachea. Another small group, the *broncho-pulmonary glands*, lies partly in the lung outside the hilum and has no communication with glands on the opposite side, and partly extra-pulmonarily along the main bronchus in the hilum; both these groups are small, and whether they play an important role in tuberculosis of the lung is doubtful; certainly gross enlargement of the tracheo-bronchial groups can occur without any involvement of the nodes in these groups.

The *tracheo-bronchial glands* mainly are concerned in the primary complex. They consist of inferior and superior groups. The *inferior or bifurcation glands* drain the left and right lower lobes and the right middle lobe of the lungs; they empty into the superior glands of the same side. The right bifurcation is the largest gland in the mediastinum; there is free communication between these glands and those of the opposite side. Some drainage, besides that following the path of least resistance into the superior glands, takes place by efferents direct into

the internal mammary lymphatic vessels. The *superior tracheo-bronchial glands* drain the upper lobes of the lungs, and they empty mainly into the paratracheal chain of glands on the same side with some slight degree of drainage into the internal mammary lymphatic vessels; there is communication between the two sides. Engel has described the drainage of the apex of the left upper lobe into two small glands: the arcus aorticus gland (lying over the arch of the aorta), and the gland of Botallo (a small gland lying beside the remnant of the ductus arteriosus). These two para-aortic glands constitute the left upper hilum, and their enlargement points to a focus in the left apex; they are not involved in upward drainage from the left

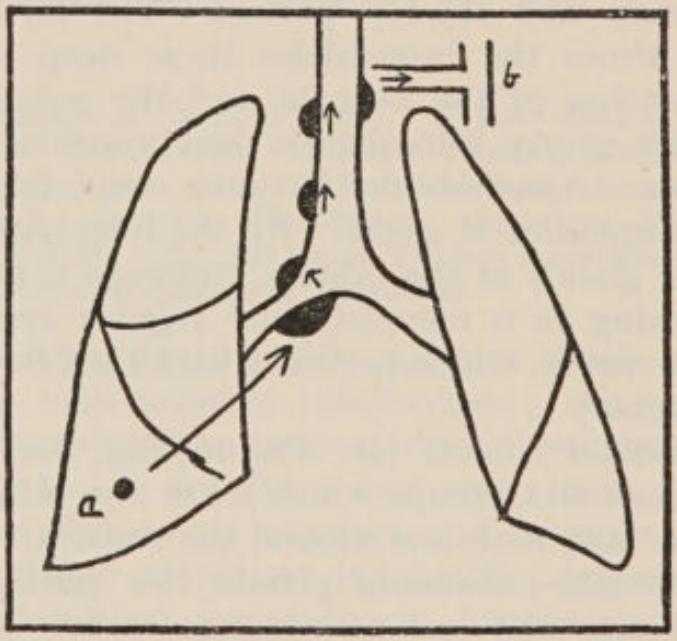


Fig. 34.—Diagram showing lymphatic drainage from (*a*) primary focus to (*b*) thoracic duct, via the mediastinal lymph-glands.

bifurcation gland, but only by direct spread in massive infections involving all the mediastinal glands. It is not certain how they empty, but it is thought that it may be direct into the thoracic duct. Besides those already mentioned, there are also two small groups on either side, the anterior and posterior hilar glands. These take no part in the drainage of the lungs, but they may be infected by direct contact with other caseous mediastinal glands; the posterior group are involved in cases of interlobar pleurisy.

The *paratracheal glands* lie wedged in between the trachea and the œsophagus. They do not drain any portion of the lung direct, but form the last of the chain of glands on the route of drainage from the tracheo-bronchial glands to the main

lymphatic ducts. They communicate with corresponding glands on the opposite side. They have no communication with the cervical glands. They empty their contents into the right lymphatic duct on the right side, and into the thoracic duct on the left side; the right lymphatic duct opens into the angle of junction of the right subclavian and internal jugular veins; the thoracic duct does likewise on the left. The paratracheal as well as the tracheo-bronchial glands give off small efferents which empty their contents into the great veins and the right lymphatic and thoracic ducts.

General Remarks.—By mediastinal gland tuberculosis is understood a clinical entity in which the primary lung focus is not seen, but the tuberculous process in the mediastinal glands remains active. There must have been a primary focus to cause the original glandular infection, but it may be invisible to X rays if very insignificant, either during or after healing, and for practical purposes it may be considered as closed, for the glandular activity dominates the picture. It is the intention in this section to deal with such glandular cases as a separate entity from the primary complex, and to call the condition 'mediastinal gland tuberculosis'.

The condition in itself tends to run a benign course, without serious illness. Yet there is, nevertheless, a danger of hæmatogenous dissemination from even a small caseous gland. This danger is greatest where there is massive involvement of glands; it decreases as healing advances, but it cannot be pronounced as completely passed until complete healing has been achieved. Many children who are positive tuberculin reactors have living tubercle bacilli lying latent in their bronchial glands; viable tubercle bacilli have been recovered at autopsy when death has occurred from other causes; bacilli have been found also in the gastric washings from cases which radiologically appear to be quiescent or healed. Thus it is clear that cases of mediastinal gland tuberculosis should receive serious consideration and must not be treated with contempt. On the other hand, every precaution must be taken in order that children with negative tuberculin reactions, showing shadows at the hilum, are not diagnosed wrongly as tuberculous.

Seen radiologically, there are two types of mediastinal gland tuberculosis: a well-defined and an indefinite form. German writers speak of these as tumour-form and non-tumour-form glands. A tumour-form gland presents few difficulties to

radiological diagnosis (*Figs.* 36 and 29B). Much more difficult is the correct interpretation of activity in the non-tumour-form gland (*Figs.* 4B, 13A, 23, 28B, 50). Here diagnosis has to be made by the recognition of local activity in the region of the glands, associated with a positive tuberculin reaction, and assisted by deduction on the basis of comparison with earlier X-ray films, where these are available.

Table XII.—Showing Type of Glands Found at Different Age Periods. (*From Engel and Pirquet's 'Handbuch der Kindertuberkulose'.*)

Age	Small Glands	Middle-sized Glands	Big Glands	Total
0– 6 mth.	1	3	6	10
7–12 ,,	2	8	10	20
2– 3 yr.	4	17	10	41
4– 5 ,,	4	8	3	15
6– 9 ,,	6	6	1	13
10–14 ,,	6	3	—	9
	23	45	30	108

The influence of age is important (*Table XII*): in infants under two years, mediastinal gland tuberculosis shows gross involvement of the paratracheal as well as of the tracheo-bronchial groups; because of glandular involvement at this age, the risk of massive blood-stream infection is greater; the large tumour-form swellings seen frequently in infants are but rarely seen in later childhood. At school age, glandular enlargement and perihilar activity are frequently seen, but are less marked; hæmatogenous spread from these is usually limited to one or two metastases, often endarterial with lodgement in bone or joint. At the 14 to 18 year age period the primary infection is often manifested by evidence of slightly enlarged hilar glands, whilst the focus may play a minor part (*Fig.* 51).

Tuberculous glands may be detected in three stages by means of X rays:—

1. *Swelling and Hyperæmia.*—This is a glandular response to infection in the lung, and is not specifically tuberculous (*Fig.* 38). If tuberculous the primary focus may or may not be seen at the same time (*Figs.* 11, 25, 35).

2. *Caseous Tumour.*—When the glands caseate they are more easily recognized as tuberculous, owing to the denser shadow of caseation (*Figs.* 4B, 23A).

3. *Calcification.*—Calcification of a gland gives a hard, sharp, and dense X-ray appearance, intensity depending on its age. There may be a single gland or a group, either at the root or in the paratracheal region. The glands are encapsulated when calcification commences, but the inner half takes years to heal. It is impossible to know when calcified glands are completely closed and free from living tubercle bacilli; the shrinkage which occurs after 5 years suggests healing (*Figs.* 16A, 54).

The ultimate fate of these tuberculous glands may be :—

a. They become progressively worse and disease extends into neighbouring glands, and so reach the blood-stream or rupture into a large bronchus.

b. They remain quiescent for a time and then either (i) heal, or (ii) disseminate.

c. They heal satisfactorily from the start, by fibrosis and calcification, and as years pass they shrink.

Hilum Shadows.—The terms 'hilum tuberculosis' and 'hilar flare' (Burton Wood) have been applied to cases which show in the region of the hilum a radiological shadow other than that usually associated with enlarged hilar glands. Such a shadow may be due to atelectasis, or possibly to interlobar pleurisy; it may also be due to a primary infiltration around a primary focus which lies near the hilum; or it may be due to caseous pneumonia developing in the neighbourhood of such a primary focus. These hilar shadows, which are very difficult to interpret, are seen most commonly between the ages of 5 and 7 years (*Figs.* 13, 14, 18, 35). These shadows often clear after prolonged rest, but in infants the prognosis is less certain.

Clinical Picture.—There is no characteristic clinical picture of mediastinal gland tuberculosis; such children generally present a rather healthy appearance, at least after the first couple of months. They seldom complain, but towards evening they may show fatigue; they may have a poor appetite, but very little else wrong can be observed. The temperature is normal or subnormal; in the mornings the child is at his best. The sedimentation rate is slightly raised at first and falls to normal, varying according to the degree of activity present in the glands. Some observers have reported an inclination to night sweats, which have nothing to do with the night sweats

of adult phthisis. The first suspicion of the condition may arise only when metastases appear. Cough when present is due to compression of the trachea or of bronchi ; in young babies with massive glands, this cough, dyspnœa, and wheeziness can be very distressing.

Symptoms.—Symptoms, if present, are mainly those of obstruction of trachea or bronchus, or of pressure on the phrenic nerve. In most older children, however, pressure symptoms are lacking. With tracheal or bronchial compression, there arise symptoms which, if not definitely diagnostic, will at least raise a suspicion of mediastinal gland tuberculosis. If expiratory stridor or dyspnœa is present, the diagnosis is nearly certain. Cough, if present, may be raw, high-sounding, and barking ; in some cases it is soft and bi-tonal. The metallic cough is due to stenosis, and may be associated with facial cyanosis, difficult respiration, vomiting, or choking ; it will persist for months, even for a year, and only ceases when the glands shrink.

Atelectasis may arise if an enlarged and caseous root gland presses on a bronchial branch (*see* p. 51). Pressure by an enlarged gland or group of glands in the hilar region on the phrenic nerve may result in paralysis of the diaphragm on that side ; this is not a common happening and is mostly seen in the earliest years of life ; the possibility should be borne in mind should a radiograph show elevation of the diaphragm, and screening show restriction of excursion, accompanied by clinical signs of dullness and diminished breathing at one base. Phrenic paralysis is generally a temporary condition, and function will return on shrinkage of the gland by healing. If, however, the nerve is destroyed, permanent paralysis will ensue (*Fig.* 15).

Physical Signs.—These are remarkable for their absence. Exhaustive examination will yield but dubious results, even in the most experienced hands. Dullness on percussion is found only if the gland tumour is of immense dimensions. Auscultation will be similarly devoid of results. Signs described in the past have to be mentioned, but they are not recommended as diagnostic aids.

D'Espine's Sign.—With patient erect, head flexed, arms folded, d'Espine claimed that whispered bronchophony could be heard below the bifurcation of the trachea in the presence of enlarged glands ; in normal cases only as far as the bifurcation. Armand Delille and others have demonstrated that glands cannot act as

sound-conductors between the trachea and the vertebral column. Even were d'Espine's sign correct, conditions other than tuberculous could produce the same effect.

Smith's Sign.—With head sharply extended, a venous blowing hum is heard over or at the side of the sternum. This hum is present in 50 per cent of normal children, and is therefore of no diagnostic value.

Diagnosis.—We have discarded one by one the older methods of diagnosis by physical examination, because they have been found to be inaccurate since the use of X rays has become universal; we must therefore reach our diagnosis of hilar gland tuberculosis by other means. We have to rely on the tuberculin test and radiology as in all primary infections. Sputum is negative, but gastric washings may show tubercle bacilli. History of contact and pressure cough and expiratory rattle, if present, help the diagnosis. The most difficult question to decide is ' What is a normal hilum ? ' Also, how can one know whether enlarged glands are due to tuberculosis or to other causes ?

The normal and the abnormal hilum are discussed in the chapter on RADIOLOGY. Engel and Kayne have recently pointed out the value of lateral radiography in detecting tuberculous mediastinal glands. But no radiologist can diagnose mediastinal gland tuberculosis without a knowledge of the tuberculin reaction, that is to say, without consultation with the clinician. As Hamburger has warned us, many children are condemned wrongly to a sanatorium life on the radiological findings of " heavy hilar shadows ", where the tuberculin test has not been applied. Every clinician who deals with childhood tuberculosis should look at and ponder over all his own radiographs, and reconsider them in the light of further developments in the case, and if possible he should compare them with post-mortem findings in the event of fatal termination. Only thus will he be enabled to make the very best use of his films, for he, rather than the radiologist, is in a position to follow the after-history of every case and thus confirm or correct his own diagnosis.

Differential Diagnosis.—

Generalized Glandular Tuberculosis.—See the section on CERVICAL ADENITIS, p. 164.

Hodgkin's Disease.—This condition is rather rare in children, and glands will be found enlarged in other parts as well as the

mediastinum ; the tuberculin test may be negative, if the child has not otherwise been infected by tubercle bacilli. The spleen is enlarged. In difficult cases a section may be cut of a piece of cervical gland and search made for eosinophils. The Hodgkin gland is usually softer, more discrete, and less nodular than are tuberculous glands ; Hodgkin glands in the mediastinum are usually accompanied by enlargement of the cervical group. The blood-picture early is normal ; later the hæmoglobin is greatly reduced.

Lymphosarcoma.—This is very rare indeed in children. It shows rapid growth in consecutive X-ray films ; at first the mediastinal shadow suggests tuberculosis. The spleen is normal and the liver enlarged in later stages. A negative tuberculin reaction will be helpful. (*Fig.* 37.)

Sarcoidosis (Boeck, Schaumann).—This condition occurs in young adult life rather than in childhood. Besides gross enlargement of mediastinal glands in many cases with negative skin test, there are also skin manifestations, which bear some resemblance to erythema nodosum. The aetiology of this disease is still obscure (*see* p. 159).

Persistent Thymus.—The tuberculin test here is indispensable. The thymus X-ray shadow is typical ; it fills only the upper half of the thorax, it moves on swallowing, and has no connection with the heart or mediastinal shadows.

Spinal Cold Abscess.—In small infants it is possible to mistake a spinal abscess occurring in the lower cervical region for mediastinal glands. Deep penetration of rays will obliterate a gland shadow, but both conditions may be present at the same time. A lateral view of the vertebral bodies should be taken.

Acute Bronchitis and Asthma.—Physical signs are more marked in bronchitis than in mediastinal gland tuberculosis ; during the first few days cough and expiratory rattle may be present in the former condition. Radiologically hyperæmia may give rise to a hilar shadow, but this will soon disappear. Subacute bronchitis is frequently superimposed on primary and mediastinal gland tuberculosis, especially in infants.

Pneumonia.—The hilar glands often appear enlarged in bronchopneumonia and in resolving lobar pneumonia, but the condition is temporary. The tuberculin test must be consulted, and both in infants and older children the clinical picture must be relied on for the diagnosis.

Whooping-cough.—The expiratory stridor of gland pressure causing bronchial stenosis should not be confused with the inspiratory spasm of pertussis. Nevertheless in small children it is not always easily distinguished, and sometimes one meets with glandular cases who are said to have "suffered from whooping-cough for a year". The cyanosis and fits of coughing, with occasional vomiting of mucus, due to gland pressure, may be confusing. Rely on tuberculin test and radiograph (*Fig.* 38).

PLEURISY

General Remarks.—We must consider views in general on pleurisy, and then apply these to the child. Although modifications will be seen to exist, yet the classification is the same for children as for adults.

Pagel classifies pleurisy into five pathological groups :—

1. Caseous pleurisy, mostly hæmatogenous, lungs often free.
2. Tubercles in pleura. Miliary dissemination or spread near focus.
3. Perifocal effusion, or collateral exudation ; pleura lies near an intra-pulmonary focus (either Parrot-Ghon or hæmic).
4. Circumscribed pleural fibrosis, covering pulmonary focus.
5. Diffuse pleural fibrosis, remnant of effusion or due to dry chronic pleurisy.

Pagel's definition of the processes concerned in pleurisy is a more exact one than those used as clinical classifications ; of the latter we are most familiar with the following :—

1. Miliary tubercles in the pleura.
2. Purulent or caseous pleurisy.
3. Dry or fibrinous pleurisy.
4. Wet or exudative pleurisy, serous or serofibrinous.

We shall refer shortly to the more rare conditions and to dry pleurisy, but the main portion of this chapter will deal with the condition which is seen most commonly in children, namely, *exudative pleurisy.*

Tubercles in the Pleura.—Tubercles may occur in the pleura as part of a miliary dissemination, or by spread from a caseating lung focus which lies close to the pleura. Both conditions are rare. The resultant exudation may at first appear to be benign, but soon the severe illness picture will suggest the more serious and fatal underlying condition. Pleural tubercles may be recognized first only at autopsy, and they may form part of widespread miliary disease such as occurs during the first year of life.

Purulent or Caseous Pleurisy.—Tuberculous pleural effusions do not become purulent, and tuberculous empyemata are not found in children except in the following very rare conditions: (1) Where hæmic foci occur in the pleura, without involvement of the lungs; (2) Where a caseous focus in the lung ruptures into the pleura during the induction of an artificial pneumothorax; this is very rare because unilateral phthisis in children is a rare disease and collapse of the lung is not frequently undertaken.

Dry or Fibrinous Pleurisy.—It is not easy to estimate the frequency of dry pleurisy in children. Pleural pain is seldom complained of, and pains due to other causes, for want of a better explanation, may be wrongly ascribed to dry pleurisy. Fibrinous pleurisy may be localized over a pulmonary focus, or it may be due to chronic dry pleurisy, or it may be a residue of a pleural effusion. In young children it seldom gives rise to clinical symptoms, the fibrin absorbs, and radiographs in such cases seldom reveal a pulmonary focus; in a few cases, however, which are associated with pain and friction rub, later films may reveal a small focus at the site of the original pain. In the majority of cases the old fibrinous pleurisy is only discovered years later by the presence of adhesions which prevent collapse during the course of an artificial pneumothorax.

Exudative Sero-fibrinous Pleurisy.—A perifocal effusion (exudative pleurisy) occurs when a tuberculous lung focus lies near the pleura; the focus may be either primary, or post-primary (i.e., belonging to hæmic dissemination or isolated bronchogenic phthisis). In children and adolescents, pleural effusion is found chiefly in association with primary tuberculosis of the lungs; in these cases the effusion is usually large and obvious. With hæmic foci of the re-infection type, which are usually situated in the infraclavicular region, pleurisy if present is localized to the cupola region; this is not easily distinguishable from an apical pneumonia or a caseous infiltration round an Assmann focus (it may coincide with the latter); thus it may escape notice until artificial collapse of the lung is impeded by residual apical adhesions.

We are chiefly concerned with the pleural effusion which is associated with primary tuberculosis; this appears in many cases without previous complaint of pain, or suspicion of tuberculous infection; on the other hand, it appears also in cases which have already been under observation for a primary

tuberculous lesion. Treatment of the primary infection seems to have no influence in preventing serous effusion ; it will occur in hospitalized as well as in unrecognized cases. This clinical fact would appear to confirm the pathological conclusion that pleural effusion depends on the anatomical position of the tuberculous focus in its relation to the overlying pleura. Bilateral effusion occurs when the opposite side is affected after the first. This is probably due to a secondary hæmic focus in the contralateral lung which lies also subpleurally. On account of the hæmic nature of this second focus the prognosis is not so good as in unilateral pleurisy (*Fig.* 39).

What is the connection between serous pleurisy and primary tuberculosis in children ? The clearest view to take, as described by Kayne, is that a serous effusion in primarily infected children represents a hypersensitive phenomenon of the pleura due to a primary focus which lies near it. This pleural reaction occurs later than the primary toxin reaction of the lung tissue, i.e., the primary infiltration ; it appears 3-15 months after primary infiltration. It is important as a pointer to primary infection and as a warning that hæmic or bronchogenic phthisis may develop, and should therefore be given due attention.

Age.—In infancy pleural effusions are difficult to recognize. It is not known in what percentage they occur after a primary complex. In infants the primary focus in many instances is situated deep in the lung tissue, and does not lie sub-pleurally, and this fact may account for the rather rare occurrence of pleural effusion at this early age period. Price at autopsy on tuberculous infants found pleural adhesions in about half the cases, but seldom fluid ; and clinically observed only 3 cases of effusion amongst 300 children under 3 years of age who were admitted to hospital for treatment of primary tuberculosis of the lung. At school age it is more common, especially in the older periods such as 10 to 12 years. The adolescent period, however, is the most usual time to find effusions associated with primary pulmonary lesions ; at this age also are found effusions associated with the infraclavicular hæmic focus and with phthisis.

Some authorities state that they meet cases which are not tuberculous ; in this connection it must be stressed that lack of radiological findings alone cannot be accepted as proof of a non-tuberculous process ; tuberculin tests up to Mantoux 1-100 must be performed before a careful investigator is satisfied to pronounce a case to be non-tuberculous.

Localization of the Serous Effusion.—(1) Diaphragmatic; (2) Interlobar; (3) Mediastinal; (4) Costal.

Diaphragmatic Pleurisy is seldom found alone, but is usually associated with costal pleurisy. Its presence may be suspected if the line of the diaphragm does not present its usual sharply defined shadow in the radiograph. Later on, adhesions may be seen in an anterior-posterior film, and are recognized by a puckering or unevenness of the line of the diaphragm; adhesions to the pericardium may also be present.

Interlobar Pleurisy is frequently diagnosed in children. Many of us, however, now conclude that the shadows so diagnosed are in reality due to other causes. It is impossible, clinically, to be certain what condition is present; exploratory puncture has seldom, if ever, been successful in finding fluid. It is impossible with certainty to differentiate radiologically between the shadows cast by interlobar fluid, by a primary focus lying near the hilum, or by enlarged hilar glands; atelectasis of a portion of the lung in that region casts a very similar shadow (*Fig.* 18). These shadows are particularly frequently seen at 5 to 6 years. If a film is taken with the child in the lordotic position, this interlobar shadow is thrown into relief, and that it is bordered by the septum is quite clear; even so, many observers think now that this shadow is not caused by fluid but by atelectasis or a deposit of exudation debris at the base of a lobe. It is said that old pleurisy leaves a line of pleural thickening, i.e., a hair line; in many cases this is now considered to be normal septum marking, or if not lying in the line of the septum a residue of atelectasis. The tuberculin test in all these conditions will prove positive, except where there is an interlobar pleurisy due to a non-tuberculous infection, possibly influenzal pneumonia. Physical signs are impossible to elicit. Differential diagnosis is from non-tuberculous pneumonia, atelectasis, bronchiectasis, and residual primary infiltrations which are bounded on one side by an interlobar septum.

Mediastinal Pleurisy.—Tuberculous mediastinal pleurisy is characterized by a small effusion of the adhesive type, and positive tuberculin test. It is usually associated with bronchial gland tuberculosis and also with certain (possibly resolution) phases of infiltration. It is due to inflammation of the mediastinal and visceral pleura in the mediastinal space; it is most certainly rare, and difficult to recognize. Large effusions are non-tuberculous.

Costal Pleurisy.—See EXUDATIVE PLEURISY, p. 76.

The Clinical Picture of Sero-fibrinous Pleurisy.—The onset is acute, and may or may not be preceded by pain and the audible friction rub of dry pleurisy. The temperature rises in one to three days to its highest peak, which may be 102° F. to 104° F.; it falls gradually during the following two to four weeks. If fever remains after three weeks, suspicion must be aroused of activity in the underlying lung focus, apart from the pleurisy. At first the child is definitely sick and may complain of malaise or abdominal discomfort. Small exudates produce but few symptoms, but large effusions may give rise to respiratory and cardiac embarrassment. The small exudate is absorbed more rapidly, the large may require tapping to relieve pressure symptoms. Physical examination will reveal deficient chest expansion on the affected side, dull note with marked resistance on percussion, inaudible breath-sounds, and diminished or lost vocal resonance on auscultation.

There is nothing typical about the blood-picture.

Radiologically a small effusion will show in the costophrenic angle; later it creeps up the axillary border. The fully established picture is that of a shadow covering a large portion of the lower lung; a straight fluid line is present in this phase, but during resolution the upper border may be curved slightly, with its highest point on the axillary side; or again during resolution it may show as cloudy patches, without any definite fluid line (*Figs.* 29B, 40). The heart may be displaced by a large effusion.

Exploratory puncture once is permissible, indeed necessary, for diagnostic purposes. It is the general opinion that further withdrawal of fluid should be reserved for cases where the size of the effusion is causing marked embarrassment. The *exudate* is a yellow or yellowish-green coloured fluid, which in at least 80 per cent of cases is clear and not hæmorrhagic. Specific gravity is from 1013 to 1022. The cell content varies: in the first week leucocytes are increased, but after that lymphocytes predominate. Tubercle bacilli are found in exceptional cases only, seldom in pure cases of 'perifocal' pleurisy; in seeking these, the fluid should be examined not only microscopically but also by guinea-pig inoculation.

Example.—Christopher S., aged 11 years. Mother in a sanatorium, father dead. Presented himself at hospital complaining of "feeling weak" on rising and after running. History of 6 weeks' illness between

Dec. 25 and March 17, 8 months previously. Hamburger positive. X rays showed resolving pleural effusion and activity in right hilar gland (*Fig.* 40). Cured by preventorium treatment.

Diagnosis of Tuberculous Pleurisy.—

1. Physical examination.
2. Positive tuberculin test.
3. Exploratory puncture and examination of fluid.
4. Typical onset, 1 to 3 days.
5. Radiograph.
6. Normal leucocyte blood-count after first week.

Differential Diagnosis.—It is wise to make a diagnosis of pleural effusion primarily on the results of physical examination, i.e., complete dullness and loss of all breath-sounds, with the finding of fluid on exploration. If the radiological picture alone is consulted, a pleural effusion of moderate size may be confused with several conditions ; primary tuberculous infiltrations sometimes closely resemble effusions, but in the latter condition tilting during screening may show shifting of the fluid line ; the heart may be displaced ; the shadow of an exudation is usually denser and more defined than that of infiltration. In lobar pneumonia and atelectasis the heart is drawn to the affected side, instead of being displaced as by effusion ; in the lower zone in infants differential diagnosis may be difficult. The fact that a primary complex is some months old when first recognized by the appearance of a pleural effusion, explains why many cases fail to show radiological evidence of primary tuberculosis after the effusion has cleared.

Example.—Charlie C., aged 10 years. Oct. 28, 1938, attended hospital extern complaining of a " sick stomach ". Hamburger positive. X rays showed shadow of an encapsulated gland in left hilum. Treated by rest at home for 5 months. Nov. 17, 1939, returned complaining of a pain across the back and anorexia. X rays showed opacity in left mid-zone with ill-defined edges ; this pleural effusion might have been mistaken for a primary infiltration had not the tuberculin reaction and X-ray appearances of 13 months ago been known. Later, 15 c.c. of typical fluid was aspirated.

Prognosis.—The immediate prognosis in tuberculous serous pleurisy is good. The effusion clears after a time, and does not become purulent. The presence of an effusion, indeed, is useful in facilitating the diagnosis of primary tuberculosis which might otherwise have been unobserved ; the treatment of the pleurisy ensures rest for the primary tuberculous lesion. After

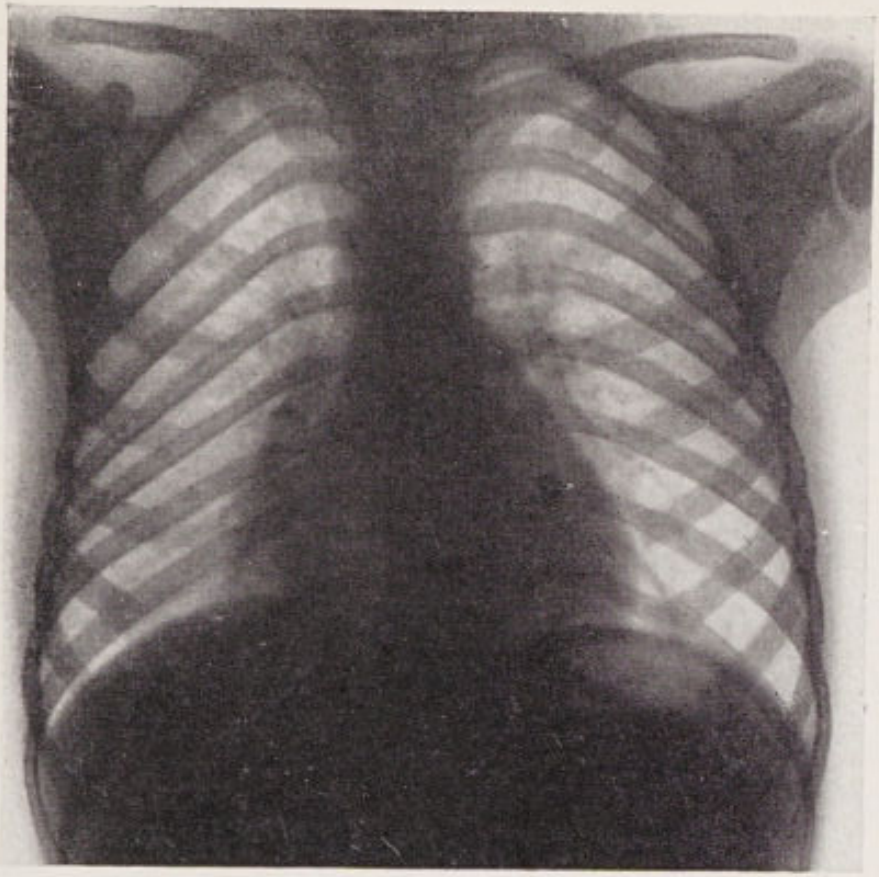
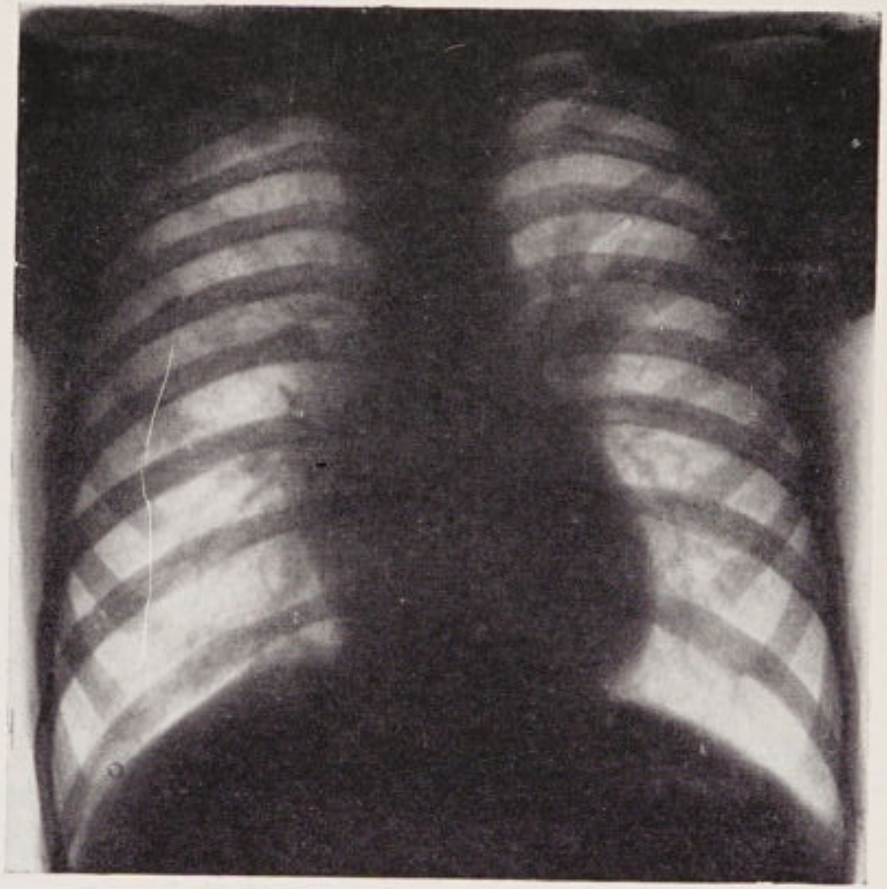


FIG. 33.—BRONCHIECTASIS.

Patricia B. Aged 5 years. (*See Example*, p. 66.) Hamburger and Mantoux 1-100 negative. The shadow in the right cardio-phrenic angle follows the line of the right hyparterial bronchus and is due to bronchiectatic changes.



FIGS. 35A, 35B.—PRIMARY INFILTRATION NEAR HILUM.

Fig. 35A.—Tom O'K. March 1, 1937; aged 8 years. Complaint of abdominal pain, Hamburger positive. Primary infiltration covers focus and gland on left; this cleared after a few weeks.

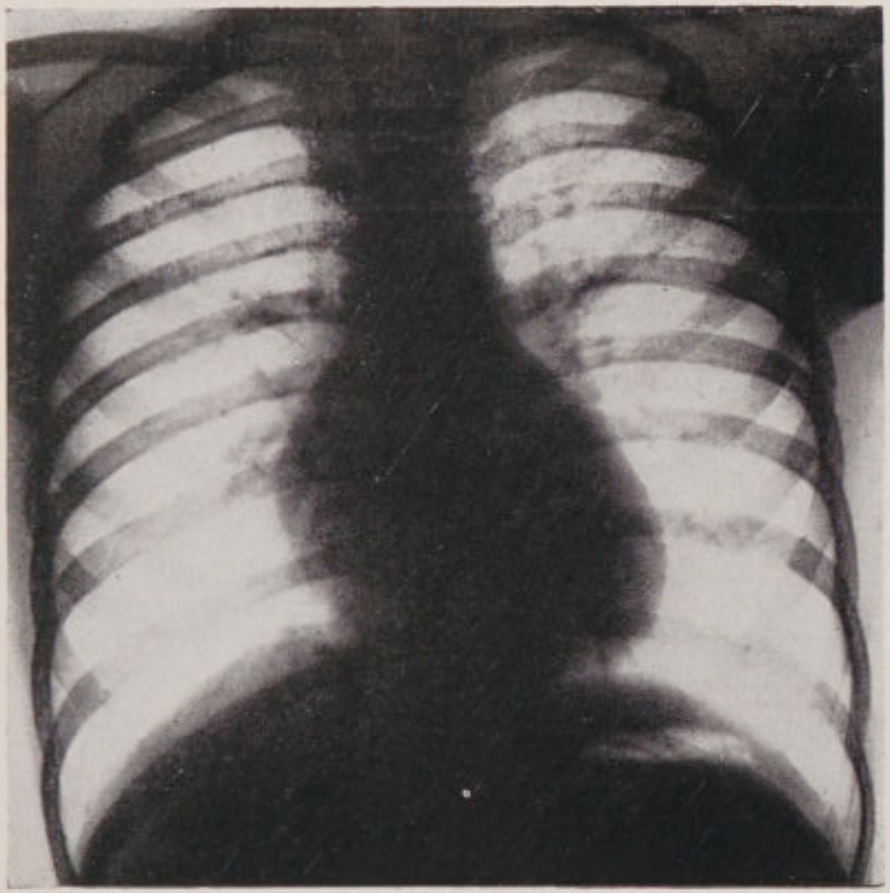


Fig. 35B.—Nov. 15, 1938. Healed.

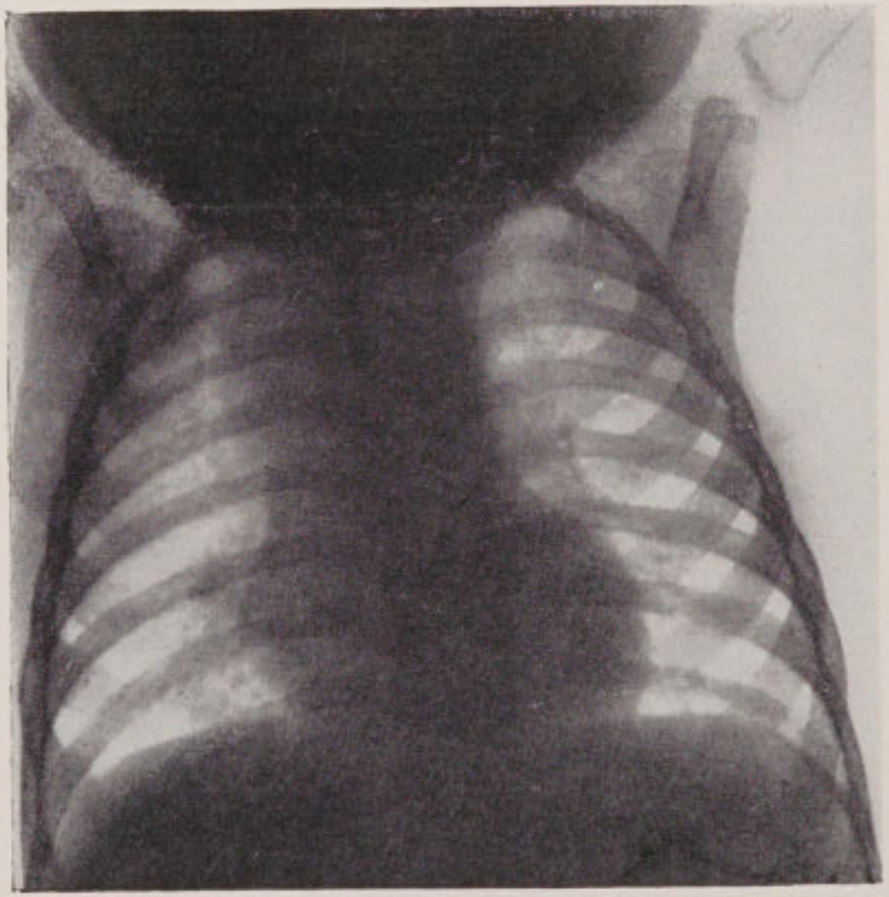


FIG. 36.—TUMOUR-FORM MEDIASTINAL GLAND.

Sarah O'B. Aged 9 months. Percutan test positive; the superior tracheo-bronchial and paratracheal glands are enlarged and cast a tumour-form shadow. Six months later a conjunctival phlycten developed.

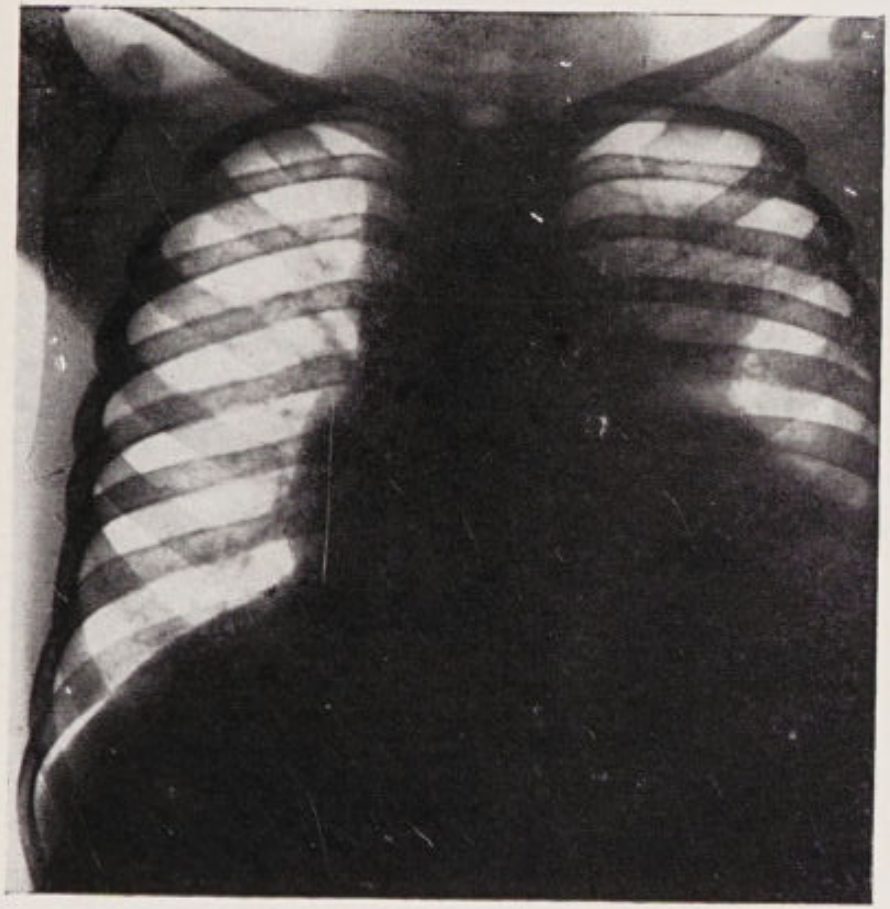


FIG. 37.—LYMPHOSARCOMA.

William C. Aged 12 years. Hamburger and Mantoux 1-100 negative. No fluid found. Gross glandular enlargement along the left border of the mediastinum.

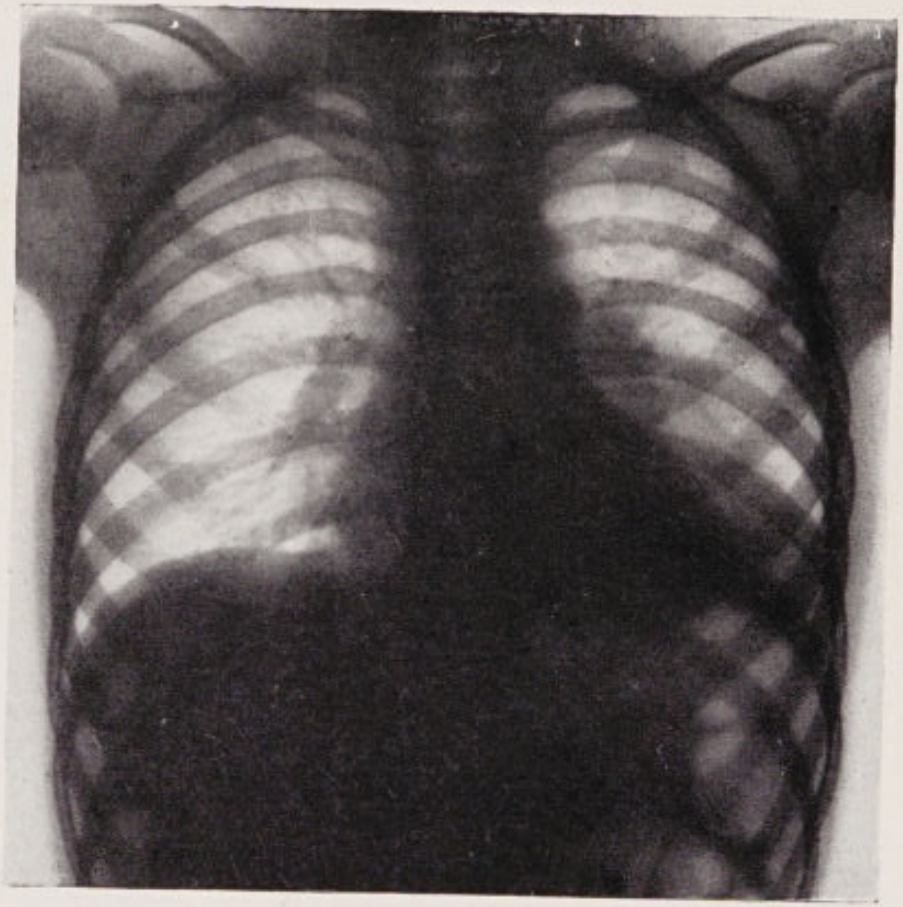


FIG. 38.—WHOOPING-COUGH.

Anastasia O'B. Aged 5 years. Tuberculin test negative at date of film and 2 months later. Gross enlargement of left hilar glands associated with a severe attack of whooping-cough.

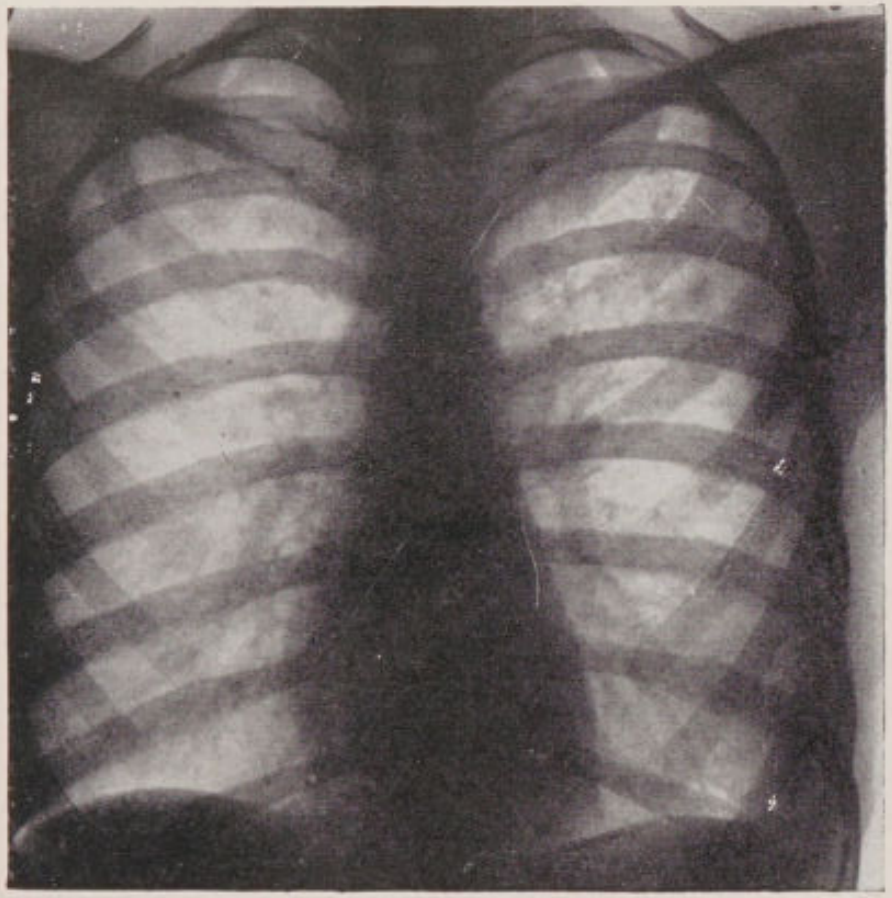


FIG. 39.—BILATERAL PLEURISY FOLLOWED BY PHTHISIS.

Eileen K. Feb. 8, 1939. Hamburger positive; right pleural effusion.

Jan. 8, 1940. Right effusion absorbed; commencing effusion on left.

May 21, 1940. Large left effusion; suspected re-infection lesion at level of 2nd interspace on left.

July 24, 1940. Left effusion reduced, lesion spreading.

Oct. 21, 1940. Aged 16 years. Right base clear, left effusion reduced; numerous active lesions in both infraclavicular regions (*Fig.* 39).

Sept. 1, 1941. Further extension of lesions.

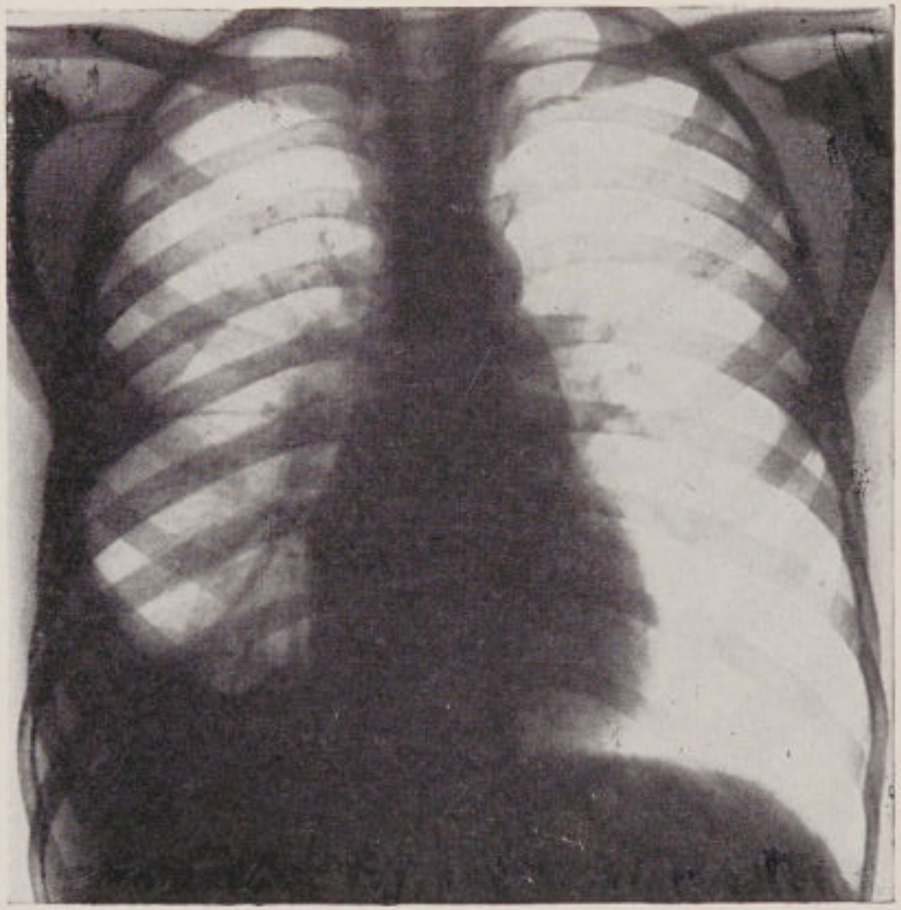


FIG. 40.—PLEURAL EFFUSION.

Christopher S. Aged 11 years. (*See Example*, p. 79.) Pleurisy in stage of resolution; calcification commencing in right hilar gland.

a pleural effusion, patients should be warned to return for supervision for 2 to 3 years. Unfortunately the effusion generally appears when the all-important danger period of the first three months after primary infection is past; therefore the recognition of pleurisy has less favourable influence on the prognosis of primary tuberculosis than has the recognition of the infection by initial fever. Pleural adhesions, futhermore, impede collapse, should the patient develop phthisis and require artificial pneumothorax. Many cases of adult phthisis give a history of a previous attack of exudative pleurisy. Prognosis depends largely on what treatment is given; where pleural effusion is treated by prolonged rest, a unilateral case will seldom proceed to the development of phthisis. The author's experience has been that where phthisis develops in young adults who give a history of previous exudative pleurisy, then it will be found that the pleural effusion had received inadequate treatment. When a pleural effusion develops in a person who has already been treated satisfactorily for primary tuberculosis, then the prognosis is as favourable as in any treated case without effusion. If the primary lesion is first recognised only at the time of the effusion, then the prognosis cannot be so favourable as when the primary lesion has been treated earlier, for the bacillæmia which occurs early in primary infection may have produced post-primary seedings which may remain unhealed in the absence of treatment. These latter cases therefore require even more prolonged rest than treated primary cases. In either event, the amount of rest accorded to the effusion influences prognosis.

Treatment.—It is hardly possible to overdo rest therapy for pleural effusion, and only too easy to be unwise enough to curtail it. In the past the period of bed-rest has been totally inadequate; 4 to 6 weeks were commonly recommended. Parents should be warned that unless rest is insisted on at this time there is definite risk of subsequent development of phthisis; and they should be instructed to bring the child for re-examination at intervals. Effusions should be treated by absolute bed-rest until absorbed; further partial rest for 6 months is recommended. It is a sound rule to keep the child or adolescent who develops a pleural effusion away from school or work for a whole year, or at least 6 months after the effusion is absorbed and the chest film clear. There is, however, no advantage in keeping a young person from work unless they are prepared to co-operate in observing part-time rest and leading an easy life. The question

of withdrawal of fluid and replacement by air as a therapeutic measure has been considered (Maurer); but it has been found in practice that when replacements are discontinued and the lung expands that pleural adhesions will still occur. This practice is not often adopted, although air replacement had the advantage of permitting inspection of the underlying lung. No treatment is so safe as bed-rest. After an effusion watch must be kept on the apical and infraclavicular regions. Bilateral effusions require prolonged and strict bed-rest, whilst sharp watch is kept for radiological evidence of hæmic lung foci. A properly treated primary infection and simple unilateral pleural effusion seldom give any further trouble.

Example.—Elsie B., aged 18 years. Developed a right pleural effusion, temperature 101° F. She was admitted to a general hospital, effusion confirmed by X rays. A second small effusion appeared on the left side, confirmed by X rays. There was great pressure on the hospital beds, and after 6 weeks in bed she was sent to a convalescent home for 2 weeks and then home. After a week at home, the parents sought further advice; a further radiograph, 10 weeks after the onset of pleurisy, showed the left effusion absorbed, some residue of the right effusion still present, and two small round 'Simon' foci were observed in the right apical zone above the clavicle. She was put on strict bed-rest at home. One month later X-ray report was "unchanged". Bed-rest was continued. Five weeks after that the film showed both effusions gone, and both apical foci had disappeared. Four months later, still on bed-rest, the film showed lungs clear and foci gone. She was now put on part-time rest, her S.R. reading was normal; progress continued satisfactory one year later, and she is resuming normal life, but not yet taking up a strenuous job.

This case illustrates the inadequacy of 2 months' rest for pleural effusion. The small second effusion should have suggested the likelihood of hæmic foci. The girl was caught in a very dangerous condition, at the time when the Simon foci were showing signs of activity; these post-primary seedings form a link between primary and bronchogenic phthisis, and the link was in the process of development.

CHAPTER VIII

THE SECOND TYPE OF TUBERCULOSIS: HÆMATOGENOUS SPREAD

MILIARY DISSEMINATION, MENINGITIS

General Remarks on Hæmatogenous Spread.—With the entry of the tubercle bacillus into the blood-stream primary tuberculosis may at any moment develop a Type II lesion; there can be no arbitrary distinction between the two, and it is only by the appearance of hæmic metastases that the second type becomes the clinical entity which plays such an important part in childhood tuberculosis. In children hæmic spread is responsible for nearly all forms of tuberculosis other than the primary complex. If dissemination occurs in young children it takes place soon after primary infection; for this reason Type II and Type III lesions cannot be termed 'reinfection' in the sense of a fresh infection from without; these lesions should rather be regarded as a direct continuation of the tuberculous process in an unhealed primary focus or gland. The dose of bacilli released into the circulation may be small and result in solitary metastases; or it may be massive and repeated and cause widespread foci with fatal issue. The immediate and most dangerous results of hæmic spread are miliary dissemination and meningitis. The more remote extra-pulmonary lesions will be considered in another chapter.

The age of the patient exerts an important influence on the course taken by a primary tuberculous infection. During the first year of life, miliary disseminations occur frequently and with extraordinary rapidity, and nearly always prove fatal. Tuberculous meningitis also has its highest incidence during the first three years of life. From the age of two to five years the tendency to generalization is less marked than during the first year. At early school age, 6 to 10 years, infection is usually confined to the tracheo-bronchial glands; these glands present a good defence owing to the fact that general resistance is better developed, and there is less tendency to generalization of the disease, together with a decrease in the incidence of meningitis.

Metastases at the 6 to 10 years age period tend to be solitary, and although they may cause considerable local damage in bone or joint, they are less dangerous to life than in earlier age periods. At late school age and at puberty there again arise increased risks of bone metastases, meningitis, and of generalization from a fresh primary complex; and there is a new danger, namely, the hæmic lung focus (usually infraclavicular), which is the forerunner of bronchogenic phthisis.

Entry of the Tubercle Bacillus into the Blood-stream.—We must now consider in detail how the tubercle bacilli enter the blood-stream; at the same time must be investigated what influence, if any, the mode of entry has on the resultant type of lesion. Entry can take place from three sources, i.e., three manufacturing depots (*see Table XIII*): (1) From primary focus; (2) From gland of primary complex; (3) From glands infected after the primary gland.

Table XIII.—Entry of Tubercle Bacillus into the Blood-stream.

Primary Focus.—Thence tubercle bacilli may reach—

- (1) Pulmonary artery → Localized miliary disease of the lungs
- (2) Pulmonary vein → Left auricle → General circulation → Brain, Bone, Skin, Kidney

Primary Gland.—Thence tubercle bacilli may reach—

- Hilar gland, Paratracheal gland → Thoracic duct →
  - (*a*) Right auricle → Miliary disease of the lungs
  - (*b*) General circulation → Brain, Skin, Bone, Spleen

These explanations of the routes followed, however, cannot entirely account for the selection of a particular site for lodgement. It is possible that selection may also be influenced by one or other of the following considerations: (1) Small end-arteries with sluggish flow favour lodgement; (2) Trauma may facilitate lodgement.

It is impossible to be dogmatic on the question of hæmic spread in tuberculous children. All that can be said with certainty at present is that massive infections in infants are nearly always found to be associated with enlarged and caseating

mediastinal glands, frequently paratracheal groups. At the other extreme, however, meningitis is at autopsy frequently found to be associated with a minimal lesion of the primary type situated in the lung or abdomen. Tuberculous lesions on the walls of the thoracic duct have been found in certain cases of miliary tuberculosis. Direct spread from chest to abdomen, via the posterior mediastinal glands, and vice versa, may occur, but the author has never seen it. In later childhood when the glandular response is more restrained, local spreads and isolated metastases are more frequently seen.

MILIARY DISSEMINATION

There are two main groups of hæmatogenous spread in the lungs of children; one is *diffuse* miliary spread and the other *localized*.

Diffuse Spread

(1) Acute miliary disease; (2) Chronic miliary disease; (3) Confluent caseous pneumonia.

Acute Miliary Disease.—Acute miliary tuberculosis occurs in one of two ways: (1) By the eruption of a caseous focus into a blood-vessel or into the thoracic duct; or (2) In the course of a bacillæmia during primary infection, a vascular focus (sometimes more than one focus) is formed in the intima; in the absence of resistance sufficient to check the multiplication of tubercle bacilli in this focus, it will liquefy; then the ejection of large numbers of bacilli may take place; thus it is usual for a caseous endangiitis to precede acute miliary dissemination, with the immediate result of formation of miliary foci in the lungs (Pagel). Studded throughout the lungs occur foci the size of a millet seed (*milium*, a millet); these foci sometimes increase in size until the small caseating nodules give the well-known radiological appearance of 'snow-storm'. In these cases the spleen and other abdominal organs are also affected. Very small millet-seed foci, even when bilateral, may at first be indiscernible radiologically; they should, however, be suspected in the presence of large non-encapsulated glands in very young children. Acute miliary tuberculosis is found amongst children of all ages, but the greatest number of cases occur during the first year of life.

Miliary tuberculosis, it has been said, is an unpredictable accident. This is true admittedly of certain cases which occur in

spite of all precautions taken amongst negative reactors, such as frequent tuberculin tests and X-ray examinations; this type of dissemination, like meningitis, occurs early in primary infection, and is acute and fulminating in its course. Nevertheless there is no doubt that miliary spread can be avoided in a number of cases by adequate treatment of primary tuberculosis; it is particularly noticeable that the incidence of generalized dissemination is low in countries where precautions, followed by adequate treatment, are taken to detect the primary lesion; in Ireland a reduction in the incidence of miliary tuberculosis in young probationer nurses is remarkable in those hospitals that have instituted tuberculin testing. In Dublin, amongst 200 cases under 5 years, treated by Price for primary tuberculosis in St. Ultan's Hospital, none developed miliary tuberculosis; yet during a five-year period in that hospital, miliary tuberculosis accounted for 36·7 per cent of all deaths from tuberculosis; in the latter case the babies first came under observation when already suffering from hæmatogenous dissemination.

Symptoms are often absent and signs negative in infants; indeed diagnosis often has been made on the post-mortem table. The condition may be recognized in a chest radiograph by the appearance of miliary tubercles and enlarged glands; in early stages these appearances may be quite indefinite; in the absence of physical signs, moreover, the taking of a chest radiograph may be overlooked. The millet-seed type of disease frequently yields no physical signs, but in later stages rales may be heard all over the lungs. In the older child physical signs are more obvious, but as they resemble bronchitis the diagnosis may be missed. At first an infant or child may gain rather than lose weight; wasting is not marked until towards the end. Appetite is not impaired; frequently an infant will take a full feed just before death. The sedimentation rate may be normal, indeed more often so than raised. If sputum can be obtained it will be found packed with tubercle bacilli; in the absence of sputum, culture of a centrifuged deposit of gastric washings will demonstrate the bacilli in a very fair proportion of cases; this is often the only means for early diagnosis of miliary tuberculosis in infants, before radiological appearances are definite.

Types.—In acute miliary tuberculosis, three clinical types are described—namely, typhoid, pulmonary, and meningeal forms. Although cases do not fall exactly into one or other category, there is a tendency for children to present predominant

symptoms, which may obscure the true diagnosis. Many infants in the first year of life do not conform to any of these types, but appear rather to die of asthenia, and are frequently labelled marasmus.

1. The *typhoid* form may be seen in the early stages of the disease; toxic and abdominal symptoms predominate; the abdomen is tender, the spleen enlarged; anorexia, high fever, full pulse, and prostration are present. Differential diagnosis is from typhoid fever and sepsis; in typhoid there will be typical blood and fæces findings.

Example.—Thomas T., aged 1½ years. Father, chronic phthisis. Child was treated at home for "bronchopneumonia". Two months later admitted to hospital, tuberculin tests negative to 1–100 Mantoux (terminal anergy). X rays showed miliary dissemination of lungs. Vomiting, abdomen distended, otitis media, wasting, anorexia, temperature 99°–102° F. Died four months after first symptoms were noticed.

2. In the *pulmonary* form respiratory symptoms predominate, with at times irritating cough, bronchitic adventitious sounds, dyspnœa, and cyanosis. In the early stages the radiological appearances may be indefinite.

Example.—Rita P., aged 9 months. Triplet, rest of family healthy. Cough and rales all over lungs, some dullness. Temperature 99°–101° F., rising during last three weeks from 100° to 105°. No abdominal symptoms; wasting. Autopsy: miliary tuberculosis of both lungs, some enlargement of tracheo-bronchial glands; no pleural adhesions; liver enlarged, spleen showed miliary tubercles; a few small caseous abdominal glands.

3. The *meningeal* form shows terminal cerebral symptoms at the end of a miliary lung infection. Convulsions are usually the first indication of cerebral involvement, but the cerebrospinal fluid does not become typical of meningitis unless the child lives for some days after their appearance. This type is due to cerebral tubercles which have formed as part of the general miliary dissemination.

Example.—Thomas G., aged 9 months. Ill at home for a month with bronchopneumonia. Then admitted to hospital as X rays showed widespread miliary disease of the lungs. Three days later, and four days before death, convulsions occurred; temperature 104° F; cerebrospinal fluid showed globulin normal, lymphocytes present, no tubercle bacilli found.

Diagnosis.—From the clinical appearances diagnosis is not always easy, for often physical signs in the lungs are neither

defined nor typical. The tuberculin test is less dependable than in primary tuberculosis; with swamping of the body with bacilli, the child may react negative even to Mantoux 1–100. As the test gradually becomes less and less positive during the last few weeks, so the X-ray appearances become more definite (*Fig.* 6). A history of home contact is helpful, if obtained. The usual duration of the acute disease is 2 to 3 months; infants may live longer. Cases which show thick firm foci in the radiograph demonstrate an effort at healing (fibrosis or calcification), and if this effort shows any marked success, they come into the category of chronic miliary tuberculosis. Acute cases seldom live more than 3 months.

Chronic Miliary Tuberculosis of the Lungs.—The difference between acute and chronic miliary tuberculosis is: in acute there are (*a*) repeated and (*b*) massive spreads of tubercle bacilli; in chronic only one or several spreads of small numbers of bacilli occur. Chronic miliary disease progresses slowly and often shows some degree of healing at first, but later the foci become active once more, or meningitis may supervene. Recoveries, however, are being reported in recent years, a few even amongst cases with typical onset and high swinging temperature; these recoveries may be due to more accurate and earlier diagnosis, followed by stricter rest therapy.

Some patients live for 6 months to 3 years before a fatal termination, others recover completely after prolonged recumbent treatment. The disease is often, but by no means necessarily, bilateral, and the individual foci vary in size from a millet seed to a small green pea. Fish found there was the same pathological condition in both types of illness, and could draw no sharp line of demarcation between the two conditions. He reported 10 cases under 14 years, of whom 4 recovered. Of the fatal cases, 3 died in 5 to 7 months' time, and 3 improved so well that they were allowed up after a normal temperature for 2 months; after getting up, they immediately got relapses and died in a short time. Hoyle and Vaizey collected from the literature 30 cases in children aged 0 to 14 years; all proved fatal in the end, although some showed remissions. Roberts and Nassau (1945) discuss chronic disseminated tuberculosis fully and report 9 cases, with recovery in 4. Price has had recovery in 5 cases of chronic miliary tuberculosis, ages 1, 2, 3, 6, and 9 years. In these bed-rest was enforced for 2 to 4 years according to the severity of the case. It is 2 to 5 years since

treatment ceased in the four younger children, who now show firm calcification and are attending school; calcification in lung foci is disappearing, but remains in hilar glands. The nine-year-old girl had a swinging temperature up to 105° F. for 4 weeks in March, 1944, radiograph then negative; suspected miliary foci seen in July, 1944, temperature then 99° F; in November, 1944, when clinically she was beginning to improve after her long and severe illness, the radiograph showed definite widespread miliary foci; in 1945 and 1946 the chest radiograph was clear, and she developed and healed a hæmic cervical adenitis. She is still recumbent on a spinal frame for a lesion of the 1st and 2nd lumbar vertebræ, first manifest in January, 1946.

Experience shows that very prolonged and absolute rest is necessary for healing in these cases.

Signs and Symptoms.—Physical examination is often negative, especially in young children. There may be fine rales in a proportion of cases; there may be a dry cough, and if there is sputum, tubercle bacilli will be present during the active stages; gastric lavage will provide evidence of tubercle bacilli in many cases, but not in the healing stages. The temperature varies: during activity it will be raised; but there are afebrile periods when disease in the multiple foci is arrested and partial healing takes place. As healing advances, the temperature remains steady at normal, but this does not indicate complete healing; reliance must be placed rather on the radiological appearances of calcification in the individual foci. The sedimentation rate, like the temperature, may be within normal limits, and is not to be relied on as an indication of complete healing. The clinical picture varies; some children appear sick and listless, others present a rather healthy appearance during quiescent periods, which can be most deceptive unless the chest is X-rayed. Healing is by calcification, only the larger foci leaving radiologically visible residue. A fatal relapse usually shows the clinical picture of acute miliary tuberculosis, or of meningitis.

Example.—William G., aged 2½ years. X rays showed widespread caseous foci over both lungs. His parents refused treatment and resented interference. After 2 years they were persuaded to allow him to be re-X-rayed, and there was no change in the picture. He died, aged 6 years, having played about at home during the intervening period, with what the parents persisted in calling "chronic asthma".

Example.—Pauline M., aged 1 year 10 months. Admitted to hospital February, 1938. History of two attacks with high fever during past

10 months ; contact, half-sister aged 15 years. Hamburger negative, Mantoux 1–100 positive. Radiograph of chest (*Fig.* 41A) showed miliary tubercles throughout both lungs. Weight 18 lb. 9 oz. Evening rise of temperature to 102° F. every second or third day. Gastric washings examined by culture, human type of tubercle bacillus present. In June, 1938, Hamburger positive ; S.R. 8 mm. per hour. She was kept at absolute bed-rest until June, 1939, but not on a frame ; she was then aged 3 years, temperature normal, S.R. 8. Weight 36 lb. 10 oz. Culture of gastric washings negative. X rays (*Fig.* 41B) showed reduction in number and size of the miliary tubercles. Allowed to sit up in bed for meals. In October, 1940, aged 4½ years, she appeared in splendid health, S.R. 8 mm. per hour, stood up in cot but never got out. After sanatorium treatment for 3 more years good calcification was seen at the roots, nearly all lung foci had disappeared. 1947, very well, at school.

Diagnosis.—Diagnosis can be certain only in cases that give a definite radiological picture of miliary tuberculosis ; sometimes fine stippling is seen (reticular strands) ; suspicions may be confirmed years later by the appearance of multiple small calcified foci. Positive tuberculin test (hard to elicit owing to lowered allergy in active stages), physical signs similar to bronchitis during active phase, positive gastric washings, fever during the acute stage, the X-ray picture—all these must be considered when forming an opinion.

Treatment.—*See* p. 130.

Confluent Caseous Pneumonia (Acute Bronchopneumonic Phthisis, hæmic type).—This infection is hæmatogenous and affects the lobular spaces ; the foci are often bilateral in distribution. The difference between acute miliary tuberculosis and hæmic bronchopneumonic phthisis is one rather of size of foci than of any aetiological or clinical divergence ; in the bronchopneumonic type larger areas of lung tissue are involved. The condition is seen occasionally in young children, but is much more rare than either acute miliary tuberculosis or acute tuberculous lobar pneumonia at these ages. Often physical signs are indefinite or absent, but at times rales will be heard, with impairment of percussion note over the whole of both lungs. The radiological picture of coalescing caseous masses is typical. The temperature is raised. The prognosis is hopeless ; wasting takes place, and the duration of the disease is only a few months.

Localized Spread

There are varying types of localized spread, which may be classed according to degree of involvement into *discrete miliary*

tuberculosis and *isolated secondary foci.* In practice amongst young children many varieties of clinical picture of localized lung and extra-pulmonary spread are seen, from the solitary focus to multiple involvement; indeed, it is impossible to enumerate all the different foci and combination of foci, for every part of the body is vulnerable. One sees at the same time as a primary lung lesion either one or more of the following: spina ventosa, tuberculides on the limbs, bone, often spine, joints, mediastinal glands, and multiple skin abscesses, all of which conditions are rather benign; but also one sees more malign conditions, chiefly those where a localized area of lung is involved by miliary dissemination (*Figs.* 21, 43). Thus it must be borne in mind that all young children suffering from primary tuberculosis, and especially where the upper mediastinal glands are heavily involved, are liable to hæmic spreads; the patient must be protected as far as possible during the early stages from intercurrent illness and fatigue, which would tend to precipitate such hæmic spread.

Example.—Jimmy F., aged 1½ years (*Fig.* 42A). Admitted to hospital suffering from a tuberculous primary infiltration at the right base, massive hilar and paratracheal glands, and 'bull-neck' secondary cervical glands with associated scrofuloderma; tonsils oozing pus; S.R. 55. After three months' conservative (bed and open air) treatment without improvement, we decided on tonsillectomy, fully aware of the risk. Immediate improvement in the cervical glands and general condition took place, but two months later he developed metastases in two metacarpal bones with several subcutaneous abscesses. He recovered completely after two years' open-air rest treatment (*Fig.* 42B).

Discrete Miliary Tuberculosis of the Lungs.—An area of lung, usually the apex, is involved in these cases. The number of discrete miliary nodules is limited. A few foci may be the residue of a previous extensive dissemination (Pagel). It is important to recognize the condition, for it may affect adversely an otherwise straightforward primary infection; the majority of cases heal along with the primary complex, if given rational treatment. The clinical appearances of discrete miliary tuberculosis are not characteristic; the condition can be recognized solely by the radiograph; even so the diagnosis is not certain in every case, and may only be suspected. Healing is by fibrosis or calcification; if healing does not occur, localized caseous pneumonia may supervene with fatal issue; alternatively the general circulation may be flooded with repeated doses of tubercle

bacilli, with resultant dissemination and extra-pulmonary metastases.

Example.—William P., aged 5 years. In good health. Hamburger positive. X rays (*Fig.* 43) show calcified primary complex in lower part of right upper zone with calcified discrete miliary spread in right upper zone.

Isolated Secondary Foci.—There are two isolated post-primary foci in the lungs, hæmic in origin, which have to be considered with special attention, as they may be the starting-point for bronchogenic phthisis.

The Apical or Simon Focus is usually single; occasionally two or three occur together; they appear at one or both apices of the upper lobes. If benign, they are seldom noticed until fully calcified at school age (*Fig.* 16); then they are usually found during the course of routine X-ray examination of positive-reacting school-children. According to Simon the focus was observed in about 5 per cent of positive reacting, and in 15 per cent of phthisical children; they are sometimes found associated with extra-pulmonary lesions. Their further development will be discussed in the chapter on PHTHISIS (p. 109). Differential diagnosis from a primary focus is made thus: (1) Rarity of primary foci in the apex; (2) Presence of primary focus elsewhere; (3) By absence of gland changes (*Fig.* 17).

The Infraclavicular or Assmann Focus.—Some of these foci are hæmic in origin, others are due to bronchogenic spread from a Simon focus (Pagel); exogenous superinfection may develop in particularly heavy contact (but many foci attributed to exogenous superinfection are in reality late primary lesions). It is from the Assmann focus that bronchogenic adult-type phthisis originates; these foci have a marked tendency to rapid caseation and cavitation. The focus is recognized as a small round infiltration in the infraclavicular zone without corresponding glandular enlargement (*Fig.* 19). It will be further considered in the chapter on PHTHISIS (p. 110). Tubercle bacilli have been found in histological sections of healed post-primary foci at autopsy in up to 50 per cent of cases (Pagel).

TUBERCULOUS MENINGITIS

Tuberculous meningitis has been classified in various ways by different authors. Speaking broadly, cases which occur during childhood fall into three main categories: (1) Terminal

meningitis, associated with advanced miliary disease; (2) Traumatic tuberculous meningitis; (3) Acute tuberculous meningitis, associated with a fresh primary complex.

1. Terminal Meningitis.—Occurs in a certain proportion of cases where the child is suffering from miliary or tertiary tuberculosis of the lung. The miliary disease may have been of moderate or of long duration before the meningeal symptoms usher in the end. Such cases may be dismissed after this brief explanation, because the meningeal involvement is but one manifestation of widespread dissemination to other organs. The duration of this type of meningitis is usually only a few days; choroidal involvement is not infrequent. Symptoms may range between well-defined convulsions, head retraction, strabismus, fixed pupil, and unconsciousness, to milder degrees of the same symptoms with an occasional convulsion or twitching. The appearance of the meningeal symptoms merely hastens the end of an already fatal disease. The cerebrospinal fluid may be typical or atypical.

2. Traumatic Meningitis.—These cases are not very common, and are precipitated by a fall or blow on the head. The distinction is a clinical one. One or two cerebral tubercles have been seeded during primary (and rarely post-primary) infection; they escape notice at the time unless they produce symptoms; they heal partially; but as a result of trauma at some later date they reactivate, and may discharge tubercle bacilli into the meninges or the meningeal spaces. The effect is similar to that of massage or injury to a tuberculous joint. The course of traumatic meningitis is the same as that of an acute tuberculous meningitis, but it develops years rather than months after primary infection. Adequate treatment of the primary complex will avert disaster in many cases; in the following example the treatment period was too short to heal this hæmic seeding.

Example.—Philomena D., aged 4 years, was admitted to hospital in October, 1935, for mediastinal gland tuberculosis, and received three months' treatment. X rays showed activity of left upper hilar glands. In March, 1937, she was quite well, with clear radiograph. In March, 1939, she was readmitted suffering from tuberculous meningitis; a few weeks previously she had fallen on her head, and this caused rupture of a cerebral tubercle, presumably seeded in 1935.

3. Acute Type of Tuberculous Meningitis.—This is of great importance and will be considered in the remainder of this chapter in some detail. Cases are met with in pædiatric and

family practice, and in the hospital wards ; they are one of the disasters of medicine. Often this illness is the first indication that the child has been infected by the tubercle bacillus ; it may be also the first indication that there is an adult with open phthisis living in the same house. Cure being impossible, interest is directed towards the possibility of prevention ; this necessitates a knowledge of the aetiology of the condition.

When we speak of tuberculous meningitis in children, we mean the acute and fatal disease which appears early in the stage of hæmatogenous spread, and also early in the whole disease picture. The meninges are involved within a few months or even weeks after primary infection in either thorax or abdomen ; thus we find the victims of this disease are well-nourished healthy children, in whom no suspicion of tuberculous infection would arise. The early supervention of the meningitis allows no time for wasting.

The incidence of tuberculous meningitis is highest under 3 years of age, and it becomes progressively lower as childhood advances. It is seldom seen under 3 months, but has been known to occur at that age. It is most commonly seen between the ages of 9 months and 2 years. In Dublin hospitals amongst 131 cases, 62 were in the 0–3 year, 42 in the 3–14 year, and 27 in the 15–25 year age periods. During the year 1944 in Eire, 118 children under 5 years (31 of whom were under 1 year) and 107 children aged 5 to 15 years, died of tuberculosis of the central nervous system.

Wallgren found tuberculous meningitis to be a complication of a fresh primary tuberculosis in 82 to 87 per cent of cases in children. Engel found it the typical death in occult tuberculosis. Engel, Stern, and Newns found, amongst 41 cases of primary abdominal tuberculosis which developed tuberculous meningitis, only 4 where there was a history of illness extending over a period of two months ; the first sign was usually the onset of meningitis. Nobécourt found that 87·5 per cent of meningeal cases aged 0–15 in Paris had a definite recent pulmonary lesion. Price, in Dublin, amongst 20 cases under 3 years, found that all developed meningitis within one year of the first infection : post-mortem examination showed 1 unhealed primary lung focus, with commencing miliary spread, 1 abdominal, and 18 with fresh primary focus in the lung. These figures are quoted to show that clinical and post-mortem examinations place tuberculous meningitis in the category of a complication of

fresh primary tuberculosis in the vast majority of cases. The majority of these cases in children are associated with recent pulmonary infection, and a small proportion have the infection situated in the abdomen.

Huebschmann stated that 75 per cent of cases of miliary tuberculosis develop tuberculous meningitis. Other observers find miliary disease present in many cases; McGregor found it in 83·3 per cent of 70 cases of tuberculous meningitis; Blacklock in 68 per cent of cases of tuberculous meningitis found coexisting miliary disease. However, in Dublin, Price found clinically, in children under 5 years, that 84 per cent of miliary cases did *not* develop meningitis or cerebral symptoms, and only 23 per cent of the meningeal cases showed radiological signs of miliary disease. It is probable that, as in other tuberculous conditions, so also in meningeal cases, different countries show variations as to the extent of pulmonary and abdominal involvement. The greatest danger of acute tuberculous meningitis occurs during the first 3 months after primary infection; this danger diminishes as encapsulation and calcification proceed (*see Figs.* 55A–55C). Wallgren first showed that after the lapse of the first 3 months the danger of meningitis lessens; now that the onset of primary infection is more accurately diagnosed, many agree with him. He reports 23 cases of tuberculous meningitis in which rational treatment had been given to the primary focus in only 3 instances; in contrast to this is his report of 428 children treated for primary tuberculosis, of whom only 3 developed meningitis. Amongst 300 cases of primary tuberculosis of the lungs under 5 years, treated by Price, one developed meningitis.

A boy aged 7 years was negative to tuberculin on repeated testing; three months after the last test he was seen suffering from tuberculous meningitis. Another boy of 6 with a history of untreated erythema nodosum was seen two months after its occurrence suffering from tuberculous meningitis. A contact aged 5½ months was admitted to the B.C.G. isolation unit in St. Ultan's Hospital with a view to vaccination; he was then negative to Mantoux 1–100. During the second week of his prevaccination isolation period he became Mantoux positive, and although the chest film was negative he was treated for primary tuberculosis and not vaccinated. Exactly eight weeks after he became Mantoux positive, he developed tuberculous meningitis and died, aged 8 months.

Certain dates should be borne in mind when seeking a source of infection. Skin allergy is established 4 to 6 weeks after the bacillus first enters the body; meningeal involvement may be clinically apparent soon after allergy appears, i.e., from 2 weeks to 4 months; thus one must look back 2 to 5 months from the date of the meningitis to find the contact; on some occasions the interval is longer.

Example.—James C., who resided in the country, developed tuberculous meningitis, aged 9 months. His only brother had died of the same disease 18 months previously. The father had been in a sanatorium and was healed for 4 years. Further inquiries revealed that the mother's brother three months previously had discharged himself from a sanatorium and had gone to stay with the C. family. (Some months before his death the elder child had been staying with his mother's relations, where this uncle resided.) The uncle was therefore the source of infection in both cases. A radiograph of James a few days before his death is shown in *Fig.* 44. At autopsy was found a fresh primary lung focus in the right upper lobe, with commencing caseous pneumonia in its immediate neighbourhood; right hilar and paratracheal glands caseous; left lung normal; abdomen free from tubercle. The appearances of the lesion fitted in with the time when contact with the uncle had taken place.

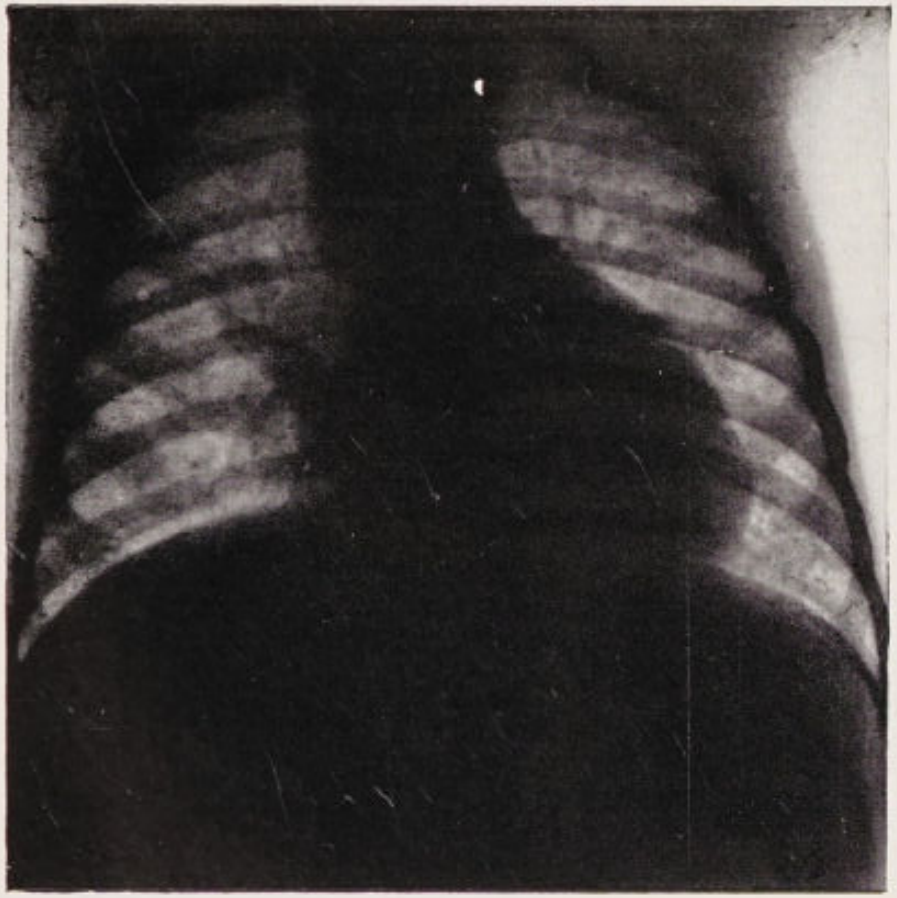
Infection of Meninges.—There are three possible modes of infection of the meninges:—

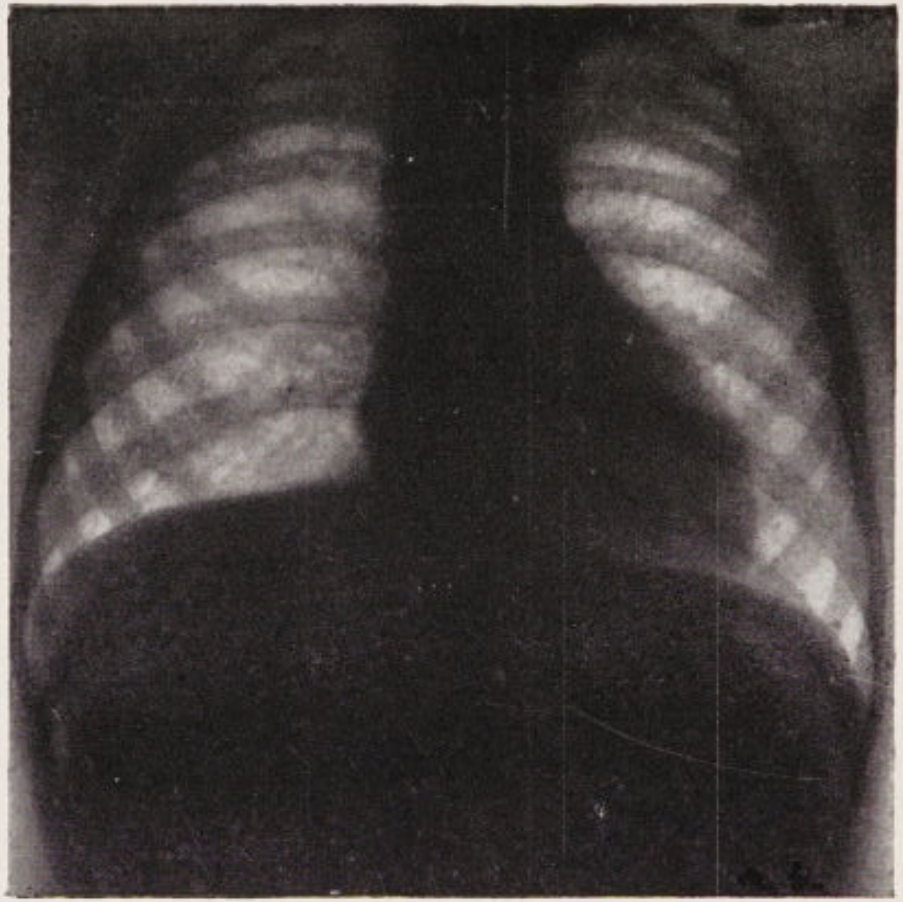
1. Multiple miliary foci in meninges, infection carried direct to meninges by the blood-stream from a fresh focus in lung or abdomen.

2. Formation of one or a few microscopic tubercles in the same manner as (1) in the cortex of the brain, cord, and bones adjacent to the central nervous system; these may extend to the meninges or erupt into the meningeal space.

3. Formation of one or more macroscopic cerebral tubercles during the course of a bacillæmia; these are not clinically apparent at the time, but if the tuberculoma lies near the surface, it may rupture into the meninges at a later date, usually following trauma; alternatively it may increase in size and cause symptoms (*see* p. 102).

Rich considers that direct hæmic infection of meninges (1) seldom occurs and cannot be produced in animal experiment. Rich and McCordock consider spread from a caseous tubercle (2) to be the usual manner of development. They described in one-fourth of their series microscopic tubercles of the meninges (plaques), often solitary. In the remainder of cases they found caseous tubercles of the cortex, 3–5 mm. in diameter, often single;

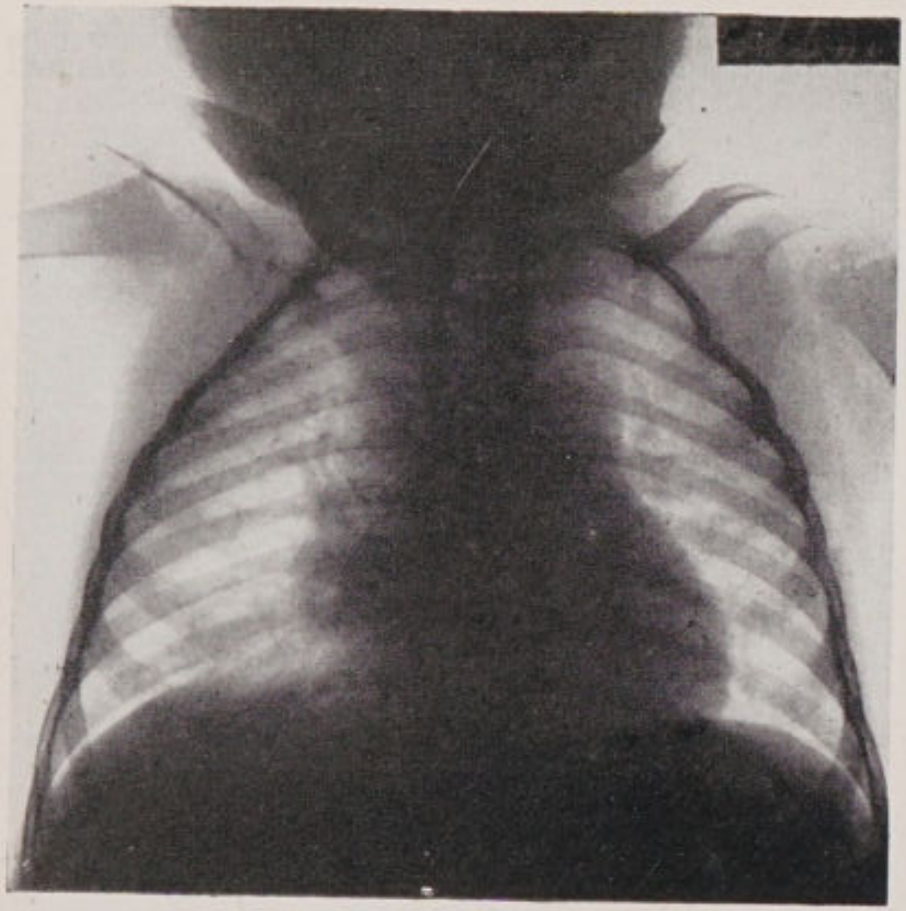


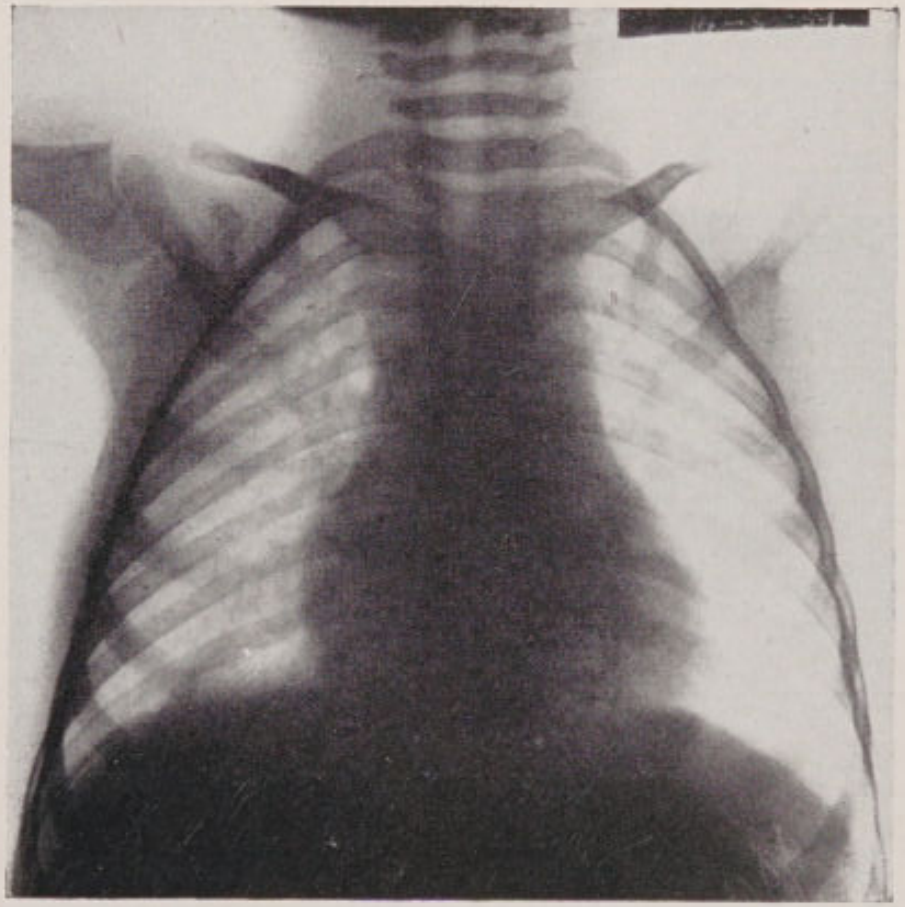


FIGS. 41A, 41B.—CHRONIC MILIARY TUBERCULOSIS.

Fig. 41A.—Pauline M. March 12, 1938; aged 1 year and 10 months. (*See Example*, p. 89.) Fine miliary spread throughout both lungs; enlarged right paratracheal and hilar glands.

Fig. 41B.—April 20, 1939; aged 3 years. Calcification commencing in many of the miliary foci; paratracheal gland reduced in size.





FIGS. 42A, 42B.—LUNGS IN ACTIVE Type II TUBERCULOSIS.

Fig. 42A.—Jimmy F. Aged 1½ years. (*See Example*, p. 91.) Primary focus at right base; massive enlargement of all mediastinal glands; suffering from multiple extra-pulmonary metastases.

Fig. 42B.—March 14, 1939; aged 3¼ years. Glands resolving.

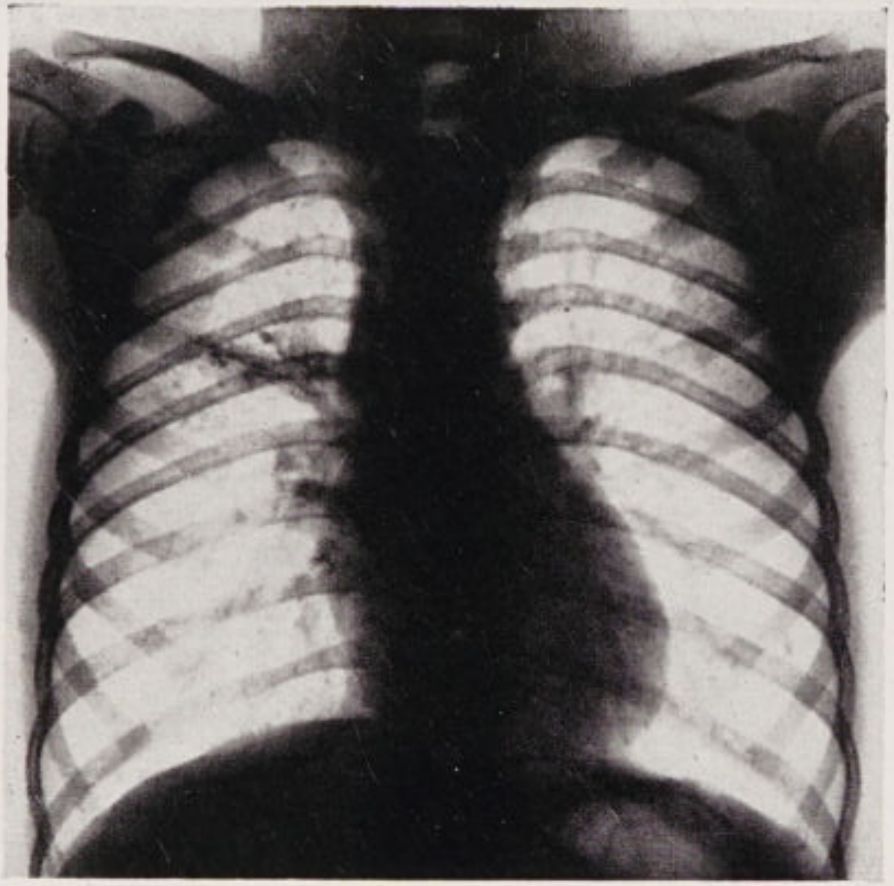


FIG. 43.—DISCRETE MILIARY TUBERCULOSIS.

William P. Aged 5 years. (*See Example*, p. 92.) Calcified primary complex in lower part of right upper zone; discrete miliary spread, very fine and calcified, throughout right upper lobe around the primary focus and also below the clavicle.

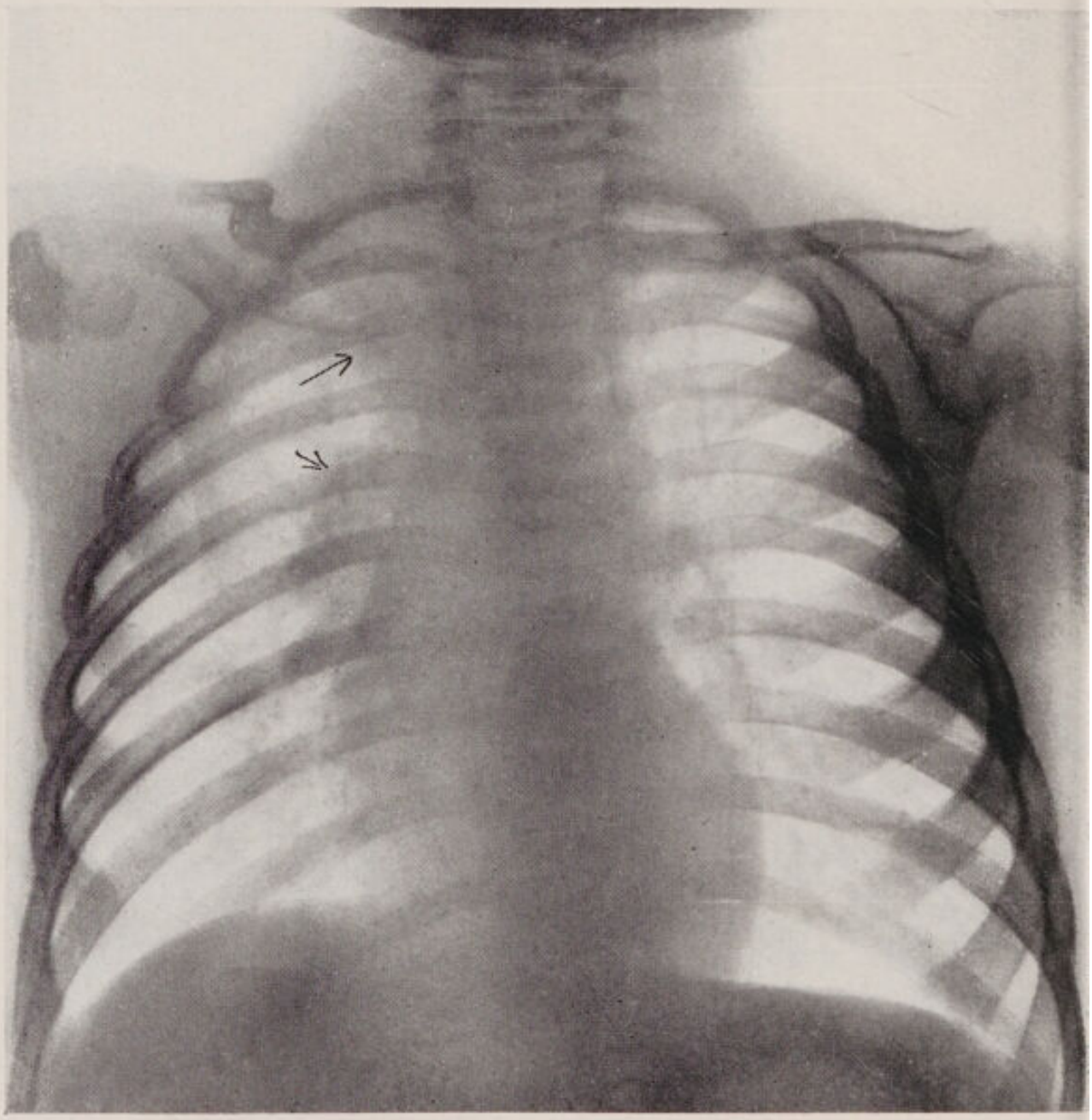


FIG. 44.—TUBERCULOUS MENINGITIS.

James C. Aged 9 months. (*See Example*, p. 96.) The arrows indicate enlarged hilar ar paratracheal glands. There is some infiltration of the right upper lobe: a primary focus con mencing to caseate. Confirmed at autopsy.

these extended into the overlying meninges and the meningeal spaces. MacGregor and Green are in agreement with these findings. Rich and McCordock found in 77 out of 82 cases of tuberculous meningitis that focal caseous lesions, older than the meningitis, were in communication with the meninges; in 75 of these cases the caseous foci were situated in the brain or in the meninges, in one the source was in a tuberculous vertebra, and in one it was a caseous mass in the choroid plexus. Engel, Stern, and Newns found in London that amongst 284 cases of tuberculous meningitis 35 (12·3 per cent) showed tuberculomata of the brain; only 1 tuberculoma occurred amongst the 41 abdominal lesions of the series, the remainder being associated with primary lung lesions. Rich, in *Pathogenesis of Tuberculosis*, describes the basis of symptoms in tuberculous meningitis as due to five effects on the central nervous system—namely, mechanical irritation, hypersensitivity, vascular obstruction, extension of the infection to the nervous tissue, and increase of intracranial pressure.

Seasonal Incidence.—Some observers, notably Engel, find a seasonal incidence for this disease, the peak points occurring in the early spring and autumn. If this is a fact, it falls in with the theory that all exudative diseases are more manifest at those seasons. On the contrary, if tuberculous meningitis follows within two or three months after a primary infection, it is not very clear in what way seasons can influence the incidence, unless it be that these special seasons have an effect in causing a primary infection to generalize.

Human and Bovine Types of Infection.—Primary pulmonary infection is responsible for most of the cases of tuberculous meningitis which occur amongst city dwellers. The incidence of abdominal cases is increased in rural areas. The question of the type of organism found must now be discussed. Clinical evidence is usually derived from urban material, and is weighted on the side of a pulmonary infection, with a known human contact. It must also be remembered that occasionally cases of pulmonary tuberculosis are due to the bovine type of bacillus, and cases of abdominal, and more rarely cervical gland, tuberculosis are due to the human type. The evidence supplied by research workers who have typed the bacillus from specimens of cerebrospinal fluid in children shows a rather high percentage of bovine strain. In Eire, Mushatt (1940) amongst 12 cases of tuberculous meningitis found: 0–4 years, 6 human and

1 bovine ; and 5–14 years, 3 human and 2 bovine. In Scotland, Blacklock (1935) found that of 114 children who had died from tuberculous meningitis 24·6 per cent were due to the bovine type. Griffith's compilation of Scottish statistics (1937) showed that amongst 203 cases 34·4 per cent under 5 years and 14 per cent aged 5–15 years were due to the bovine type. In England, Griffith (1937) compiled 265 cases of tuberculous meningitis, and found that 28·1 per cent under 5 years and 24·5 per cent aged 5–15 years were due to the bovine type of bacillus. Topley and Wilson state that one-quarter of all cases of cerebral tuberculosis and tuberculous meningitis appear to be due to the bovine type of bacillus. MacGregor and Green, in south-east Scotland (1937), found the bovine type in tuberculous meningitis in 30·1 per cent aged 0–4 years, and in 21·2 per cent aged 5–14 years. K. A. Jensen's figures from Denmark are shown in *Table XIV*, and demonstrate the high incidence of bovine infection in agricultural as against city areas.

Table XIV.—Bovine Bacillus in Cerebrospinal Fluid (*K. A. Jensen, Copenhagen*, 1935)

	Bovine	*No. of Cases*
Copenhagen	17·9 per cent	84
The Islands	31·6 ,,	19
N. & E. Jutland	40·0 ,,	45
S. & W. Jutland	67·9 ,,	28

Clinical.—The clinical picture of the disease varies according to the age of the patient. The younger the child the more insidious the onset, the more rapid the course, and the less typical the signs and symptoms. In small babies under 2 years, early diagnosis is often made only by exploratory lumbar puncture. The course in the older child follows the text-book type more closely. From perfect health, within two or three days, the child ceases to play, becomes unhappy, and unduly irritable, and shows anorexia and an inclination to sleep. Vomiting occurs in 87 per cent of cases (Engel), with photophobia and complaints of headache or abdominal pain in the older child. There is slight fever, 99° to 100° ; an inclination to constipation is more or less beginning. At this stage vomiting and headache become more strongly marked, and are almost always present. After a few days, suspicion is further aroused by the appearance of symptoms of sensory disturbance, notably hypersensitivity to light, sound, and touch, some degree of neck stiffness, with increased vomiting and headache. At the end of a few more

days, definite meningeal symptoms appear: Kernig's sign (inability to straighten the flexed knee when the thigh is flexed over the abdomen); Brudzinski's neck sign (flexion of the legs on passive flexion of the neck); twitchings or convulsions; paralysis of facial and eye muscles, the latter resulting in ptosis and strabismus; vagus irritation giving rise to slow irregular pulse; bulging fontanelle in infants, and cephalic cry in older children; dermographia; and fever of varying degree, from 99° to 102°. Constipation is usually present. This stage of deepening unconsciousness progresses to complete coma, with loss of all reflexes; the pulse becomes rapid, pupils fixed; hyperpyrexia and often Cheyne-Stokes respiration usher in the end. The duration of the disease is from two to four weeks, the average being three weeks. In infants the disease runs a more rapid course; often from the first recognition of the symptoms until death supervenes only one week elapses. At times during the course of the disease, there occurs temporary slight remisison of symptoms. Diagnosis is made rather late in the case of infants owing to the indefinite clinical picture; nevertheless the course is actually, as well as apparently, more rapid than in older children. It is important to remember that *any* sort of neurological symptoms may be produced by tuberculous meningitis, including decerebrate rigidity. It is the fact of these symptoms, rather than the kind, that is significant and makes one suspect tuberculous meningitis.

The Cerebrospinal Fluid.—When the disease is fully developed the cerebrospinal fluid is characteristic, but at an early stage the findings may be indefinite, and other factors have to be considered. Fluid on puncture generally comes off under pressure, in advanced cases under considerable pressure; it is clear, and on standing for one hour a fine cobweb coagulum, due to fibrin, forms; the coagulum is typical of tuberculous meningitis, although it occurs at times in encephalitis. The changes in the fluid are: increased globulin; glucose reduced except in the very early stages; chlorides reduced from normal of 700 to about 600 per 100 c.c., or below 0·65 per cent. The cell-count is characteristic, always showing lymphocytes, which vary from 30 per c.c. to several hundreds. If there are innumerable cells present the fluid will have a milky appearance. Tubercle bacilli may or may not be found by direct method, often not without culture. From the diagnostic point of view, the main characteristics are: clear fluid coming off under pressure (which

can be measured by a manometer), coagulum formation on standing, decreased chlorides, lymphocytes predominating over leucocytes, increase of globulins, and decrease of sugar; the two latter are present in all forms of meningitis.

Diagnosis.—Tuberculosis must be distinguished from other forms of meningitis by examination of the cerebrospinal fluid, chest radiography, the tuberculin test, and by clinical appearances. History of contact with a phthisical person three months previously is helpful. The fluid has already been discussed. Early in the disease the tuberculin test will be positive to weak dilutions; fully established cases, however, are often negative to the percutaneous test, although nearly all react positively to Mantoux 1–100 (lowered allergy). Where negative reaction to high dilutions is associated with characteristic fluid and a fresh focus or early miliary lesion in the chest radiograph, the case must be regarded as in a state of terminal anergy. A radiograph may give immediate confirmation of pulmonary involvement; in a small proportion of cases this will be negative, although at necropsy a tuberculous focus will be found in the lungs or abdomen.

Differential Diagnosis.—

Meningococcal meningitis yields cloudy fluid with typical organisms. The course of the disease is prolonged, lasting in some cases three months, with cure in a high percentage of treated cases.

Pneumococcal, influenzal, and streptococcal meningitis show cloudy fluid with typical organisms. The course is rapid and fulminating, fatal cases living only a few days.

Meningismus: In infants chiefly, but also in older children, this condition simulates tuberculous meningitis. Cerebrospinal fluid comes off under pressure and is quite clear, but on examination is found to be normal. This condition is found associated with pneumonia (usually apical), tetany, and digestive disturbances. Convulsions may occur along with other meningeal symptoms. The chest radiograph will be normal, except where there is associated pneumonia; in this event differential diagnosis must rest on physical examination, the fluid analysis, and the result of the tuberculin test in two dilutions. After one or two lumbar punctures have relieved the meningeal symptoms, these will disappear with the subsidence of the causal disease.

Encephalitis: Differential diagnosis here is difficult. One must rely on a negative tuberculin test and chest radiograph,

and on the examination of the fluid; the latter if clear, as in encephalitis lethargica, will show but slight deviation from the normal.

Acute aseptic meningitis (serous meningitis; acute benign lymphocytic meningitis): This condition presents a real problem in differential diagnosis. It is of rare occurrence, and the course is benign. It must be considered in every case where the cerebrospinal fluid is suggestive of tuberculous meningitis, when the tubercle bacillus has not been recovered; the chlorides are not reduced in this condition; on these two points is based the diagnosis. Clinically, the onset is somewhat similar to that of tuberculous meningitis, but more rapid; there are headache, vomiting, sluggish pupils, and stiff neck. The fluid is quite clear, comes off under pressure, shows marked cell increase (lymphocytes), and increased globulin. The tuberculin test if positive, as may happen amongst older children, will not be accompanied by any radiological evidence of a recent pulmonary focus. These cases are relieved by lumbar puncture repeated as often as may be necessary, and they recover in two to three weeks.

Treatment and Prognosis.—There is no treatment, but reports of the action of streptomycin show some promise. In the author's opinion, the only weapon known to us at present for combating this dread disease lies in the prevention of uncontrolled primary tuberculosis by the employment of B.C.G. vaccination amongst negative reactors, principally home contacts and particularly newborns. Relief may be afforded by repeated lumbar puncture, but even this is useless once coma has set in. Chloral hydrate by rectum, morphine hypodermically, luminal intravenously, may be tried, but they do little to mitigate the severe headache and convulsions. Puncture may be performed by the lumbar route in infants; if, after repeated puncture, entry is difficult the cistern route may be used; in infants with gaping fontanelle, ventricular puncture will also relieve fluid pressure.

The prognosis is hopeless. In published series the mortality is 100 per cent. Cremer and Bickel collected 250 cases from the world literature where recovery from this disease was claimed; 60 of these were stated to have tubercle bacilli in the cerebrospinal fluid; some of the reported cases recovered only temporarily, and showed relapse with fatal termination a few months later.

Cerebral Tubercles.—Macroscopic cerebral tubercles may occur in children; these may calcify and never produce symptoms, or they may give rise to meningitis (immediately or at a later date), or they may cause cerebral symptoms without meningitis. Those causing meningitis have already been discussed; they are usually microscopic in size, and are associated with a primary lung focus, rarely with miliary disease of the lungs. The symptoms of tuberculomata are those of cerebral tumour, the tuberculin test is positive, and the radiograph may reveal a focus in the lung. It is important for two reasons to recognize that calcified granulomata are tuberculous: (1) when the symptoms are those of brain tumour (tuberculoma) ventricular puncture is safer than lumbar puncture; (2) if such tuberculomata are operated on there is a danger of fatal tuberculous meningitis; but if not operated on they die. Decompression saves some cases. Scott reported 9 cases in which the site in order of frequency was as follows: (1) cerebellum, (2) cerebrum, (3) cerebellar peduncles, (4) base of brain. His cases usually began as small nodules situated on the pial sheath of single vessels, and some reached an inch in diameter. Stern classified 6 per cent of all cerebral tumours in children as large tuberculomata; 3 of 6 cases were operated by decompression and did not develop meningitis; only 2 of these 6 cases developed meningitis.

Example.—William S. (Mother died of pulmonary tuberculosis Dec. 8, 1937.) Admitted Oct. 26, 1937, aged 5 months. Hamburger positive. Radiograph (*Fig.* 45): primary focus in right apex. Fluid suspected in left base. S.R. 30. Feb. 15, 1938, convulsions. Feb. 23, 1938, died. Autopsy: Pleura adherent both sides especially left. Caseous focus the size of a cherry at right apex, caseous glands in right hilum and right paratracheal area. Brain showed tuberculous meningitis of base. A caseous tubercle, removed from lower posterior surface of right lobe of cerebellum, typed human bacillus.

CHAPTER IX

THE THIRD TYPE OF TUBERCULOSIS: ISOLATED PHTHISIS

ACUTE PNEUMONIC PHTHISIS: EARLY FOCI: BRONCHOGENIC PHTHISIS.

General Remarks.—Bronchogenic phthisis, as we are accustomed to see it in the adult, is rather unusual in the child. Price (1938), in Dublin hospitals, amongst 450 positive tuberculin reactors under 14 years found that 3 per cent were suffering from bronchogenic phthisis. Genevrier, Paris, 1936, in mass examination of 1500 school children found 0·2 per cent; Kayser-Petersen, Jena, 1934, amongst 2900 school children found 0·5 per cent; and Chadwick, Massachusetts, 1935, amongst 400,000 school children found 1·5 per cent to be suffering from pulmonary tuberculosis. Although rare, bronchogenic phthisis may be seen at any age between birth and 14 years; *Table XV* demonstrates

Table XV.—Analysis of 53 Tuberculous Deaths from the Tuberculosis Unit, St. Ultan's Hospital (Price, 1946)

Type of Tuberculosis	0–1 year	1–5 years	Total 0–5 years
Miliary, acute	11	12	23
,, chronic ...	—	1	1
Primary cavitation ...	3	4	7
Meningitis	1	4	5
Bronchogenic phthisis ...	1	6	7
Aspiration pneumonia ...	4	—	4
Generalized dissemination	1	2	3
Primary complex ...	2*	—	2
Amyloid disease ...	—	1	1
	23	30	53

* Aged 8 and 11 weeks at time of death.

the distribution of 53 deaths in the 0–5 age-period; post-mortem confirmation of the diagnosis was obtained in half the cases.

There is no doubt that early diagnosis followed by adequate treatment of the primary lesion will prevent the development of more serious forms of tuberculosis in many children. In most countries, however, during the past seven years war conditions have upset such measures, and consequently there has occurred generally an increase of childhood phthisis rather than the decrease which would be expected to follow an enlightened outlook on the subject. The shorter the interval between primary implantation and the development of tertiary phthisis, the more rapid and fatal will the latter disease tend to be, for with healing of the primary complex so also will heal any possible seeded hæmic foci, seldom radiologically demonstrable at this stage yet constituting a potential danger. With an unhealed primary complex the child remains in a state of hypersensitivity, and should an early infraclavicular (Assmann) focus occur, a rapid-spreading and ulcerative type of phthisis may follow in a very short space of time. In this way phthisis in the child differs from phthisis in the adult, although a condition closely resembling that in the child may be seen in the adolescent with recently converted tuberculin reaction, for example, young probationer nurses who enter hospital without having passed through their primary infection. The above explanation shows that the division of phthisis into acute and chronic, although a suitable one in the adult, is a bad classification for the child. With children it is better to follow one which is based on aetiology. In *Table XVI* are recapitulated, according to their aetiology,

Table XVI.—Pulmonary Tuberculosis in Children, Classified According to Aetiology

Primary Type.—Focal cavitation
- *a.* Acute lobar pneumonic phthisis (1)

Second Type.—Hæmatogenous spread
- *a.* Miliary tuberculosis
- *b.* Acute lobular pneumonic phthisis
- *c.* Post-primary hæmic focus

Third Type.—Bronchogenic spread
- *a.* Aspiration lobar pneumonic phthisis (2)
- *b.* Acute bronchopneumonic phthisis
- *c.* Bronchogenic phthisis
  - i. Ulcerative
  - ii. Fibroid

all the severe forms of pulmonary tuberculous lesions which are found in children ; this table serves to show how small a position in the whole series is held by the chronic type of phthisis which

is so usual in adults; even amongst these 'chronic' types are few which run a course of more than 2 years. Bronchogenic phthisis in the child shows ulcerative and fibrotic processes in varying degrees; both may be present in the same case; on the amount of fibrosis depends the chronicity of the case; in practice, unfortunately, the ulcerative type predominates during childhood years.

ACUTE PNEUMONIC PHTHISIS

Acute Lobar Pneumonic Phthisis.—This condition arises by bronchogenic spread in one of two ways, mainly from a component of a primary complex.

1. When a primary cavity fails to heal, and if death from either toxæmia or hæmoptysis does not end matters early, the cavity will by bronchogenic spread proceed to infect neighbouring tissue, and thus give rise to acute lobar pneumonic phthisis. Price has observed this condition in a few instances in the 1–3 age period; in the 0–1 period the non-healing primary cavity usually proves fatal before such bronchogenic spread has had time to occur. Pagel (1942) has observed this form of acute pneumonic phthisis in young adults.

2. The aspiration type of lobar pneumonic phthisis occurs when a caseating gland ruptures into a large bronchus, and the material is aspirated along the bronchus; thus an area of caseous pneumonia may be found at a distance from the primary focus, sometimes in the contralateral lung. The onset of illness is sudden; from the fair health usually associated with bronchial gland tuberculosis, the child becomes gravely ill with symptoms of acute bronchopneumonia and sudden rise of temperature; very rapidly death supervenes. Children over 3 years may survive (*Fig.* 13, *A–C*). This condition is not uncommon during the first two years of life (*Figs.* 23, 46). The radiological picture may or may not be typical; the following case of aspiration pneumonia provides an example of how the radiographic appearances may underestimate the extent of disease present; diagnosis must depend largely on the grave illness with sudden onset.

Example.—Frank D., aged 7 months. Father phthisical. Admitted to hospital, May 16, 1939. Patch test and Hamburger both positive. Cough, and fever 100° to 103° F. X rays (*Fig.* 46A) show an infiltration of middle zone of left lung, and broad mediastinal shadow representing enlarged glands with evidence of perihilar activity. S.R. 16 mm. per hour. On left, physical signs of dullness with tubular breathing posteriorly and a few rales. June 22, 1939, X rays (*Fig.* 46B) show

a large area of caseous pneumonia over upper and middle zones of left lung, with commencing central cavitation, periglandular activity, and a small area of infiltration in right lung at level of 6th rib behind. General condition worse. Collapse of left lung considered, but not attempted on account of involvement of glands and infiltration in right lung; the latter was regarded as a hæmic post-primary, but was found at autopsy to be the primary focus. Ten days later the child, aged 8½ months, got a severe hæmoptysis, and died instantaneously.

Anatomical report by courtesy of Dr. Walter Pagel (*Figs.* 47A *and* 47B): Primary focus in the subpleural parts of the right lower lobe in anterior axillary line. Hilar glands much enlarged, especially right bifurcation and paratracheal; regional lymph-gland liquefied and broken into the right main bronchus. A large caseous infiltration occupied the whole of the left upper lobe; this infiltration shows gross liquefaction, especially in dorsal parts; origin of hæmorrhage in liquefied area. Spleen interspersed with innumerable nodules; kidney a few nodules; large fat liver. *Comment*: There is a typical primary complex in the right lower lobe. Liquefaction in regional glands at that side shows this as the original focus. The caseous infiltration in which the hæmorrhage originated is apparently a post-primary focus due to aspiration of caseous material into the left upper main bronchus, after eruption of the right bifurcation gland into the right main bronchus.

Miliary Tuberculosis.—*See* p. 85.

Acute Broncho- or Lobular-pneumonic Phthisis.—Lobular-pneumonic phthisis may be either hæmic or bronchogenic in origin.

1. The hæmic type of bronchopneumonic phthisis is described on p. 90.

2. The bronchogenic type resembles ulcerative phthisis, but runs a much more acute course, and together with lobar pneumonic phthisis has been called 'galloping consumption'. The distribution is generally unilateral; spread occurs from a cavitating hæmic focus, usually an Assmann focus in the infra-clavicular region, and travels via the bronchi to neighbouring parts of the lung; soon large areas become involved and the resultant caseous foci tend to cavitate. This very severe illness is characterized by high swinging temperature, and death occurs within three months. The only possible means of effecting a cure is by collapse of the affected lobe or lobes at an early stage. This type of disease is very rare in infants; but it is seen during school age, although at that age it is more rare than is the adult type of disease; but between the years 10 to 14 it occurs less rarely perhaps than does acute pneumonic phthisis. This form of bronchopneumonic phthisis is confined to the lungs, and extra-pulmonary organs escape.

Example.—Veronica H., aged 7 months. Mother phthisical. Admitted gravely ill; dullness and rales over left base. Hamburger and Mantoux 1–100 negative (terminal anergy). Differential diagnosis—acute pneumonia, empyema, atelectasis, caseous pneumonia. X rays (*Fig.* 9A) show shadow over the left lower zone, and infiltration of apical zone. Temperature 102° F. Radiograph (*Fig.* 9B) taken four days before death shows involvement of whole left lung. Autopsy revealed caseous pneumonia of entire left lung, with multiple small cavities throughout. Right lung free from tubercle.

Having considered the acute forms of bronchogenic phthisis as they affect the child, and having already, in the chapter on HÆMATOGENOUS SPREAD, discussed the types of hæmic phthisis, we must refer once more to the solitary hæmic metastasis. This isolated post-primary focus is a potential link between the primary complex and bronchogenic phthisis, not only in the child but also in the young adult. The recognition of such a focus at an early stage is most important if one hopes to prevent the development of Type III phthisis.

THE POST-PRIMARY HÆMIC LUNG FOCUS (EARLY FOCI)

General Remarks.—A post-primary hæmic lung focus is implanted during the course of a bacillæmia. A bacillæmia occurs when tubercle bacilli penetrate a blood-vessel from a primary focus or gland, either directly or from glands via the thoracic duct; this may occur early in primary infection, late in primary infection, or after a lapse of years by recrudescence of activity in either component of the complex; here the complex is apparently healed, but still harbours living tubercle bacilli. There is strong evidence to suggest that hæmic seeding in young age groups takes place very early in primary infection. Price's experience amongst children under treatment is that those who die do so within a year of primary infection; and has even observed the development of tuberculous meningitis in an infant exactly 8 weeks after conversion of the skin test. Wallgren (1935) finds the danger period to be the first three months, decreasing up to one year, after primary infection. In young children mainly, but also in older ones, hæmic seeding would appear to take place very soon after infection, probably before encapsulation of the primary focus is completed; this applies to pulmonary and extrapulmonary seedings. In adults the interval between primary infection and development of bronchogenic phthisis may be rather longer, in which case the

condition may be due to reactivation of an incompletely healed post-primary focus. Pagel (1939) found 50 per cent of healed post-primary foci to contain living tubercle bacilli. A rare example in a child is shown in *Fig.* 16, *A*, *B*.

A post-primary focus has no accompanying enlargement of regional glands as seen with a primary focus ; the tissue reaction around a post-primary focus is usually more opaque to X rays and is restricted to the immediate neighbourhood of the focus, thus differing from a primary infiltration ; furthermore, the tissue reaction around the post-primary focus has a marked tendency to liquefaction and cavitation (cavitation occurs in a very small percentage of primary foci). To understand these differing tissue reactions we must examine Koch's phenomenon, and later discuss the much debated question, " Are post-primary foci due to exogenous aerogenous reinfection or are they due to endogenous hæmic or bronchogenic infection ? "

Koch's Phenomenon.—If tubercle bacilli, living or dead, are injected intracutaneously into non-infected animals, after 11 days an abscess forms at the site of inoculation and after 20 days an ulcer forms. If tubercle bacilli, living or dead, are injected intracutaneously into animals that have previously been inoculated (i.e., a reinfection is produced), in 24 to 48 hours necrosis occurs at the site, and after some days this flat ulcer heals, leaving a scar. A typical Koch's phenomenon is seen two days after B.C.G. has been administered intradermally to an individual who has been already infected with tubercle bacilli.

Exogenous Reinfection (second infection by inhalation from without).—Exogenous reinfection as an explanation of the infraclavicular focus used to be favoured clinically, especially where a known sputum-positive contact existed. Such reinfection is supported by slender pathological evidence. It is based mainly on epidemiological investigation of contacts (Opie and McPhedran) ; the theory presents some points which may be criticized. Many cases of apparently healed exogenous reinfection may be really late primary infections, under the clinical disguise of reinfection phthisis. Reinfection often occurs years after the contact has died, and in many cases the condition has been proved in the absence of any contact whatsoever. However, one cannot completely exclude the possibility of exogenous superinfection in exposed cases where the primary complex has been healed for a number of years. Exogenous and endogenous reinfection are also discussed in Chapter I, p. 3.

Endogenous Reinfection (spread from an existing primary focus in the body).—This mode of reinfection has been demonstrated pathologically. Endogenous spread takes place from a primary focus or gland which may be either active, or partially healed, or apparently healed; the route may be hæmic or bronchogenic; in children it is found that hæmic spread is the more usual; nevertheless, bronchial spread from a latent hæmic apical (Simon) focus may take place by aspiration, with further focus formation in the infraclavicular region; this second focus then becomes the true starting point for phthisis. It has already been pointed out that in childhood we have to deal mainly with endogenous infection of the hæmic type; the interval between primary infection and reinfection is but short, healing of the primary complex is not complete, immunity is poorly developed, and the child is still in a hypersensitive state. At puberty, and later, under adverse circumstances, it may happen that reactivation occurs in an incompletely healed primary complex, thus giving rise to hæmic seedings after a considerable interval of time. Pagel (1939) found 20 per cent of quiescent and calcified primary complexes to contain living tubercle bacilli. All these considerations lead to one conclusion—namely, that, if phthisis is to be prevented, treatment early in primary infection should be directed not only towards healing of the primary complex itself but also towards healing of any possible seedings which may be planted at that time (Price, 1947). These post-primary seedings, visible or invisible, prove abortive in many cases; but the proportion that remain unhealed are a potential danger to their host.

First Hæmic Focus.—

1. Apical Simon Focus.—These foci (*see* p. 92) are hæmic in origin, occur singly (*Fig.* 16) or in groups (*Fig.* 21), and are situated in the apex of an upper lobe. They are either very small and single or larger (up to the size of a pea), or again small and multiple. They arise as post-primary foci seeded during primary tuberculosis. Clinically they cannot be detected, and unless calcified they are hard to see in a radiograph. Pagel believes that "they have a special bearing on the pathogenesis of progressive pulmonary tuberculosis". He produces anatomical evidence that the apical focus can become the original starting point of phthisis; in this event bronchial aspiration from a recrudescent apical focus leads to the formation of the infraclavicular focus. Activity in the apical focus develops slowly in

contrast to the rapid appearance of caseation in the sub-apical focus ; the Simon focus is in itself benign ; there is no certainty as yet, however, as to the importance of the role which it may play as a link in the chain of events leading to phthisis (*see Example*, p. 82).

2. Infraclavicular (Assmann) Focus (*see* p. 92).—Some of these foci are endogenous hæmic reinfection foci, others are due to bronchogenic aspiration from an active apical focus, some few may be due to exogenous superinfection. The Assmann focus occurs nearly always in children in the posterior sub-apical area of an upper lobe. As already mentioned, it is seen as a small perifocal infiltration, without involvement of regional gland (*Fig.* 16) ; unless it heals, the infiltration in the child has a tendency to rapid caseation, with central cavitation (*Figs.* 20, 48). From this cavity by bronchogenic aspiration there commences a typical downward-spreading phthisis, in children generally of the ulcerative type. In a small proportion of cases the post-primary hæmic focus is situated in the apex of the right middle lobe or in the apex of a lower lobe ; these foci also tend towards cavitation, and some turn out to be primary foci, especially in early adolescence. At times one sees a group of small foci in the infraclavicular region with tendency to confluence and formation of an infraclavicular infiltration. In other cases these small nodules remain as such and may calcify ; indeed these foci really constitute a discrete miliary tuberculosis.

Clinical Picture.—The clinical picture of an early infiltration around an infraclavicular (Assmann) focus may not be marked in any way, in spite of the fact of the importance of correct diagnosis at this early stage. Prompt treatment on recognition of this condition will probably save the child from developing bronchogenic phthisis. Similar lesions in situations other than the infraclavicular region behave in the same manner. Onset is not typical ; fever, i.e., commencement of toxic effects, is usually not high at the beginning of the peri-focal infiltration phase, and lasts from 2 to 14 days. The patient has few complaints beyond a feeling of fatigue, but looks ill ; sputum in these early stages is sometimes negative if examined for tubercle bacilli ; there is no cough as yet, unless there is a pleural tear, when cough will be of a hacking character. Physical examination over the affected area of lung generally proves negative ; if adventitious sounds such as fine crepitations and bronchial breathing are present, the underlying condition

is probably more advanced, with commencing caseation. S.R. is not much raised, often under 20 mm. per hour. Where the tuberculin reaction was previously negative and now becomes positive, one is dealing with a primary infection ; if previously positive, suspect a reinfection. Radiographic appearances, if present, are usually situated below the clavicle or at the apex of a lower lobe ; a small circular infiltration about the size of a cherry stone is seen ; the edges are fluffy and the shadow diffuse and not hard ; there is no corresponding glandular enlargement (*Fig.* 19). Occasionally more than one focus is seen. Diagnosis must rest on radiographic appearances and positive tuberculin test ; a negative test (two strengths) is strong contra-evidence ; physical signs and tubercle in the sputum may be absent at this early stage. Suspect a feverish attack with malaise, resembling influenza, or the recent history of such an attack.

Differential Diagnosis.—

Apical pneumonia will yield more definite local signs and resolves rapidly. Tuberculin test may be negative. Suspect a pneumonia which fails to respond to sulpha drugs.

Bronchopneumonia. Immediate differential diagnosis difficult. Repeated negative tests, marked physical signs, and acute illness suggest bronchopneumonia.

Lung abscess shows more marked toxic symptoms. The shadow of an abscess can be confused with caseation and cavitation, but hardly with an early infiltration.

Influenza. The rise of temperature without signs of disease often causes an early infiltration to be mistaken for influenza or chill. Rely on tuberculin test, and get a radiograph if test is positive.

The Fate of the Infra-clavicular Focus.—(1) Healing ; (2) Cavitation.

That healing by fibrosis and calcification occurs has been shown in numbers of cases. It may take some years for firm healing to be established, but clinically the patient is well in a much shorter time. Central liquefaction represents the danger point, and this condition can be recognized by X rays ; although physical signs may still be absent at this time yet as a rule signs suggesting localized pneumonia will be found ; however, many infraclavicular cavities in children are not apparent by physical examination. The cavity formed by central liquefaction may heal spontaneously by emptying out its contents ; more usually,

however, some of the contents lodge in the wall of a bronchus and are aspirated thence to lodge afresh in healthy lung tissue. These 'daughter' foci, which may be situated in the opposite lung, have a still greater tendency to cavitate, and from them also there follows further bronchogenic spread, until finally is produced the picture of phthisis, spreading from the upper lobe downwards. The formation of each new focus is characterized by a rise of temperature and malaise; after the implantation of the first hæmic focus, the disease does not progress in an even manner, but with exacerbations as each new focus is formed or as an old focus cavitates. As Twining, from the radiological point of view, says: "the disease is not a steady insidious spread from the apex down, but rather it appears to extend by a series of catastrophes".

BRONCHOGENIC PHTHISIS

This may be classified as: (1) Ulcerative—infiltrative, nodular, and mixed; (2) Fibroid.

Ulcerative Phthisis.—

Infiltrative Type.—In children, unfortunately, this is the type most often seen; the total number of cases, however, is small. The lesions are pathologically exudative and clinically infiltrative, with marked absence of fibrous tissue; the course of the disease is rapid. Type III infection usually originates in an Assmann focus in the infraclavicular region; an inflammatory secretion containing albuminous fluid and blood-cells is poured into the alveoli; this fluid caseates, liquefies, and spreads from the air-cells to the lobules, and cavities are formed. Simon and Redeker, amongst 174 children with open phthisis originating in the infraclavicular region, found that 75 per cent were of the infiltrative type, and that the mortality-rate in this type was 92·4 per cent.

Clinical: The disease runs a rapid course, usually with a fatal termination within a few months; cases which live only a few weeks are known as acute lobular- or broncho-pneumonia. There is high fever, and the physical signs are those of dullness and rattling rales over wide areas. Tubercle bacilli, with elastic tissue, are plentiful in the sputum. Phthisical hæmoptysis is rare in children; Price has seen it twice in phthisical children, both aged 2 years, and once from a primary cavity; usually hæmorrhage is associated with bronchiectasis.

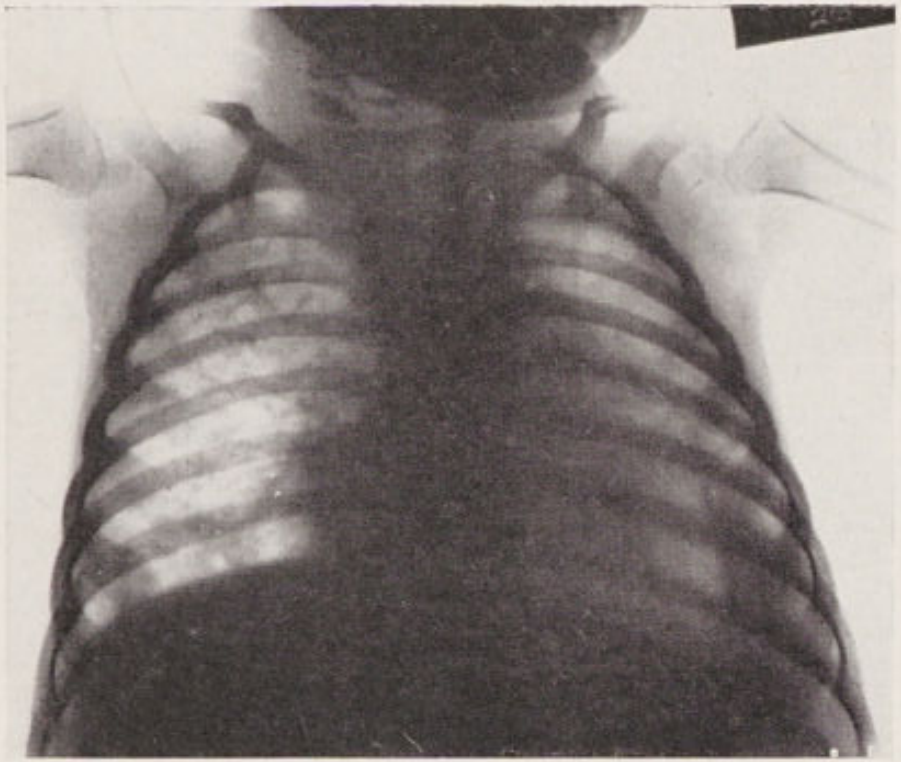
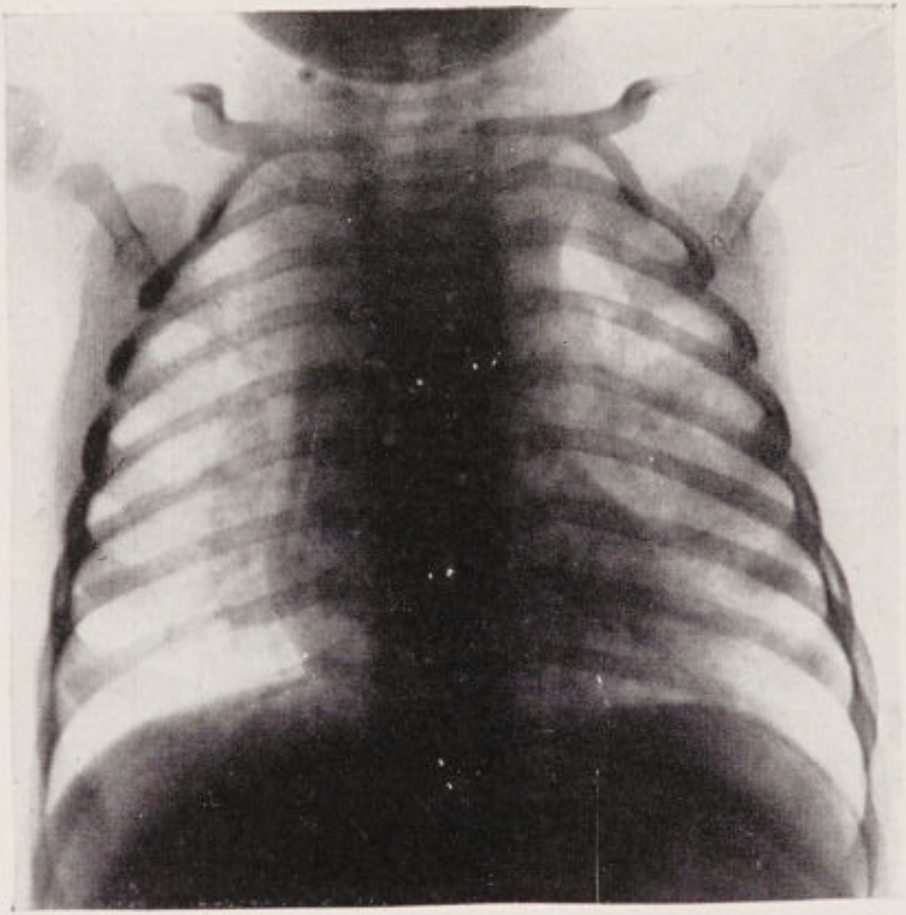


FIG. 45.—CEREBRAL TUBERCLES.

William S. Aged 5 months. (*See Example*, p. 102.) Infiltration in right upper zone; enlarged mediastinal glands; fluid at left base. Confirmed 4 months later at autopsy



FIGS. 46A, 46B.—ACUTE LOBAR-PNEUMONIC PHTHISIS (ASPIRATION).

Fig. 46A.—Frank D. May 18, 1939: aged 7 months. (*See Example*, p. 105.) Infiltration of left lung; 'ribbon shadow' with a fainter shadow of periadenitis lying parallel to mediastinum on right side.

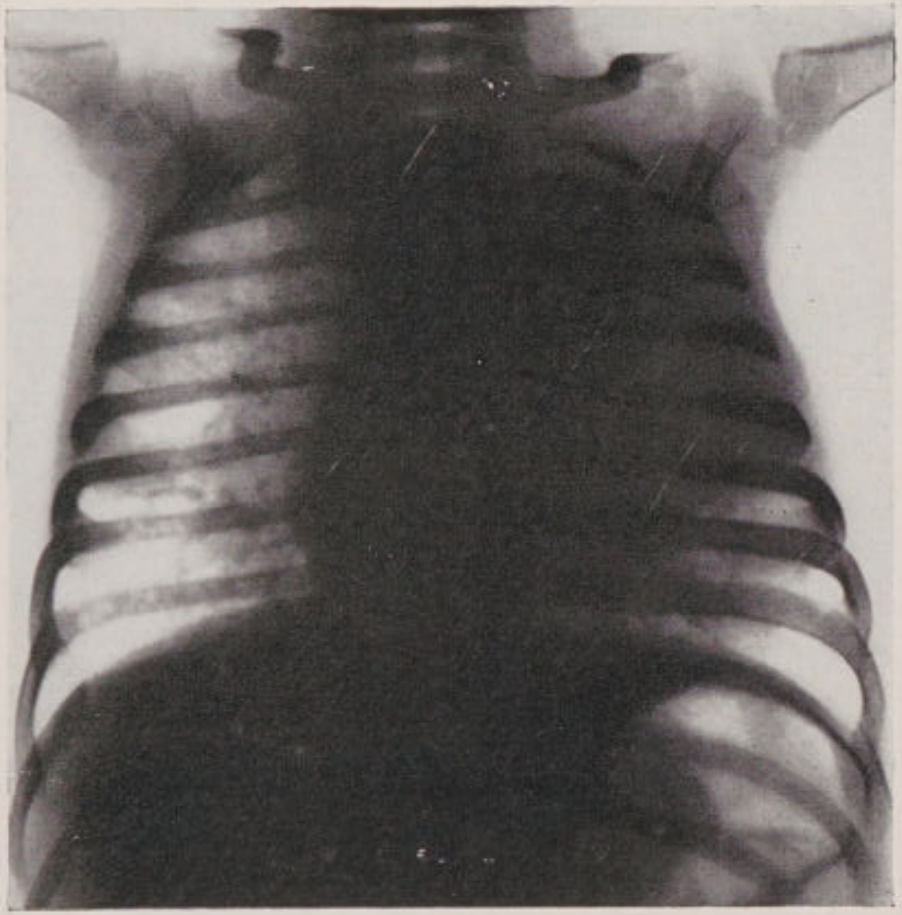
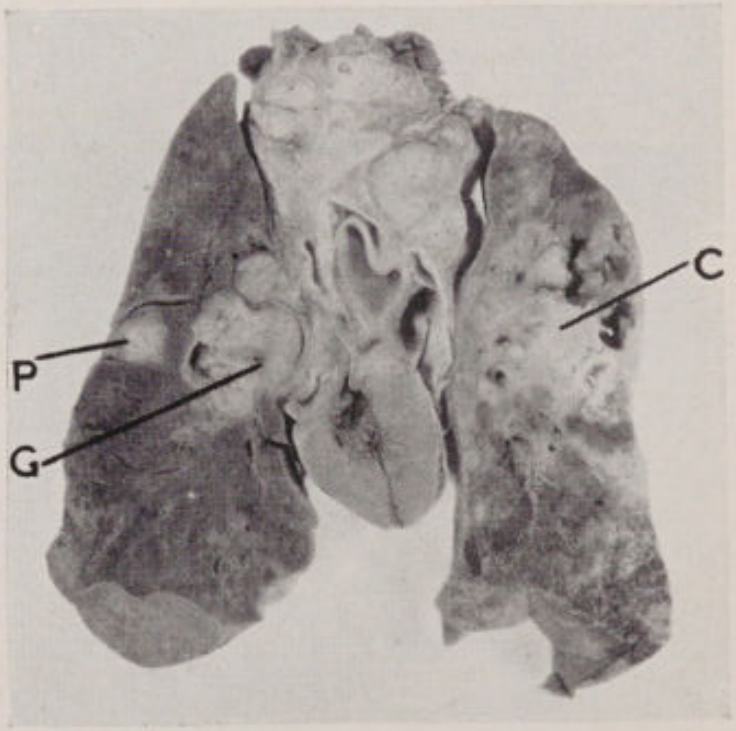


Fig. 46B.—June 22, 1939. Caseous pneumonia in left upper lobe with cavitation; periadentitis persists. The small round area of infiltration at level of 4th rib on right is the primary focus.

Note lack of radiological evidence of the primary focus compared with the picture of the anatomical specimen.



FIGS. 47A, 47B.—ANATOMICAL SPECIMEN FROM CASE SHOWN IN FIGS. 46A, 46B.

Fig. 47A.—July 2, 1939. Shows typical primary focus (P); liquefying bifurcation gland erupting into bronchus (G); large caseous infiltration with cavity formation (C), due to aspiration into left upper bronchus.

(*By courtesy of Dr. Walter Pagel.*)

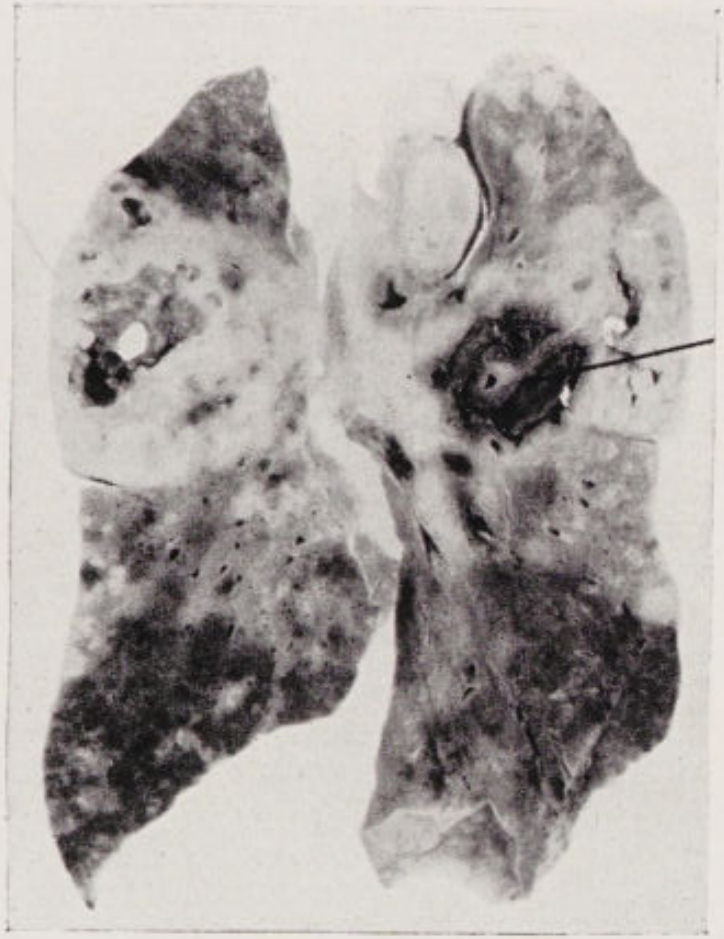


Fig. 47B.—Close-up view of large liquefying infiltration in left upper lobe, with blood-clot (arrow) the source of fatal hæmorrhage.

(*By courtesy of Dr. Walter Pagel.*)

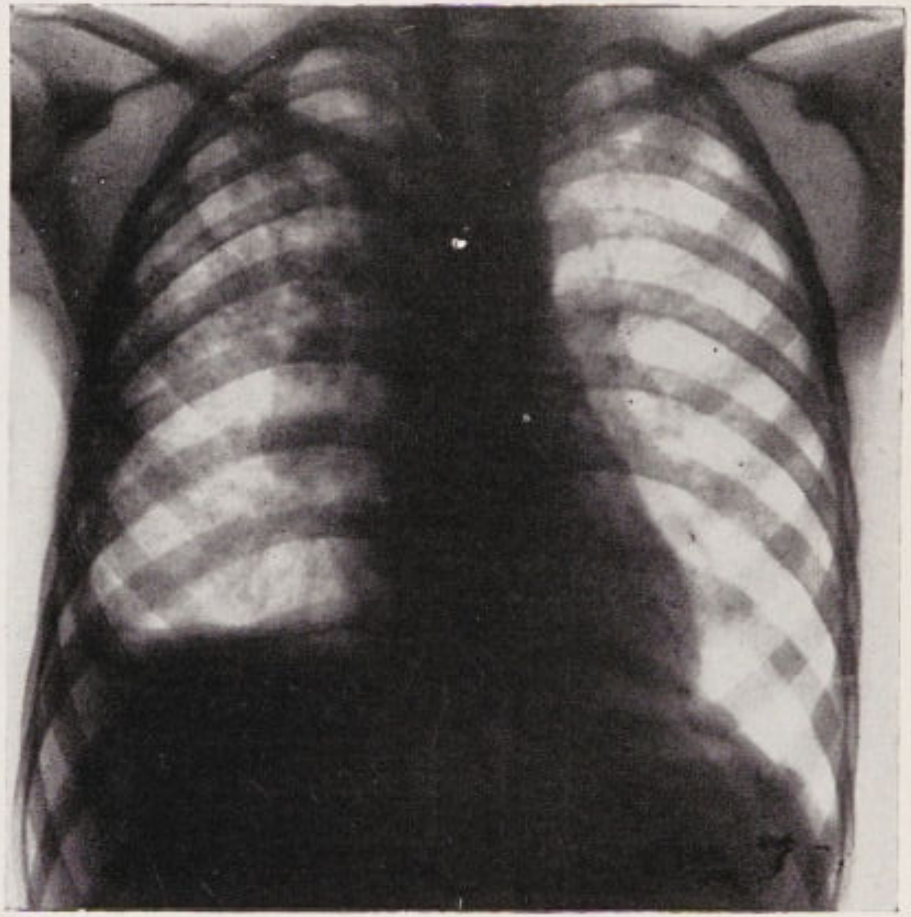
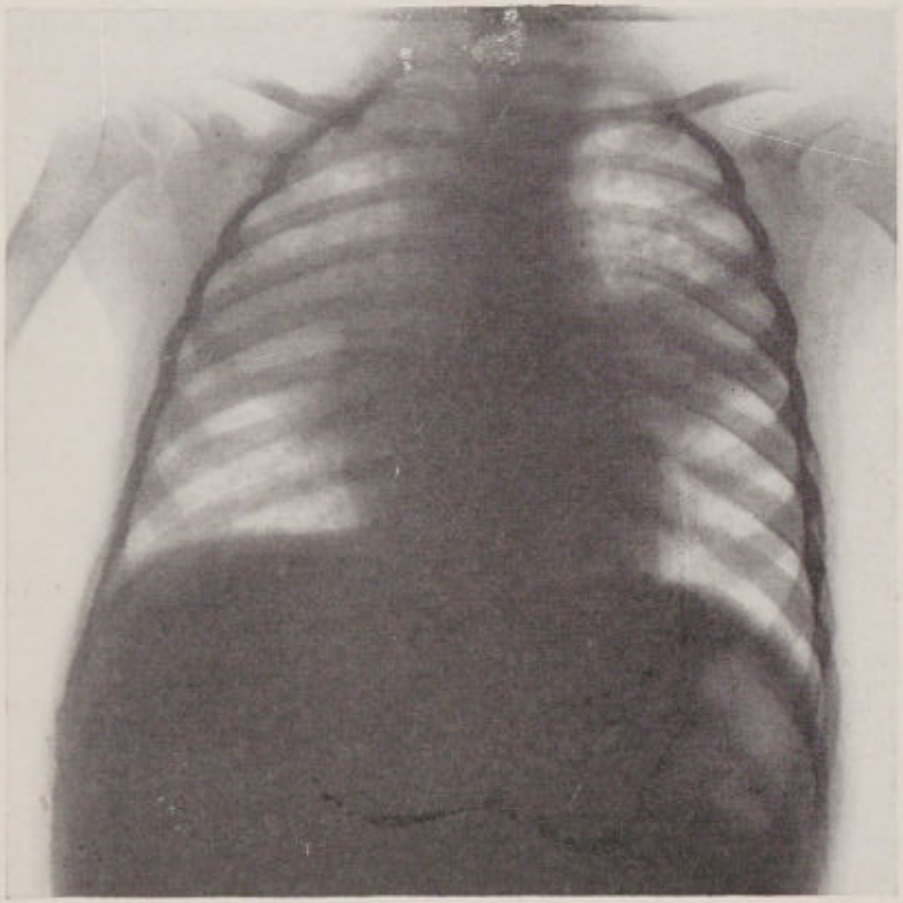
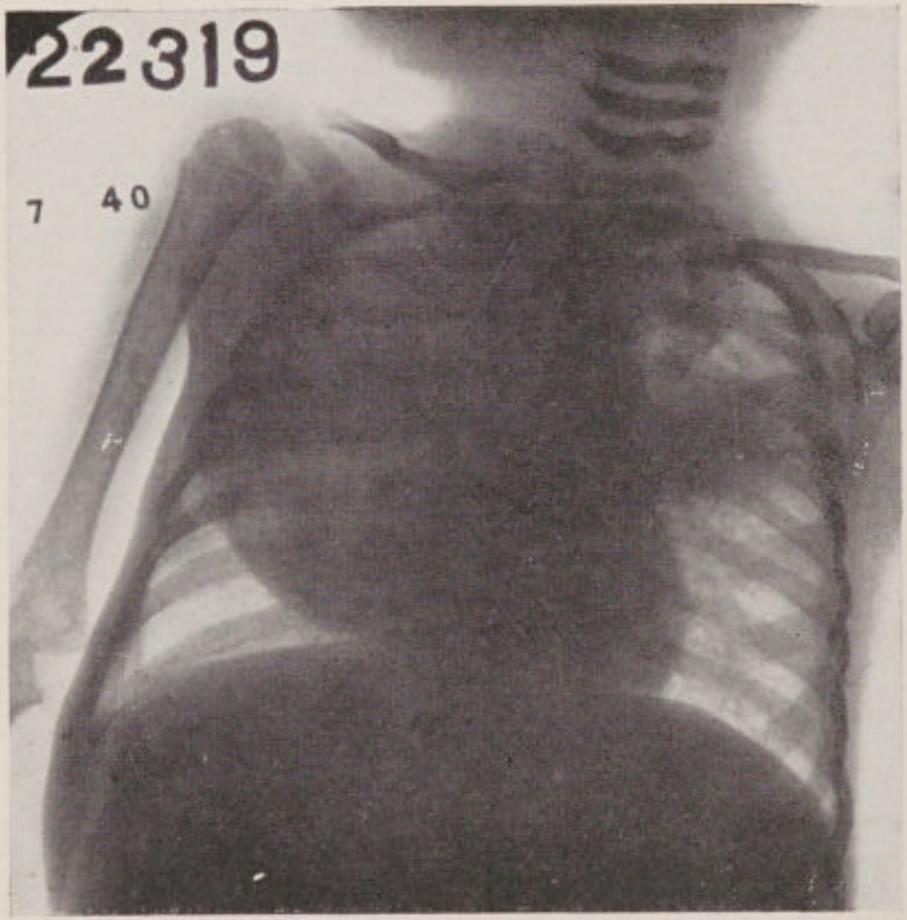


FIG. 48.—ULCERATIVE PHTHISIS.

Christopher C. Aged 10 years. (*See Example*, p. 113.) Bilatera phthisis, with enlarged gland in right hilum and fluid at the right base.





FIGS. 49A, 49B.—PHTHISIS IN A CHILD OF 2 YEARS.

Fig. 49A.—Mary S. May 4, 1940; aged 2 years. (*See Example*, p. 113.) Bilateral phthisis with cavities.

Fig. 49B.—July 9, 1940. The opacity on the right side with convex lower border was found at autopsy to be due to tuberculous pneumonia.

The larynx and abdomen are seldom involved. The radiological appearances are those of thick round confluent shadows, with central caseation. In more advanced cases large cavities are seen (*Figs.* 21, 48, 49). Radiological diagnosis between the infiltrative and the nodular types of phthisis in the child is not reliable ; the presence of large glands or fluid, with rapid extension of lesions in serial radiographs, are indications of a bad prognosis, and a more reliable guide in diagnosis than descriptions of radiographical appearances of the actual lesions.

Examples.—

Christopher C., aged 10 years. Child very ill. Sputum positive. Duration of disease one year. Sanatorium treatment begun too late. Death 3 months after the radiograph (*Fig.* 48). Bilateral phthisis, with fluid at right base and enlarged gland in right hilum.

Mary S., aged 2 years. March 28, 1940, measles rash. April 1, 1940, admitted to hospital with cough and laryngitis, which responded to treatment in a few days. Odd crepitations were audible in both lungs. Temperature varied between 98° and 100° F. The child developed otitis media of left ear and an enlarged right deep cervical gland which eventually broke down. For two days before death on Sept. 9, 1940, there was head retraction. At no time could lung condition have been diagnosed by the stethoscope. X rays May 4, 1940 (*Fig.* 49A), showed bilateral phthisis. June 17, 1940, a cavity in both upper zones was seen. July 9, 1940 (*Fig.* 49B), extension of disease on right had occurred. Sept. 9, 1940, death. Autopsy revealed a large cavity in the right upper lobe posteriorly, enlarged regional gland, with spread of the disease to right middle and left upper lobes. The opacity seen in the middle lobe was due to a tuberculous pneumonia, in which a number of tubercles were present. A few caseous mesenteric glands were found. Apparently the primary focus in the right upper lobe had necrosed, forming a primary cavity, whence occurred spread to other parts of the lungs.

Nodular Type.—In this type of phthisis there is a greater tendency towards healing than occurs in the infiltrative type. From a cavity in a post-primary focus or in a ' daughter ' focus, tubercle bacilli are aspirated, from above downwards, until finally one or both lungs are extensively involved. The aspirated bacilli lodge in the air-cells, and form nodes composed of caseous centres with epithelioid and giant cells, and with more or less fibrous tissue in the periphery ; pathologically this nodular type of phthisis is known as the ' productive ' type. The course is relatively more chronic than in the infiltrative type. The duration of the disease may be 1 to 3 years in the fatal cases. Boys are said to live longer than girls. The mortality-rate in Simon and Redeker's series was 76·6 per cent.

Clinical: The disease lasts from months to years. There is no marked constitutional disturbance, and very little fever. Physical signs in the lungs depend on the stage and extension of the disease. The sputum contains very little elastic tissue and a moderate number of tubercle bacilli. Hæmoptysis is very rare. In the later stages, the larynx and abdomen may become involved. Agassiz found secondary laryngeal involvement in 27 per cent of 48 children over 10 years suffering from active pulmonary tuberculosis, and in 12·5 per cent of another series of 72 cases. The radiological appearances are not characteristic, but tend to show thick irregular trefoil form rather than confluent shadows. When the whole lung tissue has been destroyed, death supervenes.

Example.—Edward G., aged 10 years. Admitted to a fever hospital with signs and symptoms of left apical pneumonia ; as resolution did not occur, a tuberculin test was performed and found to be positive. He was moved to the City of Dublin Hospital and sputum was found to be positive. Radiograph (*Fig.* 20A) showed bilateral upper lobe phthisis, with a large cavity on the left. Artificial pneumothorax was attempted but failed owing to adhesions (*Fig.* 20B) ; thoracoscopy revealed multiple adhesions which it was impossible to cut with the cautery, so the lung was allowed to re-expand. Sent to a children's sanatorium, where he remained from January, 1939, to March, 1941 ; he was afebrile, without sputum, and radiograph showed some healing by fibrosis, but the cavity still remained open. He died at home two years later.

Fibroid Phthisis.—Fibroid is a form of phthisis which shows a predominance of fibroid tissue ; it represents the more chronic type of phthisis with strong healing tendency. It is rather rarely seen in children. Such cases represent about 5 per cent of all cases of phthisis in children (phthisis being in itself rare), and it is found rather in middle or late school life than in infancy or during puberty. The duration, where healing is not complete, is from 1 to 10 years. The prognosis is fairly good, the mortality-rate, according to Simon and Redeker, being about 40 per cent.

Clinical.—There is little or no fever and no marked constitutional disturbance. Over the affected area of lung physical signs are mainly those of chronic bronchitis with compensatory emphysema of the lower lobe. There are few tubercle bacilli in the sputum and no elastic tissue. The X-ray appearances are those of confluent foci, without much caseation, and of increased bronchial markings.

CHAPTER X

TUBERCULOSIS IN THE ADOLESCENT

General Remarks.—No account of tuberculosis in the child can be regarded as complete unless reference is made also to the condition in the years of adolescence. The tuberculous processes which occur during the years between childhood and adult life must be studied ; it is but recently that attention has been focused on this age period. Often the first entry of the tubercle bacillus into the human body takes place during this period ; recognition of the adolescent primary infection is most important, because cure at this stage is more sure and more rapid than if one waits until the visible seeds of phthisis are sown. In adolescence, as in childhood, we have to deal with primary tuberculosis of the lungs (or abdomen), with secondary hæmic manifestations, and also with phthisis. The primary has been called the ' childhood ' type of infection, but this is a misnomer, for universal statistics show that nowadays at 14 years some 50 per cent or more of persons have still to face their primary infection. Thirty years ago, it is true, this state of affairs did not exist, for then 80 per cent to 90 per cent had already been infected at 12 years of age. Thus a new problem has arisen, and investigations show that a fresh attitude of mind must be adopted towards the adolescent. That adolescence is a period when many negative tuberculin reactors become positive has been ascertained ; that it is also a period when phthisis makes its appearance, rising to its highest death incidence in the 20 to 25 age period, is well recognized.

The connexion between primary adolescent tuberculosis and the development of phthisis in young adult life has not as yet been fully investigated, although the fact that such a link exists has a significance which is of epidemiological as well as of individual importance. It is well known that phthisis frequently develops during young adult life, especially in females ; that its morbidity and mortality rates are dependent on the ratio of adolescent tuberculin reactions has been suggested by Price (1939) ; i.e., that a low number of positive reactors at 14–18 years

tends to produce a high death-rate in the 15–25 years age period, because Type II or III may develop shortly after primary tuberculosis. Primary tuberculosis in the adolescent is not, *per se*, a dangerous disease ; if recognized and treated adequately from its inception it runs a course as benign as in the school child, and more benign than in the infant. Danger, however, in the non-treated case lies in the risk of hæmic spread, resulting not only in generalization or meningitis but also in the development of phthisis. The reasons for the special danger of rapid spread with consequent phthisis may be summed up in the term ' adolescent factors '. These factors are :—

1. An endocrine factor, especially marked in girls.
2. Body growth, with food requirements not always satisfied.
3. Great energy, with little desire or opportunity for rest.
4. Overwork at studies, anxiety, and stress of beginning to earn.
5. Increased opportunity for extra-familial contact.

Acquired immunity towards the tubercle bacillus is distinct from individual inherited resistance ; both are important assets should contact with ' open ' pulmonary tuberculosis occur during young adult life (*see Table XVII*). The influence of several generations of acquired immunity on individual inherited resistance is at present unknown. Acquired immunity can, according to our present knowledge, be achieved in two ways : either (1) naturally by a primary complex satisfactorily healed during childhood or adolescence ; or (2) artificially by preventive inoculation.

It may be argued that a high young-adult mortality-rate indicates a special susceptibility towards the disease in any given country or locality. That such special susceptibility should be due to a *racial* factor does not seem to be altogether convincing when behaviour towards the tubercle bacillus of Irish people abroad is considered. So far as information is available at present, it would appear that children of Irish race born in England and Scotland do not differ from those of the land of their adoption in their tuberculosis life. They do not show any markedly low tuberculin-positive percentages ; furthermore, Irish children born outside Ireland follow the radiological picture of their neighbours rather than of their brethren in Ireland, should pulmonary tuberculosis develop. These considerations tend to support the assumption that the main factor which has to be considered in the Irish problem is

one of *geographical* isolation. The difficulties of this particular problem can be dealt with most satisfactorily by early diagnosis and treatment of the adolescent primary infection, or still better with protection by vaccination.

It is clear by observations amongst young nurses that it is the negative reactor who is in danger of developing Types II and III tuberculosis when exposed to massive infection, unless the primary stage is recognized and treated; a young adult who possesses a positive skin test and a satisfactorily healed primary complex is less prone to develop pulmonary tuberculosis than is the negative reactor. Malmros and Hedvall (1938), amongst 3336 students in Lund, found that 30 per cent were negative to tuberculin when commencing their studies. Ferguson (1946) reports that about 80 per cent of nurses entering hospitals in Saskatchewan are negative to tuberculin. The fate of the negative reactors is given in *Table XVII.*

Table XVII.—Fate of Young Nurses in Sanatoria, according to Their Tuberculin Reaction on Entry

	Healed Positives	*Negatives*
Heimbeck (1936) 905 nurses	4·3 per cent got tuberculosis	34·2 per cent got tuberculosis
	0·0 per cent died of tuberculosis	3·5 per cent died of tuberculosis
Mariette (1936) 925 nurses	4·6 per cent got tuberculosis	36·9 per cent got tuberculosis
	0·0 per cent died of tuberculosis	3·8 per cent died of tuberculosis

Many general hospitals and sanatoria have now adopted the precautionary measures of tuberculin testing and X-raying all probationers at entry. Thus no nurse with active or early tuberculosis is admitted, and negative reactors who subsequently develop erythema nodosum or febrile (so-called influenzal) attacks are re-tested and X-rayed immediately, and if there is altered allergy, with or without a visible primary lesion, they are immediately treated by adequate rest for some months until healing is ensured; re-testing every subsequent year is performed as a routine on all negative entrants, even in the absence of symptoms. A number of sanatoria (Macklin) refuse tuberculin-negative nurses. In Scandinavia all negative probationers are rendered positive by B.C.G. inoculation before admission, with resultant marked reduction in the incidence of tuberculosis amongst their nurses.

Surveys in these islands showing the tuberculinization of the young adult population are scanty. In England, Daniels (1944), amongst 121 entrant nurses aged 17 years, found that 72·7 per cent were positive reactors; and amongst 951 aged 19 years that 80·3 per cent were positive; furthermore, he pointed out that the percentage of entrant negative nurses who came from Irish and Welsh rural districts was twice as high as amongst English and Scottish nurses. In Ireland, Crowe (1942) found 44 per cent positive reactors amongst persons aged 15 to 20 who were referred to a rural tuberculosis dispensary (*see* p. 7). In Dublin general hospitals several small surveys show that amongst entrant nurses aged 18 to 20 not less than 30 and not more than 60 per cent are positive reactors. Amongst 302 pre-medical students in Dublin University 52 per cent were found positive to Mantoux 1–1000. Price (1945) found amongst 172 healthy girls aged 18 years, leaving a State boarding school, that 30·8 per cent were positive to Mantoux 1–100. Together with this rather low tuberculinization figure, in Ireland the tuberculosis death-rate in the 20–25 age period was 2·26 per 1000 in 1944. The type of tuberculosis found in these young adults in Ireland has certain characteristics: short, if any, history of ill-health, absence of signs and symptoms, rapid extension of lesions suggestive of hæmic spread, or of infiltrative rather than fibrotic phthisis. Findings comparable with experience in Ireland are: Maoris contacting the white population of New Zealand, African troops in the war of 1914–18, and girls going to the mainland from Bornholm. Since early diagnosis of primary tuberculosis has become possible by means of tuberculin tests, large-size films and mass radiography, many cases are healed before Type II or Type III tuberculosis develops.

Primary Tuberculosis.—Negative reactors in the adolescent period are found mainly in non-tuberculous households; thus their chance of meeting infection when they go into crowded factories, schools, or offices is increased; recognition of the primary infection is hampered because they are not known to the Tuberculosis Officer as home contacts, and unless really ill they come under no special medical care. During the activities of youth, circumstances allow little for the *rest* which is essential for the healing of the primary focus. The endocrine factor must exert an influence which is deleterious both on primary and tertiary tuberculosis, but this has not yet been proven in any exact manner.

Diagnosis.—

1. Tuberculin test becoming positive from negative.
2. Initial fever.
3. Vague complaints, fatigue, headache, abdominal discomfort.
4. Radiograph of the chest.
5. Erythema nodosum.
6. Pleurisy.
7. Conjunctival phlycten.

Of these aids to diagnosis only (1) is infallible, and it may be accompanied by (2); in light infections, however, fever may be so slight as to escape notice. Complaints of vague symptoms of ill health may or may not be present. If a chest radiograph is taken during the initial fever, some focal infiltration may be visualized in a proportion of cases, but at that and later stages more often hilar gland enlargement only is seen, totalling about one-third of cases. Radiographic appearances are less marked than with primary infection in the child, and failure to find radiological appearances of the complex must not influence the diagnosis in a case where a negative skin test has become positive, although it may modify the treatment given. There is complete absence of physical signs in the chest and of cough. Fatigue is rather universal, with disinclination for work. In a certain proportion of cases erythema nodosum coincides with the initial fever, and is a very great help towards diagnosis; its incidence is higher during adolescence than during school age, and is very much greater amongst females than males. Price found erythema nodosum coinciding with the onset of primary tuberculosis in 13 per cent of girls infected between 14 and 18 years. Phlyctens are rather uncommon at this age, and appear generally some months after the occurrence of the primary infection. Pleural effusion also appears late, from 3 to 15 months after primary infection. Nevertheless these two late manifestations, phlyctens and pleurisy, are often the first indication of an unrecognized tuberculous primary infection, although by that time radiological evidence of the primary complex may have disappeared, and thus doubt may be cast on the diagnosis. Full examination of the abdominal and cervical gland areas must be undertaken as a routine in searching for a primary focus; this is especially important when the chest radiograph is negative. The detection of primarily infected abdominal glands at an early stage is difficult, and diagnosis often uncertain.

Differential Diagnosis.—

Influenza: Initial fever may be mistaken for influenza or chill. In the latter cases pains in the limbs are present with malaise and the temperature seldom remains febrile with suitable drug therapy. Initial fever is symptomless, and usually lasts for 7 to 10 days. The tuberculin test is invaluable if previous reaction is known to have been negative.

Typhoid Fever: Prolonged fever accompanied by complaints of abdominal discomfort suggest typhoid fever (also abdominal tuberculosis). Specific tests for typhoid must be made. A negative tuberculin test, up to 1–100 Mantoux, is a contra-indication for primary tuberculosis.

Prevention.—*See* p. 139.

Treatment.—Rest is the one and only important element in treatment: one month in bed, even if temperature is normal before the month is up; then half-time rest for a further period of 3 to 6 months, depending on the severity of the case; careful supervision for some years (*see also* p. 127 et seq.). Compare results in the three examples quoted below with *Fig.* 39.

Example of Erythema Nodosum in an Adolescent.—Irene G., 15 years. Developed erythema nodosum on June 11, 1937, temperature 104° F., falling to normal in 10 days' time. Hamburger test negative Oct. 10, 1936, positive June 14, 1937. X rays June 18, 1937, showed enlarged gland in left hilum. She was kept in bed 1 month, rest at home in country for 3 months, then easy work at school for 3 more months. No games for 18 months. Subsequent radiographs showed encapsulation of gland and finally calcification.

Examples of Hilar Gland Enlargement and Initial Fever.—Maeve H., 18 years. On Feb. 6, 1938, complained of headache and fatigue during an influenza epidemic. Temperature 99° F. p.m. for a week. Hamburger negative Sept. 9, 1937, positive Feb. 8, 1938. On Feb. 14, 1938, chest radiograph (*Fig.* 50) showed enlargement of glands in right hilum, primary focus not visualized. She was kept in bed for 2 more weeks, and remained at school working half time and resting until the Easter holidays. When she went home she was greeted by the news that her mother had been taken to a sanatorium and had just died. (The girl had been in contact with her during the Christmas holidays.) Four months after her primary infection the girl developed a right pleural effusion. She remained at home for a year with good improvement, then finished her school course, and has kept well since. Chest radiograph now negative. Her brother, aged 12 years, not treated in 1938, was found in April, 1940, to be suffering from tuberculosis of the knee-joint.

Vera C., 18 years. On Dec. 7, 1937, complained of sore throat, temperature normal. On Dec. 10, 1937, her temperature rose to 102° F. and remained so for 10 days; she complained persistently

of abdominal pain. Hamburger negative Sept. 9, 1937, positive Dec. 12, 1937. No physical signs in chest at any time. Differential diagnosis between typhoid fever and abdominal tuberculosis. X rays (*Fig.* 51) on Dec. 22, 1937, showed a small Ghon focus in the outer left lung field and an enlarged regional hilar gland. Whilst in hospital she contracted diphtheria, and was thus kept in bed for nearly three months ; this rest suited her very well. After a rest period at home she returned to school four and a half months after her initial fever ; three months later she passed her final examination with honours, and then took a further period of half-time rest for 3 months at home. Serial radiographs show commencing calcification of the primary complex. She kept remarkably well until Jan. 16, 1939, when she had a left pleural effusion after a bad cold. She took a year off work and then completed her studies ; X-ray film still clear in 1946 ; she is now teaching (1947).

Type II Tuberculosis.—Tuberculous meningitis and acute miliary and bronchopneumonic phthisis occur in a proportion of infected adolescents ; one wishes all these cases could be avoided, as they constitute tragedies with their abrupt onset and rapid course. The history of illness is short and in nearly all instances the condition is directly consequent on hæmic spread from a fresh and unrecognized primary complex. Spread from an apparently or partially healed infection, acquired during childhood and reactivated under adverse conditions during puberty, is also a possible theoretical presumption ; usually a satisfactorily healed childhood focus protects from reinfection. In practice it is found, however, that in the great majority of cases the spread is rapid and follows closely after a first infection acquired during adolescence. Chest radiograph in tuberculous meningitis will reveal, in a percentage of cases, some involvement of the hilar glands. Often it is only at autopsy that the primary complex is found.

Example of Tuberculous Meningitis.—Edward C., 14 years. No history of any illness prior to a few days before admission to hospital suffering from tuberculous meningitis. Previous tuberculin reaction unknown. Hamburger positive on admission. X rays (*Fig.* 52) show an enlarged gland in the left hilum. The boy was very strong and healthy and could have passed through his primary infection a short while before without complaint ; no family history of tuberculosis.

Example of Acute Miliary Tuberculosis.—Sheila F., 18 years. A fat healthy girl, never ill. Moro and Mantoux 1–100 tests were negative in October, 1940, 1941, 1942, 1943. Between October, 1943, and March, 1944, her only complaint was that she fainted on November 25, 1943, but had no rise of temperature and did not go to bed. On March 17, 1944, she developed a temperature of 102° F., and remained in bed ; no symptoms and physical examination repeatedly negative.

On March 20, Moro test was positive, and a diagnosis of *initial fever of primary tuberculosis* was made ; 10 g. of sulphathiazole was given, 1·5 g. per day. On the eighth day the temperature fell to normal, remained normal for twenty-four hours, then rose again and took on a pyrexial swinging character until the end. On March 27, on admission to hospital, her chest radiograph was negative. On April 15, a second film showed nothing abnormal save a questionable enlargement of the right paratracheal gland. Weakness and drowsiness increased rapidly ; on May 4, the third chest film showed " diffuse mottling in both lungs, especially the right ; commencing miliary tuberculosis ". She died on May 27, 1944, exactly two months after the onset. Meanwhile it had been found that her brother, whom she saw once a week, was going about with an unsuspected early cavity.

In this case the primary infection was recognized and treated at its inception. (It is, however, remotely conceivable that on Nov. 25, 1943, primary infection took place, for no test was performed then on account of a fainting attack, over in a moment.) Hæmic spread probably occurred during the first week of the infection (March 17), with twenty-four hours remission between initial fever and the development of the clinical signs of Type II infection. This girl must have had a total lack of resistance to the tubercle bacillus. The only possible means of avoiding such catastrophes lies in immunization of the negative reactor with B.C.G. vaccination.

Type III Phthisis.—A very considerable proportion of the casualties from phthisis in the 15–25 age period are undoubtedly due to neglect of a late-acquired infection where healing of the primary complex has not been ensured. A very few cases may be due to extension from a hæmic focus acquired earlier in life, which has the radiological appearance of calcification (*Fig.* 21). In this age period one sees all degrees of phthisis from the exudative to the fibrotic type ; the former predominates in adolescence, and has given rise to the term ' puberty phthisis ', which has been employed by some authorities to describe this fulminating condition.

In practice one finds that a hæmic post-primary focus is the most frequent mode of reinfection during adolescence. Reasons for the tendency of primary adolescent infections to progress to further stages of tuberculous disease are as follows :—

a. Adolescent factors already mentioned.

b. The relatively small number of primary infections diagnosed as such in the adolescent ; lack of rest therapy impedes healing and the disease may progress to the second or third type without interval, whilst the patient is still hypersensitive on account of the primary activity and because relative immunity has had as yet no opportunity to develop.

Early Diagnosis of the Infraclavicular Focus.—After an interval of time has elapsed after the primary infection (indeed it generally happens within the first year), the development of activity in a fresh focus is ushered in by a febrile attack, which is accompanied by feelings of malaise. Diagnosis must be sought by physical examination, which is often negative in the very early stages, and by radiological examination, which is as a rule more helpful (*Fig.* 19). At times an infiltration will yield physical signs in the absence of radiological appearances, but this is exceptional. Sputum, if present, may show some tubercle bacilli. The short febrile period may be entirely overlooked, for symptoms other than fatigue are rarely complained of. The S.R. is not unduly raised ; it may be about 20 mm. per hour.

Example.—Maureen C., aged 15 years. Some systemic disturbance. No special treatment was given for pulmonary condition (*Fig.* 53). Differential diagnosis by radiograph lay between 'epituberculosis' and unilateral early phthisis. The girl returned 16 months later with advanced bilateral phthisis.

Differential Diagnosis.—From apical pneumonia or influenza.

Treatment.—Absolute bed-rest with radiological control until healing is established. Early collapse is indicated, for too long delay (i.e., until liquefaction and cavitation has occurred) may render collapse impossible, owing to pleurisy and subsequent adhesions. (*See* pp. 130, 134.)

Diagnosis of Phthisis.—Liquefaction and cavitation of the Assmann focus, with or without pleural effusion, may be mistaken for apical pneumonia. Each further spread with formation of fresh foci is accompanied by fresh febrile attacks. Radiography is the only sure means of speedy diagnosis, as all cases will not yield physical signs, or these signs may be in no way typical. Sputum may show but few tubercle bacilli in the very early stages.

Prevention.—Prevention is effected by early diagnosis or by B.C.G. vaccination.

Example of Prevention.—Price (1945) observed 172 girls for four years in a State school, from entry, aged 14, to leaving, aged 18 years. At annual examination the negative reactors were retested and also during the year if ill or during an influenza epidemic ; all positives had chest films taken. During this 4-year period, 31 became positive from negative ; of these, 16 (51·6 per cent) showed no illness and had negative radiographs ; 7 showed ill health ; and 8 were definitely ill ; of the last, 1 died of miliary tuberculosis (*see Example*, p. 121), and the 7 others showed evidence of primary tuberculosis in their chest films. With appropriate treatment 30 conversions got well, with no extension of disease ; they were followed for 3 to 13 years and the results are given in *Table XVIII.*

Table XVIII.—262 Healthy Adolescents reviewed after 13 Years (Price, 1947)

	T.B. Mortality	T.B. Morbidity
	per cent	per cent
1927–33		
90 adolescents (14–18) untested		
Reviewed in 1946	1·1 (phthisis)	3·3 (phthisis)
1933–44		
53 positive leavers (18 years)	0·0	0·0
119 negative leavers (18 years)	0·0	4·3 (early phthisis)
1933–44		
31 conversions from positive to negative (14–18)	3·2 (miliary)	19·0 (primary)
30 reviewed in 1946	0·0	0·0

CHAPTER XI

TREATMENT, PROGNOSIS, AND PREVENTION OF PULMONARY LESIONS

TREATMENT

With all forms of pulmonary tuberculosis in children, treatment may be divided into *general* and *specific*. In this chapter it is proposed to deal with the treatment of pulmonary tuberculous lesions in all stages; the treatment of individual extra-pulmonary lesions is discussed in those sections which describe the conditions. All pulmonary lesions in children are treated in general in the same manner, but with degrees of strictness which vary according to the type of infection. The main line of treatment is bed-rest. The merits of institutional as against home treatment will first be discussed.

Institutional *v.* **Home Treatment.**—*Primary lung tuberculosis* requires treatment in an open-air preventorium. For reasons of necessity or choice, certain cases may have to be treated in their own homes; this is permissible only where it is assured that rest rules will be observed. It is, however, always advisable to remove young children to a tuberculosis hospital or preventorium for the first three months after the development of primary tuberculosis of lungs, abdomen, or cervical glands; here they learn to remain happily in their cots and this period allows of observation as to the course which the disease is to take. The institutionalization of radiologically visible primary lesions in older children, adolescents, and young adults for a few months is also most advantageous; it is extremely difficult to ensure bed-rest in the older age periods, when parents have less control; puberty phthisis may ensue in certain cases imperfectly rested in their own homes. Therefore experience shows that although primary tuberculosis may be healed by home rest, this healing, with avoidance of subsequent development of phthisis, is more certain if the patient rests under authority in an institution. In mild cases treatment may be continued at home after three

months, with weekly or fortnightly attendances at the tuberculosis clinic, where progress can be checked by continued observation of weight gain, S.R., temperature, and radiography. Healing at home may be slower than in the preventorium, especially if the home conditions are not good; but if the rest hours are strictly enforced by the physician, and the mother and child good co-operators, many cases of light primary infection will heal satisfactorily at home.

In the present state of our information, it is advisable to separate children suffering from primary tuberculosis from convalescent children who are negative reactors, although risk of infection from primary tuberculosis is very slight. It has been suggested that the preventorium should have a school attached; the writer's opinion is that whilst children are in hospital for primary tuberculosis they should be mainly confined to bed; on discharge they will have plenty of time for schooling, but whilst undergoing treatment they should not be made to get up and perform a routine school course, however few the hours; a visiting teacher who will give them lessons in bed is admirable. The more they rest whilst in the preventorium, the sooner they will get back to normal school life. If the treatment accorded to primary tuberculosis is too short some cases will return in the category of Type II or III.

Types II and III Tuberculosis.—Such cases are seldom suitable for home treatment. The question then arises as to the kind of institution to which they should be sent. Bone and joint tuberculosis requires an open-air surgical hospital; pulmonary tuberculosis requires an up-to-date sanatorium; both institutions should be equipped with theatre, X-ray plant, laboratory, resident doctors, and visiting specialists. As all these requirements (with the exception of a theatre) apply equally to an efficient preventorium, the ideal arrangement is to have all three types of tuberculosis treated in one institution, but in separate units. Such a children's tuberculosis hospital should consist of a large preventorium, a large surgical tuberculosis unit, and a smaller sanatorium or phthisical unit. On admission to an observation ward the medical staff will decide the unit to which the child is to be transferred; probably a small proportion of cases will prove to be non-tuberculous. A further advantage of treating in one institution all types of tuberculosis in children is that, although the processes of disease are the same, yet the feeding and nursing of children under five years calls for special

experience. The physicians and nursing staff become experts both in dieting and the handling of tuberculosis in children, and thus the very best results may be achieved. In time, if the preventorium department works efficiently, the demand for surgical and phthisis beds will decrease. An alternative arrangement, which the writer has found satisfactory, is to have a tuberculosis unit attached to an infants' hospital (under 5 years) as a completely separate roof unit ; here all forms of tuberculosis of the lungs are treated, also surgical tuberculosis pending vacancies in the surgical open-air hospital. Cases of primary tuberculosis over two years finish their cure, after 3–6 months in this unit, by transference to a preventorium outside the city ; nursing of cases aged 1 month to 2 years is expert and better if the nurses are trained in the general wards of the infant hospital. It is most necessary to have a number of single or 2-cot wards for observation on admission and also for subsequent isolation should need arise ; treatment wards should never be more than of 10-cot size, and should vary from 3 to 8 cots. It is essential to have much balcony space ; these should face not only south, but also east, west, and north, so that children can remain out all day ; many cases are unsuited to the hot midsummer sun of a south balcony. These balconies should be open to the sky, although some covered space should be provided for showers. Covered balconies attached to wards make the wards dark, cold, and sunless, especially in winter. The practice of allowing phthisical children to remain in the wards of general children's hospitals is to be condemned.

General Treatment

General treatment in primary pulmonary infection due to the tubercle bacillus may be summed up in one word—'rest'. It is more beneficial to healing for a child to lie in bed in front of an open window in the city, than for it to be running about and getting over-tired in the best country air. When this essential rest has been provided, the child requires other accessory factors towards healing : namely, fresh air, suitable food, correction of digestive disturbances, and the avoidance of intercurrent infections, both respiratory and epidemic.

Rest.—Rest is required for the diseased lung, and the best way to achieve this is to put the child to bed. The amount of rest required depends on the type of lesion and the degree of activity present.

Primary Tuberculosis.—Complete bed-rest should be given during the infiltrative stage. The age of the patient is an important factor; young children require longer rest to heal their lesions. *Children under 5 years* run a greater risk of hæmic spread than do older children. They need to spend the whole of the first three months after infection in bed, but may be allowed to stand up in their cots if they so desire. Most children take six to twelve months under the best conditions before their S.R. falls to normal and their radiographs show regression of shadows. Therefore in small children rest must be prolonged. *The school child* should rest two months in bed after primary infection; if then the temperature is normal and the S.R. under 10 mm. per hour, the child may be allowed up for first one hour per day, and then for two hours. If the S.R. remains under 5 mm. per hour, temperature normal, and the radiograph shows that the primary complex is clearing, the time out of bed may be increased gradually until it reaches 11 a.m. to 5 p.m. daily. This scheme for gradually shortening bed-rest works out in this way: the child is completely in bed for eight weeks, then up one to three hours a day for another month, and by the fourth month he may be up for six hours with one hour's rest in between. For the next two months of the cure, he can be up from 10 a.m. to 6 p.m. daily, with one hour's midday rest. Selected cases can do a couple of hours' lessons during the second three months, but the majority should do no lessons for six months. Cases treated at home should on no condition be allowed to attend day-school until the primary lesion has healed: six months in the mild cases, nine to twelve months in the more severe.

Erythema Nodosum.—*See* p. 49.

Primary Cavity.—These cases should be immobilized until a film shows that cavity has closed completely. Partial immobilization of small children may be effected by a restraining spinal corset, the straps of which encircle the mattress. If complete immobilization is required (as in chronic miliary tuberculosis) the child must lie on a spinal frame with restraining corset and thigh straps.

Mediastinal Gland Tuberculosis.—*Young children* show great enlargement of mediastinal glands, and consequently run more risk of hæmic spread. Here the first three months' treatment is of vital importance, and these children should be placed in preventoria and kept at rest until calcification in or regression

of the glands is observed. They need not be confined to bed after the first six months, if the S.R. is normal, but may be up for a short time every day and may do a little kindergarten work. Morning and evening rectal temperature must be recorded and the sedimentation rate estimated every two weeks ; any rise in either of these demands a return to complete bed-rest. Their stay in the preventorium will probably last for about eight to twelve months. This condition in the *older child* is on the whole benign, but tubercle bacilli are frequently present in the gastric washings and one has to guard against hæmic metastases to bone and joint, and to meninges. Their stay in the preventorium should be for at least three months, until the most critical danger period is over. The rest should be complete for the first month, and partial for the later period. If they return home after three months' preventorium treatment, they should not attend day-school for a further period of three months, and they should continue part-time rest at home in the interval. These children should be supervised for a couple of years after.

Atelectasis.—This condition should be treated in the same manner as mediastinal glands ; the children should be kept in the preventorium until re-aeration of the lung has taken place. *Young children* under 3 years are often affected, and in that age period treatment must be prolonged until healing is, clinically speaking, completed. Resolution of the gland or of the bronchial plug may take from two months to two years ; during this time phlyctens requiring treatment may arise. If the S.R. is raised and the gastric lavage contains tubercle bacilli, one suspects plugging of the lumen of a bronchus, and rest treatment must be much more strictly enforced than in gland-pressure cases : if the S.R. is normal and gastric lavage repeatedly negative, the condition is probably due to pressure from without by a gland ; in this case absolute rest is not essential, but every effort to facilitate calcification of the gland must be made. In the latter type of case, daily deep breathing exercises may be given with benefit. Atelectasis in the *older child* does not require absolute, only partial, rest after the first three months of infection. Any interference is unnecessary ; these cases clear well on rest (*see* p. 51).

Pleurisy (*see* p. 81).—These cases should be kept in bed until the effusion is absorbed ; then half-time rest for a further period of 6 months.

Secondary Hæmic Spread.—Young children suffering from hæmic metastases, such as localized miliary lung lesions, spina ventosa, tuberculides, etc., require to be confined to their cots until healing is well established ; this may take two years. Localized ultra-violet radiation is beneficial in spina ventosa. They should be accommodated in a preventorium or sanatorium for children. Many cases have open abscesses which discharge matter containing tubercle bacilli to a greater or less degree ; they are therefore a possible source of infection to other children, although the risk is not very great. Light plaster splints are valuable to immobilize hands, arms, or feet, which are infected by metacarpal, metatarsal, or phalangeal tuberculosis. Discrete miliary tuberculosis requires some degree of immobilization, less than provided for chronic miliary disease. Acute miliary tuberculosis responds to no treatment ; but as at first it is not to be distinguished from chronic miliary tuberculosis, it is advisable to ensure immobilization at the commencement of the disease, or as soon as diagnosed. Chronic miliary tuberculosis calls for the co-operation of doctor, nurse, and child, in achieving immobility of the whole body. Immobilization (*see* p. 88) followed by recumbency in these cases must be extended until clinical signs have disappeared, temperature has been normal for months, and sedimentation rate also normal, but chiefly until the radiological appearances indicate that healing is complete. If this rule is not adhered to, relapses will occur when the child is allowed up. Even in bed, certain cases will relapse and end fatally with acute miliary tuberculosis or with meningitis. The fact that at times calcified miliary foci are seen in the lungs of healed osseous lesions suggests that the complete immobilization of such patients may have contributed to their pulmonary cure. Open-air treatment, but no sun-bathing, is most important. Fish and other observers advocate as harmless and perhaps helpful the injection of gold.

Phthisis.—In children, phthisis must be treated by total bed-rest, as far as possible in the open air. In the early stages where there is reasonable hope of effecting a cure, complete rest is worth insisting on ; this applies particularly to children who have an early tertiary lesion in the infraclavicular region ; it applies also to those where a more extensive apical lesion has failed to collapse or to those who show commencing caseation and cavitation in any situation in the lung, more usually in the mid-zones and hilar regions. If, after a fair trial of this

complete rest, the disease continues to extend and the prognosis appears hopeless, the child may be relieved of wearisome rules.

Removal from Contact.—This has been regarded as an essential therapeutic measure in the treatment of childhood tuberculosis. In the past it has been considered a most important step, indeed in many cases the only necessary precaution, whilst rest treatment has not been insisted on. Reasons for removal were founded on the recognition of the extreme hypersensitivity of the young child who has been infected by the tubercle bacillus, combined with the fact that the child has no time in which to develop any appreciable degree of acquired immunity; for these reasons he is thought to be extremely susceptible to repeated and massive doses of further infection, such as he would receive from a phthisical mother. Repeated exposure to infection has hitherto been regarded as responsible for the occurrence of those widespread lesions and fatal forms of tuberculosis which we commonly associate with early child life. Wallgren, however, produced some striking and convincing views, which are in agreement with the findings of the more advanced school of pathology. These arguments may cause moderation of our idea of the need to maintain separation of an infected child from further contact. Wallgren's view is that repeated infecting doses are only inimical to a child whilst he is in the pre-allergic state, and that once allergy is established, the further entry of a few bacilli by the air-passages is too insignificant to influence the course of an existing primary pulmonary lesion which contains already millions of tubercle bacilli. He adopts the attitude that hospitalization is necessary only because rest-therapy, fresh air, and good food cannot always be provided at home, and not on account of danger of superinfection: his view is revolutionizing our procedure on the subject. Still, it is hard to kill the old familiar idea—namely, that continued close contact with a phthisical mother adversely affects the infant's infection after allergy is established. Until this question has been more widely discussed and the new view proven and accepted, one cannot feel justified in taking any other course in practice except the old one of removing all infected children, who are undergoing treatment, from contact with phthisical adults. At present, one can perhaps already moderate one's treatment in so far as to allow partially healed cases to return home at an

earlier date than would previously have been permitted, provided rules can be carried out in the home.

Fresh Air.—Fresh air is most important, not only for its beneficial action in producing resistance to the tuberculous condition, but also in the general strengthening of the child, so that he may be enabled to withstand intercurrent infections. Children soon become very hardy and can remain out of doors in their cots all day. Infants and very small children must, however, be treated rationally, and must not be exposed beyond the limits of their endurance; no child should be allowed to become blue with cold. Open-air treatment must be administered judiciously in cool climates.

Sun.—In all cases of pulmonary tuberculosis, that is to say, Types I, II, and III, exposure of the thorax to strong sunshine or to ultra-violet rays must be avoided absolutely during the active, and especially infiltrative, stages. Both natural sun and artificial light are known to be releasers of exudation; therefore they are contra-indicated in all conditions of an exudative nature; this includes all active primary pulmonary lesions, tuberculous mediastinal glands if active, and the great majority of Type II and III lung lesions in the child. Sunshine has a beneficial effect on bone and joint infections, on discrete cervical glands, and on abdominal tuberculosis, for these lesions are rather of a productive type. But the lung does not benefit in the same way; the ill effects of over-exposure to sunlight of pulmonary lesions is witnessed by spiking temperatures, general malaise, and non-healing or even extension of the infiltration as seen radiologically. Children with active pulmonary lesions should be out in the fresh air all day, but in hot summer they should be kept in the shade; in the winter, as well as in the early spring and late autumn, in these climates, it is safe to expose to the midday sun. As healing advances, and calcification commences, and mediastinal glands become encapsulated, it is safe to expose to morning and evening summer sun, and to protect only against midday summer rays. At all stages of the disease, children should not be allowed to become grossly overheated, and even in later stages it is wiser to keep the chest covered with a vest, and to tan only the arms, legs, and face. Secondary pulmonary dissemination, miliary disease (discrete and chronic), as well as Type III phthisis should on no account be exposed to either sun or ultra-violet rays at any time, although fresh air and sky-shine in abundance are most beneficial to these cases.

DIETS FOR CHILDREN AGED 3–5 YEARS IN T.B. WARD

DAY	BRKFST	9 A.M.	10 A.M.	LUNCH	TEA	6 P.M.
Monday	Cereal Milk Sugar	Egg flip Bread Butter Jam	Orange Juice	Mince meat Broccoli, minced Potato, mashed Apples, Custard WATER	Orange Juice Scrambled Egg Baked Custard Bread, Butter, Milk, Salad	Cocoa Bread Butter
Tuesday	Porridge Milk Sugar	Egg flip Biscuits Butter	Orange Juice	Cod, minced Spinach, sieved Potato, mashed Prunes, Custard WATER	Orange Juice Tomato Eggs or Scrambled Egg Milk Pudding and Egg Bread, Milk, Butter	Cocoa Bread Butter
Wed'sday	Cereal Milk Sugar	Egg flip Bread Butter Jam	Orange Juice	Mince meat Cabbage, sieved Potato, mashed Apples, Custard WATER	Orange Juice Boiled Egg Baked Custard Milk, Bread, Butter	Cocoa Bread Butter
Thursday	Porridge Milk Sugar	Egg flip Biscuits Butter	Orange Juice	Liver, sieved Peas, mashed Potato, mashed Gooseberries and Custard WATER	Egg & Tomato or Blancmange and Egg Milk, Bread, Butter, Jam	Cocoa Bread Butter
Friday	Porridge Milk Sugar	Egg flip Bread Butter Jam	Orange Juice	Whiting, mashed Spinach, sieved Potato, mashed Rhubarb and Custard WATER	Boiled Eggs Baked Custard Bread, Butter, Milk Orange Juice	Cocoa Bread Butter
Saturday	Cereal Milk Sugar	Egg flip Biscuits Butter	Orange Juice	Kidney, minced Broccoli, sieved Potato, mashed Apples and Custard WATER	Rasher Tomato or Scrambled Egg & Tomato Baked Custard Milk, Bread, Butter Orange Juice	Cocoa Bread Butter
Sunday	Porridge Milk Sugar	Egg flip Biscuits Butter	Orange Juice	Mince meat Cabbage, sieved Potato, mashed Rhubarb and Custard WATER	Orange Juice Scrambled Egg Milk Pudding and Egg Bread, Butter Jam, Salad, Milk	Cocoa Bread Butter

Oranges : 1/child/day
Eggs : 1/child/day
Milk : 3 and 5 years old—2 pts./day ; 1–2 years old—1½ pts./day.

ANALYSIS

DAY	PROTEIN	FAT	CARBO-HYDRATE	CALORIES	CALCIUM	IRON	VIT. A	ANEURIN	RIBO-FLAVIN	NICOTINIC ACID	VIT. C	VIT. D
	g.	g.	g.		mg.	mg.	i.u.	i.u.	mg.	mg.	mg.	i.u.
Sunday	70	76	195	1764	2056	9·3	4238	320	2·07	4·6	121	1759
Monday	71	76	198	1780	2046	9·6	4656	347	2·1	5·1	119	1759
Tuesday	71	74	203	1770	2056	9·8	8756	344	2·17	5·3	127	1789
Wednesday	70	76	197	1772	2057	9·4	4246	323	2·07	4·7	122	1759
Thursday	72	74	202	1786	2055	12·0	8984	379	2·6	8·2	146	1759
Friday	71	72	196	1742	2041	9·0	8366	320	2·12	5·2	121	1759
Saturday	70	75	200	1775	2029	11·0	4716	388	2·38	6·3	128	1748

FULL FIGURES FOR SUNDAY'S MENU

Porridge	2	1·2	10	59	8	1·6	0	24	0·02	0·2	0	0
Beef 1 tablespoonful	4·4	3	0	44	1	1·2	14	12	0·04	1·5	0	0
Cabbage 1 oz.	0	0	1	4	15	0·2	80	6	0·01	0·1	8	0
Potato 4 oz.	2	0	20	56	8	0·8	32	48	0·08	1·6	16	0
Rhubarb	0·1	0	0·2	1	5						2	
Egg 1½	8	7	0	95	33	1·7	700	28	0·19	0·05	0	35
Milk 2 pts.	36	44	52	800	1200	0	1200	120	1·6	0·4	0	40
Bread 6 oz.	18	3	78	408	56	4·2	0	56	0·08	0·88	0	0
Butter ¾ oz.	0	18	0	162	3	0	800	0	0	0	0	8
Sugar 1 oz.			26	104								
Salad					5	0·2	300	10	0·01	0	2	0
Orange 1	0	0	8	32	48	0·4	112	16	0·04	0	45	0
Vitamin C tablet 1*											50	
Ostocalcium tablet 1*					700							500
Cod-liver oil 2 teaspoonfuls							11,760					1176
Totals	70	76	195	1764	2056	9·3	14,998	320	2·07	4·6	121	1759

* The dose, 1 to 3 tablets, is ordered by the doctor.

DIET SHEET, WITH ANALYSIS OF FOOD VALUES

(*Prepared by Miss Hazel Judd for the Tuberculosis Unit, St. Ultan's Hospital*, 1947)

Diet.—Frequently in the early stages of primary pulmonary tuberculosis the child suffers from dyspepsia. Babies vomit and older children complain of abdominal discomfort and show anorexia. A very bland and light, though nourishing, diet should be given for two or three weeks until these symptoms have disappeared. A good prescription for this condition is :—

Pink Mixture

Bismuth Carbonate	1 gr.
Sodium Citrate	2 gr.
Tinct. Cardamom. Co.	2 min.
Glucose	5 gr.
Water to	1 drachm

One drachm three times a day after food. Older children may take two drachms three times a day.

It is useless to force a heavy fat-rich diet on these children at first. When they have regained their appetites and are able to digest well, give a good simple wholesome mixed diet, without stuffing the child, with sufficient protein and fresh fruit, and plenty of vitamins C and D. If cod-liver oil is not well tolerated at first, give 5 to 10 drops of ostelin in glycerin daily. Later, give up to a tablespoonful of fresh cod-liver oil once a day.

Drugs.—Medicine is unnecessary, with the following exceptions : Pink mixture for digestive disturbance ; cough mixture for superimposed catarrhal conditions ; calcium 10 to 20 gr. daily with a minimum of 1500 I.U. of vitamin D ; cod-liver oil, malt, and Parrish's food in moderation, two to four drachms daily. A low hæmoglobin percentage is frequently found during primary infection, but will improve when the dyspepsia is cured and normal appetite returns ; in cases where the anæmia persists, ferrous sulphate is well tolerated, and will speedily raise the hæmoglobin percentage. Orange juice daily supplies vitamin C and also prevents constipation, and should an aperient be required syrup of figs gives satisfactory results.

Ferrous Sulphate Mixture

Ferrous sulphate	6 to 10 gr.
Glucose	10 gr.
Water to	2 drachms

Two drachms three times a day after food.

Specific Treatment

Artificial Pneumothorax.—This procedure would be an unjustifiable and wholly unnecessary interference in the treatment

of *primary lesions* in pulmonary tuberculosis. Collapse of the lung tissue around a primary focus will not accelerate calcification in the focus; spontaneous healing is safer; it is not slow and it is very sure under rest therapy. Spreads of a secondary nature are prevented by rest better than by collapse, which cannot affect the mediastinal glands and indeed may only give a false sense of security, whilst a glandular danger point is as active as ever. If, however, the primary focus does not heal, and caseous pneumonia occurs at the site with central cavitation, then collapse therapy should be considered; if collapse can be successfully achieved in such a case, bronchogenic spread from the cavity will be avoided. In young children the spontaneous closure of primary soft-walled cavities is by no means unusual; but in infants one seldom finds a non-healing primary complex without some hæmic metastases; the latter are contra-indications for collapse therapy. It has been suggested that artificial collapse of the lung during the course of a massive pulmonary atelectasis will prevent the subsequent development of bronchiectasis; theoretically this measure might prove to be of value in those cases of primary tuberculosis when atelectasis is due to plugging of a bronchus, and where an infective element arises.

The main indication for artificial pneumothorax is in the older child during the early stages of a caseous infiltration around an Assmann focus. The induction of an artificial pneumothorax is not to be undertaken lightly in a young child or infant for several reasons. Even if the initial collapse is successful, it is not easy to keep it up; air has to be replaced froquently, two or three times a week over a considerable period; the small child is difficult to nurse or feed when it has a ' needle dread ', in fact it is almost necessary to give an anæsthetic at each refill. Often adhesions are present due to small pleural effusions. In practice, therefore, it will be found that even though collapse can be induced at any age, no matter how early, yet in many cases it has to be abandoned long before the cavity is closed or the lung healed. In children of school age there is some difficulty at times in obtaining collapse, but it is generally successful; indications are early tertiary lesions in the infra-clavicular region. Tuberculosis of a caseous nature in the hilum region presents a very real difficulty, for these lesions are very resistant to collapse, owing to their anatomical position and to pleural adhesions. At late school age and during

puberty, those ages when the therapeutic value of the procedure is greatest, collapse of free lung tissue is as simple as in the adult provided the operator has gained the confidence of the child. Even cauterization of adhesions can be practised on children by a physician whom the child knows and trusts.

Phrenic Crush.—This operation is permissible and often successful in a few chosen cases, usually lower-lobe phthisis. These cases are, however, extremely rare in children, and the operator must have assured himself beyond a doubt that he is dealing with Type III phthisis and not with a primary infiltration or an atelectasis, which are much more common in this situation. The presence of tubercle bacilli in the sputum indicates phthisis.

Thoracoplasty.—This would seem to be a drastic procedure in children ; nevertheless it may be performed in cases where no other form of treatment is of any avail, and in a few cases it may prolong life. In considering such an operation, it must be remembered that in the child the course of phthisis is rapid, the mortality-rate high, and there must be no undue optimism as to the results. The operation is borne as well by the older child as by the adult.

Gold Treatment.—Gold has not been used very widely in the treatment of pulmonary tuberculosis in the child. Its employment is contra-indicated in the first stage, where it might have a deleterious toxic effect on a hypersensitive condition which responds very well to rest and non-interference. Its only use perhaps is in the treatment of rare chronic forms of Type III phthisis or of chronic miliary tuberculosis. Fish found it to be of benefit in chronic miliary disease of the lungs, although he did not attribute his cures entirely to its effects. Other experts advocate the use of gold by muscular injection in cases where a successful collapse of a phthisical lung has been obtained and subsequently the other lung shows signs of commencing miliary spread. Fish gave solganol intramuscularly, beginning with 0·005 g. at weekly intervals increasing up to 0·2 g.

Tuberculin Therapy.—Tuberculin therapy is contra-indicated during any condition of pulmonary activity. When the lung lesion is completely healed, there is a sphere of usefulness for tuberculin in the treatment of single isolated and remote foci, if these persistently resist other methods of treatment. Such cases are mainly those of chronic eye lesion and of tuberculosis of the kidney. Forms of dosage in such cases are discussed under the appropriate diseases.

What to Avoid.—In treating tuberculosis of the lungs in children, avoid :—

Exposure to strong sunshine.

Exposure to ultra-violet rays.

Tuberculin therapy in all cases with active pulmonary or abdominal lesions.

Irritation therapy, such as poultices, inhalants, etc. ; these may be administered temporarily for intercurrent bronchitis or upper respiratory infections during the healing stages of the primary infection. They should, however, be avoided over the site of the primary lesion or in tertiary phthisis.

Irritation of the lungs due to respiratory infections. Colds, bronchitis, influenza, measles, whooping-cough, and pneumonia—all react adversely on the course of a primary tuberculosis, and may be extremely dangerous in Types II and III tuberculosis.

Intercurrent infections, such as scarlet fever, measles, or other epidemic infections of childhood which may penetrate into hospital wards are to be avoided as extremely dangerous to tuberculous children. Specific prophylactic and early therapeutic inoculations considerably reduce the mortality during such epidemics. Infection by Vincent's angina in nose and throat of small children is particularly dangerous in tuberculosis. Three months after the primary infection, the consequences of intercurrent infections become less grave. Strict isolation for 2 to 3 weeks in special wards, on admission, will reduce the incidence of intercurrent infections to a great extent, and should be insisted on in every hospital or preventorium for tuberculosis in children.

After-care.—After the first 6 months of treatment, there should be supervision of children suffering from primary tuberculosis for a further period of six months fortnightly in special clinics. For the second year, the child should attend the clinic once a month. For the third year he should attend once every three months. After that the parents should be warned to return at intervals, or if the child is not well ; and also they should be warned to be specially vigilant during the years of adolescence. Experience shows that satisfactorily healed primary foci at all ages do not tend to break down after two years have passed. This cannot be taken as a dogmatic rule, but generally it will be found to be the case. In Type II manifestations and bronchogenic phthisis not only will actual treatment have to be prolonged, but also very strict after-care will be necessary

for some years ; in these cases the danger of recurrence is greater than in the satisfactorily healed primary complex. In all after-care supervision, one should err on the side of taking radiographs too often rather than too seldom.

PROGNOSIS

A summary of the theme of the preceding chapters will indicate in a few words what is the prognosis of tuberculosis in children. Prognosis in primary tuberculosis generally is favourable. In the established disease of Type II or III the outlook is much less favourable, and in the young age group extremely bad. Thus any consideration of prognosis in primary tuberculosis must include not only the immediate but also the long point of view.

Immediate prognosis in primary tuberculosis is good ; it is good also even in the absence of treatment. The majority of primary tuberculous infections tend to be self-healing, and about half of them are symptomless, amongst children over 5 years of age. A small number of deaths occur in primary tuberculosis *per se* ; these are due to toxæmia (in infancy) and to cavitation of the primary focus (mainly in infancy, but also in adolescence). The fatalities directly due to primary tuberculosis, then, form but a very small proportion of cases so infected, yet they do occur from time to time.

The real danger associated with primary tuberculosis lies in the hæmic seeding which takes place, in many cases very early in infection ; these seedings may be present even in cases where there is no radiological evidence of primary focus or glands in the thorax. Determining factors for the development of subsequent disease are unknown, but the fact remains that extrapulmonary post-primary seedings will on occasion give rise to meningeal or bone and joint tuberculosis. In the same insidious manner, intrapulmonary post-primary seedings may develop into miliary disease, or even a single post-primary focus may be the forerunner of bronchogenic phthisis.

Ultimate Prognosis.—Thus it is clear that although the immediate prognosis of primary tuberculosis is nearly always good, and attended with very small risk to life, yet the *ultimate* prognosis in any given case may be unfavourable. It is impossible to predict which case will heal and which will proceed to the development of one of the more serious forms of tuberculosis. Until recently few figures were published to show what proportion of cases of primary tuberculosis run an unfavourable course, that

is, by extension of disease ; testimony to the fact that a number of cases do extend is provided by the patients to be found in sanatoria and hospitals for surgical tuberculosis and also amongst the fatalities from tuberculous meningitis ; all these persons were at one time suffering from a simple primary complex. Evidence having regard to the mortality and morbidity rates amongst young entrant nurses, published during the last decade, creates a strong impression that in the adolescent period primary infection not infrequently leads to the developments of Types II and III tuberculosis within a short space of time. Experience further shows that in all age groups the prognosis (ultimate as well as immediate) of primary tuberculosis is extremely favourable where rest therapy is instituted. In the acute forms of Types II and III tuberculosis the prognosis is much less favourable, although the chronic forms may be treated successfully ; in children, indeed, the more chronic forms are rare, and when once these types are established even rest may prove as unavailing in stemming disease as any other form of therapy ; in the early age periods recovery is rare. Thus it becomes evident that a favourable prognosis depends on the diagnosis of tuberculosis in the primary stage ; at this time efforts to heal all lesions (primary and post-primary) will influence the ultimate as well as the immediate prognosis.

Various factors are quoted as having an influence on the prognosis of tuberculosis. Four of these will be mentioned here, but rather as indicating guides to treatment than as factors which determine prognosis : (1) Inherited resistance is undoubtedly of great importance, but impossible to assess. (2) Housing—it has been demonstrated that under conditions of overcrowding healing is delayed. (3) Diet—a deficiency below certain standards retards or inhibits healing, especially of the primary lesion ; in the 0–5 age period insufficient calcium absorption is deleterious to the healing of primary tuberculosis. (4) Age—tuberculosis is dangerous and prognosis less favourable in infancy and in adolescence ; the 5–12 age period is a more benign one, but not free from various risks.

The influence exerted by these four and any other factors on the course of tuberculosis may be overruled to a very considerable extent by the adoption of two important measures ; these are :—

a. Early diagnosis of primary tuberculosis by means of the tuberculin test, followed by X-ray examination of positive reactors.

b. Rest—bed-rest at the earliest possible moment during primary infection, rest adequate to ensure healing of the primary complex and also of all possible (but hidden) post-primary seedings.

The efficacy of these measures (early diagnosis and rest) has been proved by the author (1947) when dealing with ill-nourished infants from Dublin slums ; that is to say, at the worst possible age from a most unfavourable milieu. But it is fair to add that, in spite of all precautions, certain cases will slip through one's fingers ; for example, three cases, recognized within a few days of conversion from negative to positive, and treated, ended fatally within three months : two were infants under 6 months, awaiting B.C.G. vaccination, who died of primary and meningeal tuberculosis respectively ; and an adolescent who died of miliary disease (*see* p. 121). The author's experience over many years has led to the following conclusion : where early diagnosis and rest are employed a good immediate and ultimate prognosis may be expected in about 98 per cent of cases of primary tuberculosis ; where rest is not instituted or is given too late, a favourable prognosis cannot be given with the same degree of confidence. The prognosis in hæmic and bronchogenic tuberculosis, once the lesions are established, is distinctly unfavourable in children, extremely bad in infants.

PREVENTION

General Remarks.—The aim of prevention of tuberculosis in young life may point in two directions :—

1. The total eradication of tuberculosis.

2. The prevention of secondary and tertiary tuberculous manifestations : (*a*) By preventive inoculation ; (*b*) By early diagnosis of the primary stage of tuberculosis.

1. The total eradication of tuberculosis may be achieved in years to come by the compulsory control of all persons with positive sputum, and by the extermination of all tuberculous cattle. The eradication of mouth-to-mouth infection by human beings can only be effected by compulsory notification of all persons with positive sputum, by diligent search for these, and by their compulsory segregation until they become sputum-negative. This segregation must be accompanied by sanatorium rest, and also by the various more modern methods of cavity closure, such as collapse therapy, phrenic evulsion, thoracoplasty, etc. The adult case will have to be tempted to come

forward at the earliest possible stage by the offer of increased sick benefits to those who are in a stage capable of lung collapse. Tremendous compulsory powers would be required for the effective conduction of such a campaign, although it could be done. Therefore other measures have to be examined which might be undertaken more easily and immediately for the protection of child life from infection by the tubercle bacillus.

2. (*a*) There is one outstanding prophylactic measure which has passed from the phase of experiment to the stage of established fact; that is B.C.G. preventive vaccination. It is now well recognized that this measure has passed beyond the realm of doubt or danger, and can (in conjunction with other measures) reduce childhood tuberculosis mortality to very low figures. To achieve this a reliable vaccine must be employed and certain rules must be followed closely. A negative reactor is given a small, harmless, and controlled primary complex in an arm or leg, sufficient to immunize against further exogenous reinfection, and without danger to the child of immediate or remote sequelæ of the inoculation.

(*b*) The alternative scheme is to allow the child to acquire his primary complex by chance from a human source of infection; to recognize its inception by routine methods of tuberculin skin testing, and to give immediate treatment which will be adequate to ensure complete healing of the primary lesion, and thus prevent the development of those forms of tuberculosis which are inimical to health or even to life itself. In other words the infection is restricted to the first stage.

Routine examination of contacts, fruitful as it may prove, is not sufficient; search by means of the skin test must be made amongst groups of apparently healthy children, as well as those complaining of ill-health. If such a scheme is worked widely amongst the child population of any community, there will follow a reduction in the incidence of the more serious forms of tuberculosis and the mortality rate will be lowered.

Eradication of Adult Tuberculosis.—Such a discussion is outside the scope of this book. Two important points, however, require to be mentioned. Firstly that the elderly chronic phthisical grandparent, who forms such a source of danger to the small child, should be segregated from the younger generation. Secondly, the phthisical young mother who is pregnant should be given special attention; if she has a collapsed lung, she should get a refill as soon as possible after

parturition ; thus in many cases the post-partum exacerbation, so full of danger to the nursing infant, will be avoided ; this procedure is practised in many places now.

Eradication of Tuberculous Cattle.—This has been done in some countries, for instance Norway, with extraordinarily good results in effecting the reduction of tuberculosis due to the bovine type of bacillus. Where it is not done, all milk fed to infants should be boiled, except from a tubercle-free herd. The habit of pasteurizing all milk coming into a city has a beneficial effect on the incidence of tuberculosis in that city ; such measures, however, tend towards the production of dirty milk, and the child fails to acquire resistance against other disease, such as undulant fever for example. It seems that it would be easier to eradicate tuberculosis in cows than in humans, yet this campaign has not been undertaken seriously ; the total cost of such a campaign would hardly be greater than the cost of subsidizing the many institutions where the victims of bovine tuberculosis remain for months and years.

Bacillus Calmette-Guérin Vaccine (B.C.G.).—

Rationale.—No satisfactory immunity against tuberculosis has as yet been produced by artificial means other than that of introducing living tubercle bacilli into the tissues in a manner that will create a primary complex. B.C.G. is a vaccine of bovine-type living tubercle bacilli which have been rendered avirulent by numerous passages on glycerinated bile-potato medium. A measured dose of these avirulent bacilli is introduced intradermally into the upper arm of a hitherto uninfected individual. The resultant controlled primary complex is harmless to the subject and at the same time sufficiently active to produce tuberculin allergy (positive skin test). It has been proved by trial that the tuberculin allergy produced by a minimal B.C.G. lesion is capable of providing an immunity which affords protection against tuberculosis for a number of years. Originally two routes were employed in B.C.G. vaccination of infants, oral and intradermal. The oral method has been discarded because there is no guarantee what number of bacilli penetrate the tissues, although a measured dose may be administered, and because a primary complex in the ileum and mesenteric glands cannot be inspected. Therefore the intradermal route is now employed in infants as well as adults as being more efficacious. Good reports are also quoted recently of transcutaneous vaccination, that is, by a method of multipuncture

devised by Rosenthal and elaborated by Birkhaug; it would appear that there is little to choose between the transcutaneous and intradermal routes.

If B.C.G. vaccination is to prove a successful immunizing agent for the protection of infants, children, and adults against tuberculosis, then certain rules must be observed :—

1. Only tubercle-free persons are injected (negative reactors).
2. Tuberculin sensitivity must follow injection (positive test).
3. Contacts must not be incubating tuberculosis at the time of vaccination (tuberculin negative 6 weeks after removal from contact).
4. The vaccinated person must not be exposed to infection until allergy is established (positive test).
5. A reliable vaccine in a fresh condition must be employed.

Results.—Since Professor Calmette first used B.C.G. vaccine on humans in 1922, some millions of newborns, children, and young adults have been inoculated. No case of death from a pure strain of B.C.G. has been reported; nor is there evidence that a return to virulence of B.C.G. takes place in human tissues. It is true that reports in the literature do not show a similarity of good results; but on examination of these reports it will be found that where the above five rules have been observed the results are extremely favourable, whilst even with partial observance they are good; but where the vaccine is administered without due regard to the underlying principles which are based on a knowledge of the development of primary tuberculosis, results suggest that it is hardly worth the trouble entailed. Reference will now be made to a few of the good results. Outstanding amongst children is the work of Wallgren (1941), begun in Gothenberg in 1927 and subsequently carried on by Anderson and Belfrage (1939); they succeeded in that city in reducing the infant tuberculosis death-rate from 3·4 to 0·5 per thousand in a decade. Reviewing 905 vaccinated children after 3 to 10 years, they found that none had died of tuberculosis and only 2 had contracted very mild forms of primary tuberculosis; only 207 of these had no known exposure to tuberculosis in the interval. Reports from Scandinavia (Heimbeck, Nordwall) indicate that protection is afforded to young nurses (exposured to infection) by B.C.G. vaccination, and leave an impression that this artificial immunization is capable of protection equal to that conferred by a naturally acquired and healed lesion. Ferguson (1946), employing B.C.G. vaccination in hospitals and sanatoria

in Saskatchewan, succeeded in reducing the incidence of tuberculosis in young nurses to one-fifth of that amongst negative nurses who had not been vaccinated. Rosenthal (1945), from Chicago, reported of non-contact infants under 1 year, that amongst 1204 B.C.G.-vaccinated there was 1 death and 3 cases of tuberculosis, and amongst 1213 non-vaccinated controls there were 4 deaths and 23 cases of tuberculosis ; he further reported amongst 98 vaccinated newborn contacts no deaths and 1 case of tuberculosis, whilst amongst 63 non-vaccinated control contacts there were 3 deaths and 4 cases of tuberculosis. Aronson (1946) reports on a series of American Indians aged 0–20 years, reviewed after 6 years ; amongst 1550 vaccinated cases 4 had died and 40 contracted tuberculosis, whilst amongst 1457 non-vaccinated controls 28 had died and 185 had contracted tuberculosis ; in this series there was no possibility of arranging for pre- and post-vaccinal isolation, but nevertheless the good effect of B.C.G. vaccination was demonstrated.

The duration of immunity after vaccination varies according to the individual and according to the vaccine employed. Wallgren found that 97 per cent of his 905 vaccinated children still reacted positive when reviewed between three and ten years after vaccination. Reports from American observers indicate that reversion from positive to negative appears to have taken place in about 20 per cent of their cases six years later. When reversion to negative takes place, re-vaccination has to be considered.

Administration.—Only non-infected children are suitable for vaccination. To ensure true negativity, the child must be negative immediately before vaccination to an end-point of 100 tuberculin units (T.U.), that is, Mantoux 1–100 or P.P.D. 0·002. When vaccinating contacts, it is necessary to make sure that they are not incubating tuberculosis ; this is done by removing child from contact or contact from child for a six weeks' pre-vaccination isolation period and then re-testing before vaccination. After vaccination no child should be in contact with an 'open' case until the vaccination has 'taken', as evidenced by a positive tuberculin reaction. Newborn contacts may be vaccinated when a few days old, provided that they have been removed *at birth* from contact ; but they must then remain in a tubercle-free environment until they react positive ; newborns suffer no ill effects from vaccination. The response to tuberculin after B.C.G. vaccination is seldom very strong, and experience

shows that for three months after vaccination it is safe to employ as a single test Mantoux 1–100. The time taken for B.C.G. allergy to develop varies with the vaccine employed and according to the individual; no arbitrary period of six weeks can be laid down; the author, employing Gothenberg vaccine, which owing to transport delays was between 7 and 12 days old, found that the pre-allergic period varied between 21 and 56 days for no obvious reason. If the test remains negative and no nodule is felt two months after vaccination, the inoculation should be repeated. As soon as the test is positive, the child may return to his tuberculous milieu. In non-contact negative reactors, vaccination may be performed in an extern clinic, and pre- and post-vaccinal isolation may be omitted; such cases must return after six to eight weeks for tuberculin test, otherwise immunity cannot be ensured. Dr. Wassén's Gothenberg vaccine must be used within fourteen days of preparation; 0·1 c.c. is introduced intradermally in upper arm or thigh; the injection must be true intradermal, raising a white weal. No malaise, rise of temperature, or reaction of any kind is experienced after vaccination; two or three days later there may appear slight reddening at the site which disappears after a few days (this is not to be confused with Koch's phenomenon, i.e., quick response of a positive reactor to B.C.G.); nothing is then seen or felt for some weeks. After a few weeks a nodule is felt and may also be seen: the tuberculin test becomes positive about this time; seldom is the gland component of the B.C.G. complex palpable. Two to four months after vaccination there may occur slight oozing from the B.C.G. nodule, which should be covered but not compressed; oozing occurred in one-third of the author's cases and was regarded as a good 'take'. The end-result of the nodule is a small scar, which may fade after some years. Abscess formation or adenitis following intradermal vaccination with the Gothenberg vaccine seems to be extremely rare in competent hands. The author experienced a necessity for pre-vaccinal removal from contact, for 15 per cent of contact children under 2 years of age developed primary tuberculosis during their six weeks' stay in hospital whilst awaiting vaccination; had this precaution been omitted these cases would have been accounted 'failures' for B.C.G. In the same manner, unless post-vaccinal removal is provided in cases exposed to contact, primary tuberculosis may be contracted during the pre-allergic post-vaccinal period, thus creating more 'failures' for B.C.G. It is essential to follow-up

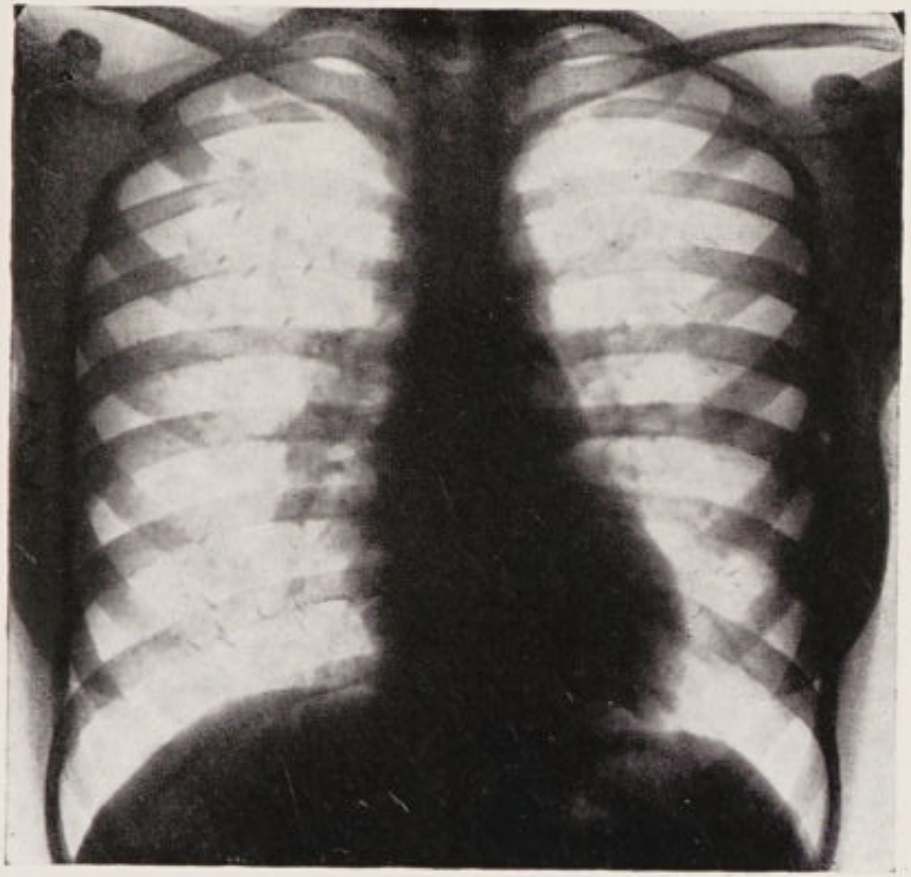


FIG. 50.—PRIMARY TUBERCULOSIS IN ADOLESCENCE.

Maeve H. Aged 18 years. (*See Example*, p. 120.) Enlarged gland in right hilum associated with initial fever. Primary focus not seen.

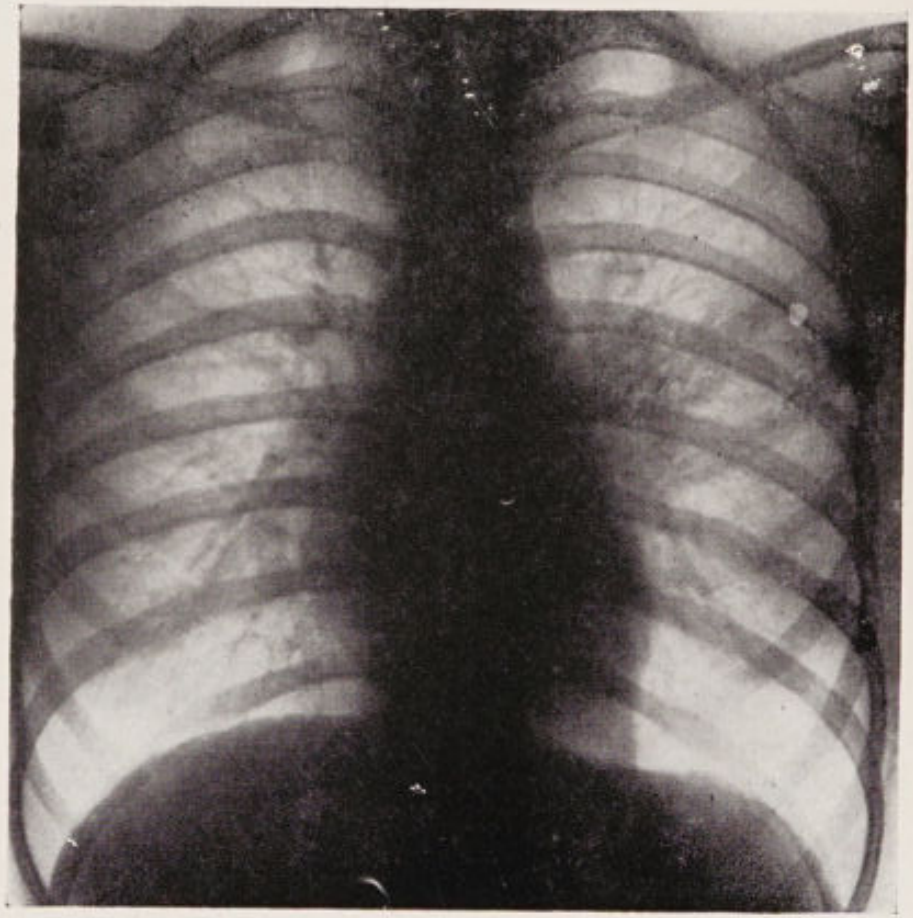


FIG. 51.—PRIMARY TUBERCULOSIS IN ADOLESCENCE.

Vera C. Aged 18 years. (*See Example*, p. 120.) The primary focus is seen in the left lung just below the artifact; the regional left hilar gland is enlarged and shows periadenitis.

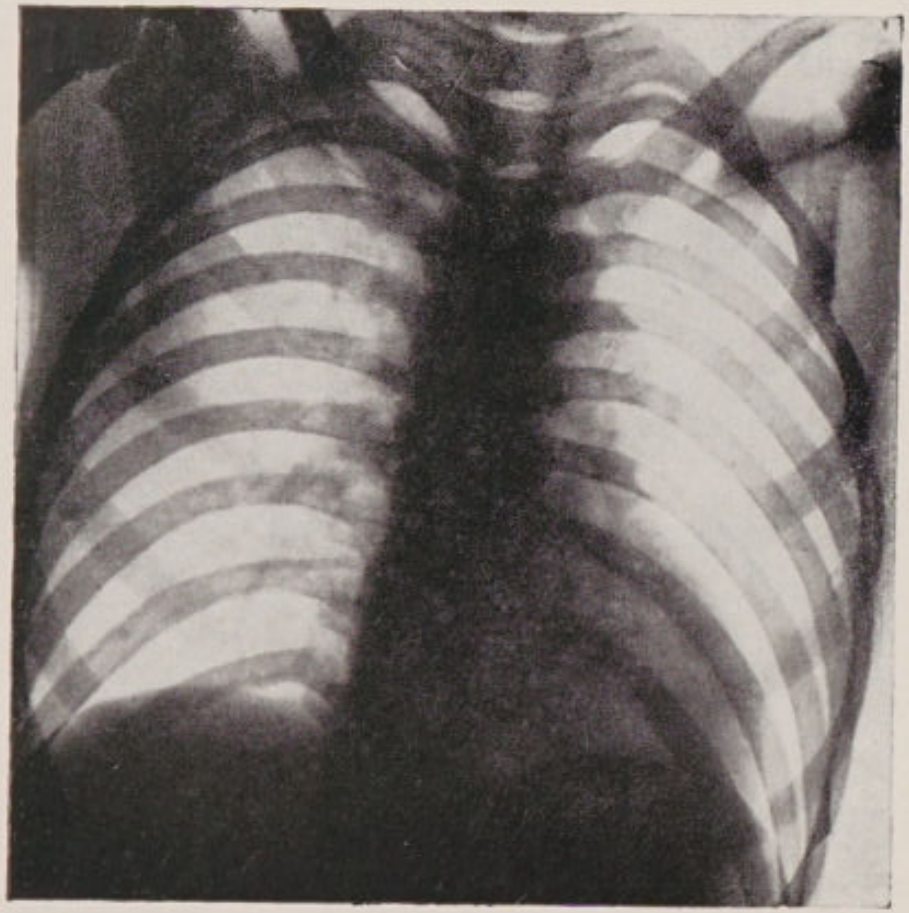


FIG. 52.—TUBERCULOUS MENINGITIS.

Edward C. Aged 14 years. (*See Example*, p. 121.) Tuberculous meningitis; enlarged gland in left hilum, primary focus not seen.

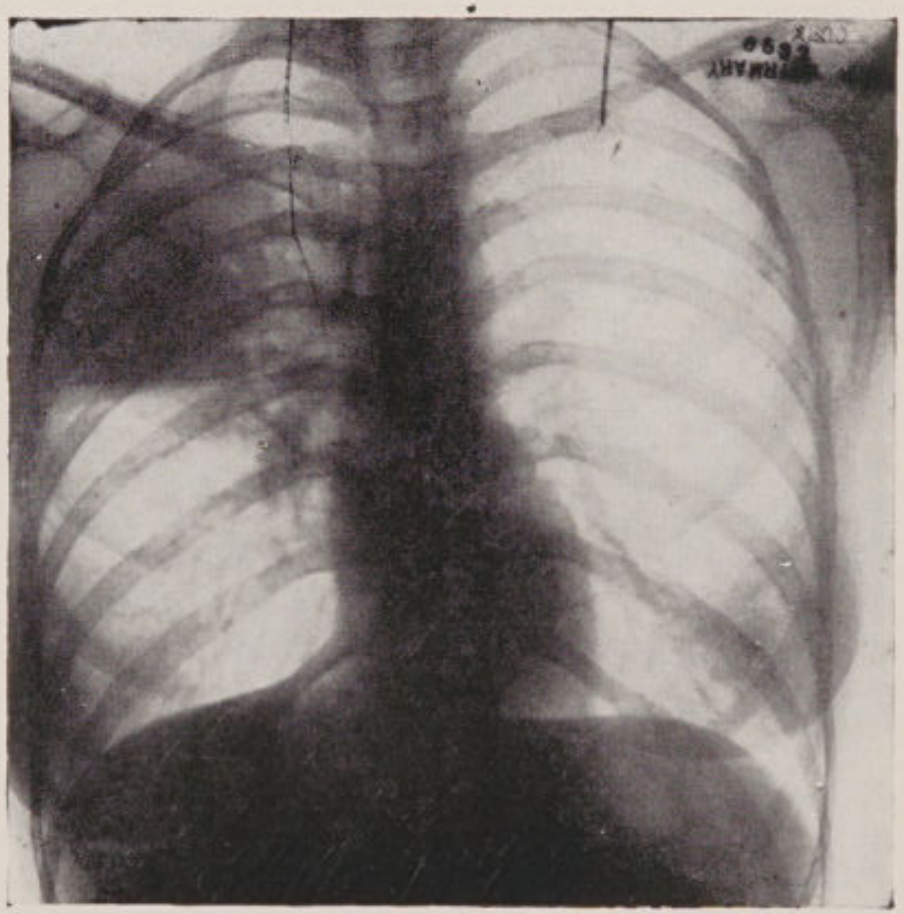


FIG. 53.—TERTIARY PHTHISIS IN ADOLESCENT.

Maureen C. Aged 15 years. (*See Example*, p. 123.) The opacity of the right upper lobe indicates a pneumonic rather than an atelectatic condition. Sixteen months later the girl had advanced bilateral phthisis.

vaccination cases by annual tuberculin test and chest film; annual testing will show should allergy wane, which on occasion may happen within two years of vaccination; annual radiological supervision is advisable because tuberculin tests can no longer be a guide to diagnosis of disease. In countries where B.C.G. has been in use for years, however, these precautions are less strictly observed; tuberculin testing is only performed after a lapse of several years, and chest film only taken when there are symptoms suggestive of tuberculous infection.

Early Detection of Primary Tuberculosis in Children.— —This can be achieved only by the co-operation of private practitioners, Child Welfare Officers, Tuberculosis Officers, and School Medical Officers, and by all physicians attached to intern and extern departments of hospitals where children are seen. Not only those children who come from a known tuberculous home, but all children, should be tuberculin tested on every available opportunity. If the percutaneous test is used, this will be an easy matter, as no instruments are required. Every child in a Baby Club should be tested at least twice a year, and every school child at least once a year. These tests may be safely performed and read by nurses who have been trained to do so. All positive cases must be X rayed immediately, and the decision made whether the case requires treatment or merely supervision. All doubtful cases, as well as all positive cases, can be referred to Tuberculosis or Hospital Clinics for further examination. Children's parents should be in a position to give immediate information as to the intrathoracic tuberculous condition of their children; they should know which are positive, and whether healed, and so avoid unnecessary repetition in radiography; and they should know which are negative and the date of the last test, just as every mother knows whether her child has been immunized against diphtheria. This tuberculin skin-reaction knowledge should be carried on to the danger period of adolescence; particularly at this age in schools, colleges, factories, and other works should the skin allergy of these young people be watched so that any change from negative to positive may be noted, and immediately dealt with further by radiological examination and appropriate treatment. There is no other way in which the disease of tuberculosis can be limited to the primary stage. Mass radiological examination of large numbers of school children and adolescents will catch a few cases during the infiltrative or glandular stage of the primary infection, or

in the stage of early infraclavicular infiltration ; many of these films are impossible to interpret correctly without the knowledge of the tuberculin skin reaction ; some may be labelled tuberculous which are not, and some may be missed.

This skin-reaction examination, as already remarked, should not be abandoned at 14 years, but should be carried on into young adult life. Nowadays at 14 years, over 50 per cent of children have still to face their primary tuberculous infection, and to desist because the child had left school would be a blunder. Particularly should young probationer nurses and medical students be examined on entry to work and on subsequent occasions each year, and also during any febrile attack. An initial febrile attack, often mis-named ' influenza ', when a known negative tuberculin test becomes positive, is a very real assistance in tracking down the actual moment when the tubercle bacillus enters the body for the first time. To reiterate, primary cases must be sought by widespread use of the tuberculin test in baby clubs, schools, colleges, factories, and amongst other groups of adolescents.

CHAPTER XII

EXTRA-PULMONARY TUBERCULOSIS: (1) EYES; (2) SKIN; (3) CERVICAL GLANDS; (4) ABDOMINAL TUBERCULOSIS; (5) RENAL TUBERCULOSIS

1. TUBERCULOUS EYE LESIONS

Our concern in this book is with the more common eye conditions which are seen in tuberculous children; such lesions demand correct diagnosis and treatment from the pædiatrician as well as from the ophthalmic surgeon and the tuberculosis specialist; recognition of their tuberculous nature is important, for it may suggest tuberculosis in hitherto unsuspected cases. Details may be found in the text-books on ophthalmology; here the clinical findings will be mentioned very briefly with particular reference to their relationship to the pulmonary condition. Tuberculous eye conditions in children may be divided into: (1) Primary or focal infections; (2) Secondary or blood-borne infections; (3) Allergic manifestation of tuberculosis (the phlycten).

1. Primary Tuberculous Eye Lesions.—Conjunctival tuberculosis is a primary infection of the conjunctiva, due to direct contamination by the tubercle bacillus in a hitherto uninfected child. This can occur when trauma of the conjunctiva is produced by rubbing with infected fingers, as for example in milkers who have been handling a tuberculous udder, or by the flick of the contaminated tail of a cow across the eye. The hypertrophic type of tuberculous conjunctivitis is the most usual, showing the cock's-comb excrescence on the fold of the lower lid; direct smear from this lesion will show tubercle bacilli. These cases are very rare. Blegvad sees 4 or 5 cases every year in the Finsen Institute from the whole of Denmark; he finds that they all come from rural districts, are due to the bovine bacillus, and occur mostly in children. Other cases can occur by direct spread from lupus of the skin in the neighbourhood,

such as nose or eyelid. Primary infection of the conjunctiva is suspected in farm workers where the pre-auricular glands situated in the temporal region become enlarged, whence at a later date infection spreads to the cervical group ; these cases, previously negative to tuberculin, now become positive reactors, and no other primary tuberculous focus is found.

2. Secondary Hæmic Infections.—

Irido-cyclitis is due to bacillary hæmic spread with lodgement in the iris, occurring secondarily to a lung infection. In some cases where the eye has been removed, tubercle bacilli have been found in the iris. Uncommon in children, the more usual age incidence is 14 to 25 years ; yet in older children the possibility of its occurrence must not be overlooked. The condition is a residue of a primary lung lesion, and it is usual to find a calcified primary complex in the chest radiograph. *Clinically*, tuberculous nodes amongst engorged blood-vessels may be seen in the iris. Treatment is often satisfactory if tuberculin therapy is undertaken (*see under* PHLYCTEN, *below*). The tuberculin injections must be continued for at least a year, often for two years, but lung must be healed first. General hygiene is important, with daily sun or light baths, again only when the pulmonary lesion is healed.

Miliary Choroiditis is a condition which also arises through hæmatogenous spread, but it is part of a generalized miliary tuberculosis. Tubercles may be seen in the choroid. There is no specific treatment, beyond that for the general condition.

Periphlebitic Tuberculosis of Retinal Vessels is seen in young subjects and occurs by hæmic spread. It is a very rare disease, and during its course hæmorrhages occur into the corpus vitreum. Tuberculin therapy should be considered.

3. Allergic Tuberculous Manifestation : Tuberculous Phlyctenular Kerato-conjunctivitis.—Hitherto phlyctenular ophthalmia has been considered as belonging to the province of the ophthalmic surgeon. But the condition should be approached rather from the tuberculosis angle, for this eye lesion is really an incident in the course of tuberculous disease. The condition according to Duke-Elder is " an allergic phenomenon—a reaction of the hypersensitive epithelium of the cornea and conjunctiva to any protein, although in the majority of cases the protein in question is tuberculo-protein". The author (1941) sums up the condition in these words : The conjunctival phlycten develops in children some time during the healing stage of primary tuberculosis, between five months and two years after primary infection,

most commonly nine months. This late appearance makes radiological pulmonary evidence scanty in a proportion of cases, and is responsible for the doubt thrown in the past on the tuberculous origin of the phlycten. The tuberculin test is positive in nearly all cases. Our lack of knowledge as to the connexion between the conjunctival phlycten and tuberculosis has been conveniently disguised by the employment of the term *allergic phenomenon*, whose precise meaning it is difficult to interpret. The phlycten has but little in common with other allergic phenomena associated with primary tuberculosis ; its tendency to recur provides a great problem. At present it is most readily acceptable to regard the phlycten as an allergic response to the constituents of the tubercle bacillus (possibly to tuberculo-protein), accompanied by a return of the hypersensitive state. The determining factor is the exposed position and high sensitivity of the conjunctiva.

Not all phlyctens can be proved as due to tuberculin sensitivity ; a small percentage may be caused by other, but seldom demonstrable, factors. It is safe to say that no case should be termed non-tuberculous until it has been shown that the child is negative to 1 mg. Old Tuberculin, given intradermally and repeated more than once. That the great majority of cases are associated with tuberculin allergy is shown in *Table XIX*.

Table XIX.—The Tuberculous Aetiology of Phlyctenular Disease

Author	Age in years	Number of Cases	Positive Tuberculin Test	Positive Radiographs
			per cent	per cent
Goldstein (1934)	—	60 Phlyctens	96·6	67·6
Siwe (1935)	0–13	140 Phlyctens	88	42
Sorsby (1936)	0–6	369 Phlyctens	79·6	73
	6–17		84·8	
	0–6	551 Blepharitis	9·5	16·1
	6–17		19·8	
Price and MacManus (1943)	0–18	140 Phlyctens	98·5	55 (43 (active primary); 12 (Type II or III))
McArevey (1944)	0–20	249 Phlyctens	85·9	37·3
		102 Controls	13·7	20 (45 cases)

A study of 140 cases of phyctenular ophthalmia from the tuberculosis angle will now be summarized (Price and MacManus,

1943). Out of 140 cases, 138 were positive tuberculin reactors, and of these about half had active lesions. Amongst 57 cases aged 0–5 years 70 per cent had active tuberculous lesions, and amongst 83 aged 5–18 years (80 of these were under 15) 44 per cent had active lesions. The first appearance of the phlycten is common at the age of greatest hypersensitivity to the tubercle bacilli and its products, that is in the 0–5 period. The primary focus amongst 138 cases was situated as follows : pulmonary 102 cervical glands 5, abdomen 1, middle ear 1, not found 29. There were physical signs in the chest in 7 per cent of cases, whereas in 55 per cent there were radiologically demonstrable active lesions (excluding calcified complexes and doubtful glandular enlargement). There were active primary lesions in 43 per cent of cases, and progressive lesions of Type II and III in 12 per cent of cases. There was no dental defect in 78 per cent of cases examined, and the tonsils were normal on inspection in 75 per cent of cases. The actual first appearance of the phlycten was observed in 34 cases, of which 18 were already under treatment for primary tuberculosis (6 domiciliary, 12 in hospital) ; it is interesting to note that in the 12 which were already in hospital when the phlycten first appeared, no recurrence occurred and there was no corneal involvement. In *Table XX* it is seen that the majority

Table XX.—Time of Appearance of Phlycten in 34 Cases of Primary Complex.

Less than 1 year	after	primary	infection	27 cases
1 to 1½ years	,,	,,	,,	5 cases
1½ to 2 years	,,	,,	,,	1 case
3 years	,,	,,	,,	1 case

Of the 27 cases which occurred in the first year the time was:

—	6 months	7 months	8 months	9 months	10 months	12 months
Cases	7	3	3	9	1	4

of phlyctens appeared within a year of primary infection, usually 6–9 months after ; this represents in treated cases a healing phase in primary tuberculosis, when hypersensitivity is waning and some degree of immunity has been established ; on 27 occasions amongst 57 in the under-5-year group the appearance of the phlycten coincided with pressure atelectasis.

Amongst 140 phlyctenular cases observed, osseous lesions subsequently developed in 7 instances.

Hip : Primary tuberculosis of lungs, Jan., 1941, aged 4 years. Phlycten, July, 1942. Hip, Nov., 1942.

Spine : Primary tuberculosis of lungs, June, 1937, aged 1 year. Phlycten, Aug., 1938. Spine, Nov., 1938.

Knee : Primary not known. Phlycten and enlarged paratracheal glands Oct., 1940, aged 7 years. Knee, Sept., 1941.

Shoulder : Primary not known. Phlyctens and shoulder, 1939. Shoulder not healed and enlarged hilar glands, aged 10 years, Jan., 1942.

Elbow : Primary tuberculosis of lungs, Jan., 1937, aged 1 year. Phlyctens, May, 1938. Elbow, Jan., 1941.

Elbow : Primary not known. Phlyctens, March, 1943, aged 11 months. Elbow and dactylitis, May, 1943.

Dactylitis : Primary not known. Dactylitis and phlyctens seen together, chest radiograph negative, Feb., 1943, aged $2\frac{1}{2}$ years.

It is necessary to differentiate between first attacks (Fresh Phlycten) and subsequent attacks (Recurrent Phlycten) ; such a distinction by the ophthalmic surgeon as well as by the physician is essential to correct treatment and prognosis. It is the neglected fresh phlycten which is liable to corneal involvement and to recurrence, and prognosis is more favourable where a fresh phlycten is treated promptly for ocular and tuberculous lesions. In the series quoted above, the radiological appearances were positive in 66 per cent of fresh and in 49 per cent of recurrent phlyctens. All fresh phlyctens benefit by hospital treatment, in order that the tuberculous lesion may be discovered and treated according to need, and in order that local application to the eye may be satisfactorily carried out. With recurrent phlyctens, local medicament is usually of first importance, but examination for tuberculous lesions must also be made ; nevertheless in practice it will be found that a week's bed-rest during a flare-up in a phlycten is extremely beneficial in addition to local treatment. The fact that phlyctens develop for the first time in children during treatment in a tuberculosis hospital suggests that the development cannot be accounted for by lack of vitamins, poor diet, dirt, or parasitic infections ; these may, however, play a part in multiplying recurrences. Nor is treatment of primary tuberculosis wholly successful in preventing the development of a fresh phlycten, although it is experienced that in these treated

cases the phlycten tends under local treatment to abort and does not recur, at any rate to any such degree as the neglected one.

Clinical.—A phlycten is a node arising by exudation of leucocytes into the deeper layers of the conjunctiva; centrally the node shows polymorphs, and peripherally mononuclear cells with an occasional giant cell, but never have there been found tubercle bacilli. The end-result is resolution or necrosis. Corneal phlyctens are similar to conjunctival; but they run a less favourable course and frequently proceed to ulceration; these deep ulcers are most resistant to treatment and tend to become permanent.

The tuberculous phlycten appears first as a small greyish-white node, about the size of a pinhead, on the ocular conjunctiva, usually at the limbus. The node has a reaction halo round it in the form of serous exudation and hyperæmia. There is a varying amount of secondary conjunctivitis, with engorged blood-vessels. There may be an insignificant phlycten in the centre of a widespread conjunctivitis, or the phlycten may predominate and the conjunctivitis be of minor significance. Ulcers, if present, show sharply defined on the cornea, in size from a pinhead to a hemp seed. These phlyctens tend to disappear and then to reappear. with recurrent exacerbation of symptoms. The symptoms vary in the fresh forms where a pinhead phlycten is just visible, to severe cases in which there is engorgement of the surrounding conjunctival vessels, photophobia, blepharitis, redness and swelling of the lids, with or without lacrimation. In tuberculous phlyctens the skin test will always be positive, usually at the first test; if negative use Mantoux 1–100. If all tests are negative (the dilution being fresh and potent) and if the radiograph of the chest is also negative, then one must suspect some protein other than tuberculo- as causative agent. If the tuberculous phlycten is first seen when the lungs are healing or healed, the general health may be good. If, however, it is first observed when associated with an active primary lung lesion, the child may present a scrofulous appearance, with enlarged cervical glands, running nose, thickening of the lips, poor appetite, and sometimes slight fever. The most common age for the appearance of the phlycten is between 1 and 4 years; at this time phlyctens are difficult to treat, because small children rub their eyes, and resist local medication; for this reason hospitalization of these patients is essential for an early cure. Phlyctenular keratitis shows the

above symptoms at their very worst; there is a very strong tendency to exacerbation and recurrence, and non-healing cases suffer grave disability; the child with chronic corneal phlyctenular disease has very poor vision, cannot attend school, and grows up with few prospects of earning a livelihood. It is in these older and more stubborn cases that tuberculin therapy acts most satisfactorily. Small children who suffer from phlyctens occasionally develop blepharospasm of a severe type peculiar to tuberculous keratitis. The child buries his face in the cot or on the floor; he has to be forced to eat, which he will do with tightly closed eyes and flexed head; for weeks on end he will remain in this position; during that period mental development is arrested, although the child may thrive physically and gain weight. This condition may be avoided by early institutional treatment of the simple phlycten thus ensuring adequate local medication; attacks of blepharospasm when present may also be curtailed in the same way.

Example.—Raymond G., aged 2 years. History of a previous attack of photophobia and blepharospasm lasting 10 weeks. Admitted to hospital in 1937 with positive Hamburger; X rays showed a trace of primary infiltration remaining at right base, and enlarged right hilar and paratracheal glands. Bilateral phlyctenular conjunctivitis, photophobia, and blepharospasm. S.R. 30 mm. per hour. Scrofulous appearance, with thickened lips and running nose. Elder sister suffering from pulmonary tuberculosis. The blepharospasm was cured after 6 weeks' treatment in a semi-darkened solo-ward, and he left hospital after 3 months apparently cured. He returned 6 months later with a recurrence of the blepharospasm, which was treated by strapping the child into a sitting posture in his cot for part of the day, dark glasses, and determined introduction of medication between the closed lids; this attack was cured in 2 weeks, and he remained in hospital for some months' further treatment, open air, and rest. Later he returned to the extern department at the commencement of an attack, and this time his mother was induced to carry out the treatment at home with weekly hospital attendances, and the attack was aborted. The boy has had no attack since and the phlyctens have healed along with his pulmonary lesion.

Diagnosis.—This depends on the typical appearance of the nodes, with surrounding infiltration on the limbus, on the presence of a positive tuberculin reaction, and in the majority of cases on signs of an active or healed lung lesion seen radiologically. Certain cases with positive skin reaction show negative radiographs; this is so more often with a recurrent than a fresh phlycten, and especially when the condition is long-standing. Family and personal history are important.

Treatment of phlyctens is both local and general.

General treatment must be directed towards the healing of the primary lung lesion. Rest in bed in the early stages when the lesion is active is most necessary, with the addition of good food and fresh air. The operation for the removal of tonsils and adenoids should *not* be undertaken until it has been ascertained by X rays that there is no evidence of pulmonary tuberculous activity, and that the temperature and sedimentation rate are normal and the child not losing weight. Exposure to ultra-violet rays is contra-indicated in the presence of an active pulmonary lesion. For the treatment of blepharospasm firm handling is important ; the medication must reach the affected part, and its application must be regular and continuous. Dark glasses are most helpful and may be worn after 1½ years of age ; a green tennis shade may be worn if there is danger of breaking glasses by head-burying.

Local treatment must be continued over a considerable period of time, and should be resumed immediately on signs of recurrence. Medicaments must be properly applied, in view of tendency to relapse. For this reason calomel powder flicked on to the eyeball is in the early stages a more successful measure than the insertion of drops or ointment against a child's strenuous opposition. Secondary or associated conjunctivitis may be treated with 20 per cent argyrol. If corneal ulceration is present, or seems in danger of occurrence, it is necessary to employ atropine 1 per cent ; indeed it is advisable to dilate the pupil for some days in all cases that come under treatment ; nevertheless, early simple fresh phlyctens may be aborted and never recur if calomel treatment alone is given sufficiently promptly and regularly ; this should be done in a tuberculous child, even when a doubt exists as to whether the inflammation will develop into a true phlycten or not.

Tuberculin therapy in selected cases is of great value. Indications for its employment are (1) a healed lung lesion (healed for at least 3 years), (2) no other active tuberculosis in any other part of the body. It is used with the greatest advantage in the case of recalcitrant corneal phlycten, where the lung lesion is long since calcified, and where the eye disease is resistant to all forms of local treatment. It is obvious, therefore, that tuberculin therapy is contra-indicated in the case of young children, because at these early ages one cannot exclude the possibility of activity in the primary lung lesion.

Example.—Ellen C., aged 15. Girl was incapacitated by recurrent corneal phlyctenular disease which resisted all forms of continued local treatment. Percutaneous test positive. Radiograph of chest showed calcified Ghon, hilar and paratracheal gland (*Fig.* 54). History of "pneumonia" 8 years previously which lasted 6 months; tonsils removed soon after recovery from "pneumonia", and phlycten appeared a month later. After tuberculin injections for 6 months she recovered so well that she left off attending, against advice. A recurrence of blepharitis and conjunctivitis brought her back, and she got further injections for another year, with good result.

Method of administration of tuberculin: Before administering the first dose, test the sensitivity of the patient by giving a very weak initial hypodermic dose—for example, 0·01 c.c. of the emulsion. If this produces no reaction, then begin weekly therapeutic doses, subcutaneously; the first dose is 0·1 c.c. of bacillary emulsion, strength 1–100,000. Repeat the dose once a week, increasing it each time by 0·05 c.c.; thus the doses will be 0·1 c.c., 0·15 c.c., 0·2 c.c., and so on. A local reaction which lasts about two hours may occur about four hours after the injection; this is not abnormal, but usually absent. Should it, however, be accompanied by any general systemic disturbance such as vomiting, malaise, etc., it is wise the following week either to repeat or to reduce the dose. When 1 c.c. of the 1–100,000 solution has been reached (after 3 or 4 months), begin with the next bottle of 1–10,000 solution, and give 0·1 c.c. of this first, increasing weekly by 0·05 c.c. until 1 c.c. has been reached. If this treatment has to be repeated at a later date in the event of relapse or incomplete cure, begin with exactly the same dosage as was used the first time (0·1 c.c. of 1–100,000). Inunction of the skin with tuberculin ointment is also a good method of treatment, but the dosage cannot be regulated in so exact a manner as with the graduated tuberculin hypodermic syringe. In order that this treatment may produce the maximum of benefit, it must be given regularly once a week over a long period (sometimes for 1 to 2 years). If attendance is irregular relapses will occur.

2. TUBERCULOUS SKIN LESIONS

Tuberculous skin lesions in children may be classified according to the point of view of the observer. The dermatologist sees them in two main groups:—

1. Those conditions which are definitely tuberculous, both histologically and by the finding of the tubercle bacillus in the

lesion (either direct or by guinea-pig inoculation). These include: Miliary tuberculosis of the skin; Scrofuloderma; Lupus vulgaris.

2. Those conditions where the tubercle bacillus is rarely found in the lesion, and inoculation of tissues into a guinea-pig is negative, and where the histological picture, though not typical of tuberculosis, is suggestive of it, and the family and personal history suggests tuberculosis. Such cases are usually referred to as 'tuberculides', and include: Papular and nodular tuberculides; Lichen scrofulosorum; Boeck's sarcoid; Erythema induratum; Granuloma annulare; Erythema nodosum (tuberculous type).

In this chapter it is intended to dwell chiefly on those manifestations which belong to the second group, particularly the papular and nodular tuberculides; for these are the conditions which are associated with tuberculous lesions in children. Erythema nodosum, the most important of all, is discussed under PRIMARY TUBERCULOSIS, p. 49.

The recognition of the relationship between many of these skin lesions and pulmonary or glandular tuberculosis is of the utmost importance in order that the child may receive treatment which is suitable, not only to the skin, but also to the underlying pulmonary lesion. The tuberculin reaction in all these skin lesions will be found positive; the chest should be X-rayed in all such conditions so that suitable rest therapy may be given to active lesions, and such measures as generalized ultra-violet ray therapy not prescribed in the presence of pulmonary activity. Treatment of the underlying condition is most effective, whereas local treatment (with the exception of lupus vulgaris) confers little benefit. It is common experience that two different types of tuberculous skin lesions are often present in a case at the same time, again excepting lupus vulgaris.

CLASSIFICATION.—A simple classification for the clinician may be made according to the stage of tuberculous disease at which the various skin manifestations may appear:—

Primary Infection of the Skin: Extremely rare in children; it is discussed shortly in the chapter on ROUTES OF ENTRY, p. 19.

Coincident with Primary Lung Infection.—**Erythema nodosum** of the tuberculous type. Tubercle bacilli are never found in the nodes (*see* chapter on PRIMARY TUBERCULOSIS, p. 49).

Occurring during Late Primary or Secondary Stage of Hæmic Spread.—

Acute hæmorrhagic tuberculosis of the skin Acute miliary tuberculosis of the skin Scrofuloderma	True tuberculous processes
Tuberculides, papular and nodular Lichen scrofulosorum Boeck's sarcoid Erythema induratum	Tuberculides

Not Associated with any Stage in Particular.—Lupus vulgaris; either hæmic (endogenous) or by direct inoculation (exogenous).

Late Superinfection.—Verrucosa cutis (butcher's tubercle) is not seen in children.

Acute Hæmorrhagic Tuberculosis of the Skin.—This is a purpuric type of exanthem, with widespread distribution. It appears during the course of a primary lesion in a few isolated cases; it has no prognostic significance, and such cases may recover completely; the purpuric spots last for a few days and then fade. It also appears occasionally as a terminal phase of miliary tuberculosis of the lung, when the prognosis is hopeless. It is extremely uncommon.

Acute Miliary Tuberculosis of the Skin.—This condition is very rare; it occurs as part of a generalized miliary tuberculosis. The prognosis is bad.

Scrofuloderma.—This is due to secondary infection of the skin, usually by sinus spread from diseased bone or gland. The usual site in children is around the neck, associated with gross enlargement of the cervical glands. A node forms in the subcutaneous tissue, generally through a sinus which burrows its way from an infected gland; this node increases in size and rises to the surface, finally breaking through the skin and forming an abscess which has indolent edges and dirty floor. There is seldom true caseation, but a few tubercle bacilli can be demonstrated in the contents of the node. The process is painful, but less so than is a pyogenic abscess, and the child, although scrofulous, is not otherwise in poor health. These abscesses heal in time, slowly, and generally only after improvement of general condition and healing of the original focus.

Papular and Nodular Tuberculides.—This convenient name has been applied to various forms of nodes and papules which

appear in the subcutaneous tissue in tuberculous children; they usually appear during the second stage of hæmic metastases, and may be seen associated with enlarged mediastinal glands, tuberculous dactylitis, and also at times with lichen scrofulosorum. Histologically, the nodes show early tubercle formation with a few giant cells; tubercle bacilli are rarely found in them, and guinea-pig inoculation seldom yields a positive result. On account of the stage at which they appear, these nodes are more probably due to the circulation in the blood-stream of attenuated or dead tubercle bacilli than to an allergic tuberculo-protein reaction; the allergic phenomenon belong rather to the primary stage. But this point is still open to dispute. Some nodes necrose, whilst others form abscesses, suggesting in the latter case the presence of some few tubercle bacilli. The tuberculide nodes appear chiefly on the face and on the extremities; there may be two or three of the larger and more of the smaller variety. They appear as small round or oblong bluish-red nodes lying under the skin, in size from that of a small pin-head to a broad bean. The skin over the nodular tuberculide is tightly stretched, producing a typically glazed appearance. Some of the pustular types of tuberculide grow larger and break down, forming subcutaneous abscesses. The majority, however, of the very small papular-necrotic type fade away gradually, often lasting six to twelve months. These conditions are most usually seen during the first three years of life, but they may occur at any age up to puberty. It is important to recognize their appearance, for this may lead to a diagnosis of tuberculosis in some cases which would otherwise remain undetected. The condition is not uncommon in young children. Redeker mentions seeing 5 per cent to 10 per cent of such tuberculides amongst his tuberculous children. Price in 1938 found 2 to 4 per cent in tuberculous children under 5 years, but has seen less in recent years. Prognosis is more favourable than would be suggested by the general appearance of illness in such children prior to treatment, by the radiological pulmonary appearances, and by the fact that other hæmic lesions may be present at the same time as the skin lesions; prognosis is most favourable where the skin lesions are the only ones produced by hæmic spread.

Treatment must be guided by the radiological appearances; bed-rest for a prolonged period is essential to healing. Tuberculin therapy is contra-indicated, for papular and nodular tuberculides

occur in young children during the period of active primary tuberculosis. It must also be remembered that light treatment is contra-indicated; this is especially the case where other hæmic metastases are present in addition to the skin lesion.

Example.—Bridget D., aged 1 year. Enlarged paratracheal gland on right side. Tuberculin test positive. One nodular tuberculide on right calf and one on right hip; tuberculous infection of left metatarsal bone. Hospital treatment with foot in plaster for nine months effected a cure sufficient to allow child to return to a careful home, where a subsequent complete cure was effected.

Lichen Scrofulosorum.—This benign miliary-form involvement of the skin usually appears during a late phase of primary tuberculosis of the lung or in the second stage of dissemination when hæmic foci are apparent. In older children it is seen in association with bone and joint tuberculosis, the latter often with radiological appearances of enlargement of mediastinal glands; in younger children it is nearly always associated with such glandular enlargement and with other hæmic metastases of the more benign type, such as metacarpal and metatarsal tuberculosis, or with tuberculosis of the spinal vertebrae. The tubercle bacillus is rarely demonstrated in the lichen, which begins as single elements of reddish-brown spots the size of millet seeds, which meet together to form groups. The trunk, especially the lumbar region, is the favourite site. There is no itching and they do not affect the patient adversely. The condition is benign in older children, but the prognosis is not always so good in young children, where the general prognosis in tuberculosis is less favourable. The condition is not seen so commonly as are the papular and nodular tuberculides.

Example.—Bernard L., aged 1 year. Admitted to hospital with mediastinal gland tuberculosis, collapse of bodies of 6th and 7th cervical vertebræ, and positive tuberculin test. After a few days he became very unwell, temperature 101° F., and erythema nodosum appeared on his legs (post-primary erythema nodosum); one week after this he developed lichen scrofulosorum on his back and buttocks. Two months later he was very well again, although the lichen did not fade for some months. He died one year later, from an abscess in the trachea, in a surgical hospital where he was under treatment for spinal tuberculosis.

Boeck's Cutaneous Sarcoid.—This is a rare condition in children. When present it is generally associated with bone lesions, mainly a cystic condition involving small bones, such as phalanges and metatarsals; an eye lesion is also often

present. The sarcoid plaques may be single, but often they consist of small reddish-brown nodules arranged in groups which appear on the face, neck, and elbows. The condition remains for years and does not ulcerate (*see* p. 74).

Erythema Induratum (Bazin's Disease).—This condition will only be referred to in passing, for it is seen in adolescent and young adult females rather than in children. Hard indolent nodes appear on the calves, few in number and painless. The chest radiograph rarely reveals any active disease, although the tuberculin test is positive.

Lupus Vulgaris.—It is stated in text-books that 50 per cent of cases of lupus vulgaris start before the 12th year; if this is so, from the point of view of treatment it is important to recognize the lesion at an early stage. Those, however, who have to deal with childhood tuberculosis seldom report this early condition. The origin of the lesion may be exogenous, when entry is usually by direct inoculation at an orifice (a favourite site is the mucous membrane of the nostril); or they may be, and often are, endogenous in origin and due to hæmic spread from a lung focus. It rather commonly accompanies or follows scrofuloderma (chin-strap lupus). Beginning as a millet-seed nodule, in a few months fresh nodules appear; these are brown or yellow-brown, and do not disappear on pressure; under a glass slide they resemble apple jelly. The course of the disease is prolonged and there is no spontaneous cure.

Treatment in the past was generalized ultra-violet rays (if no active pulmonary lesion) and local application of the Finsen lamp. Recently good results have been obtained by large doses of calciferol.

Example.—Joan M., aged 9 years. Early lupus vulgaris of mucous membrane of left nostril. Radiograph of chest showed two calcified primary foci in right apex and a calcified gland in left upper hilar region, with some thickening of the interlobar fissure on the right. The appearances of the primary complex suggest that the lesion has been healed for some years. The lupus therefore may be either exogenous or endogenous, probably the latter.

3. TUBERCULOUS CERVICAL ADENITIS

By D. S. Price and H. F. MacAuley

Recognition of the various types of tuberculous adenitis is necessary from the point of view of diagnosis, prognosis, and treatment. The tuberculous involvement of cervical glands

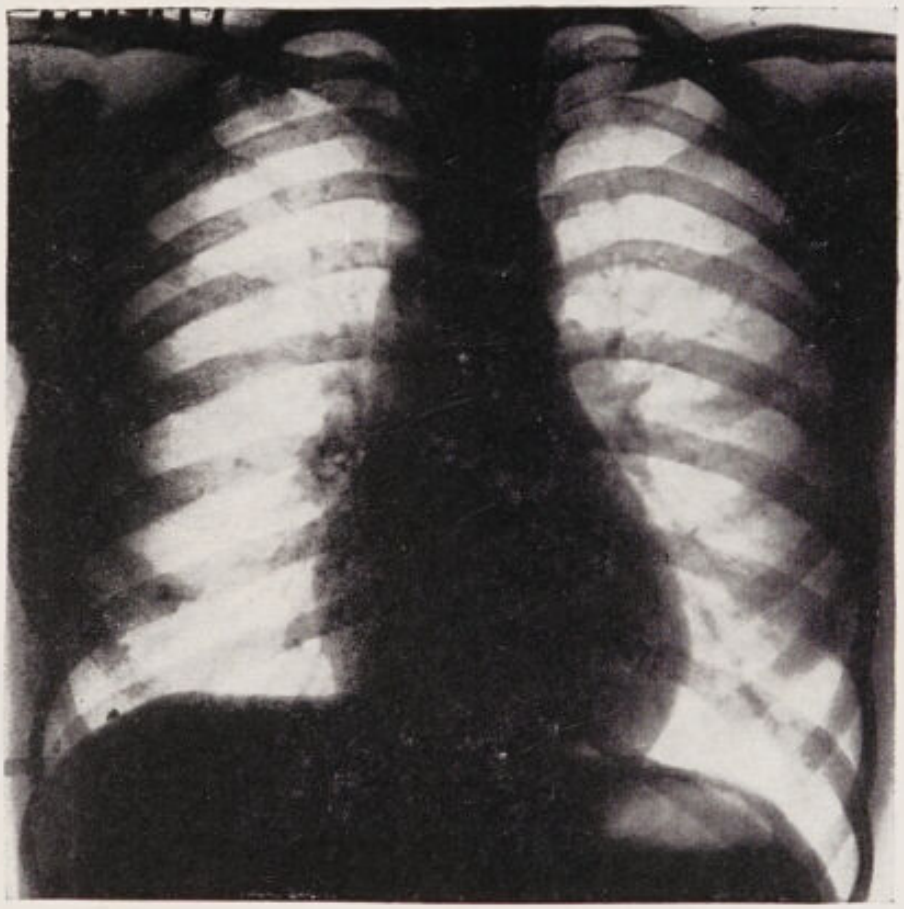
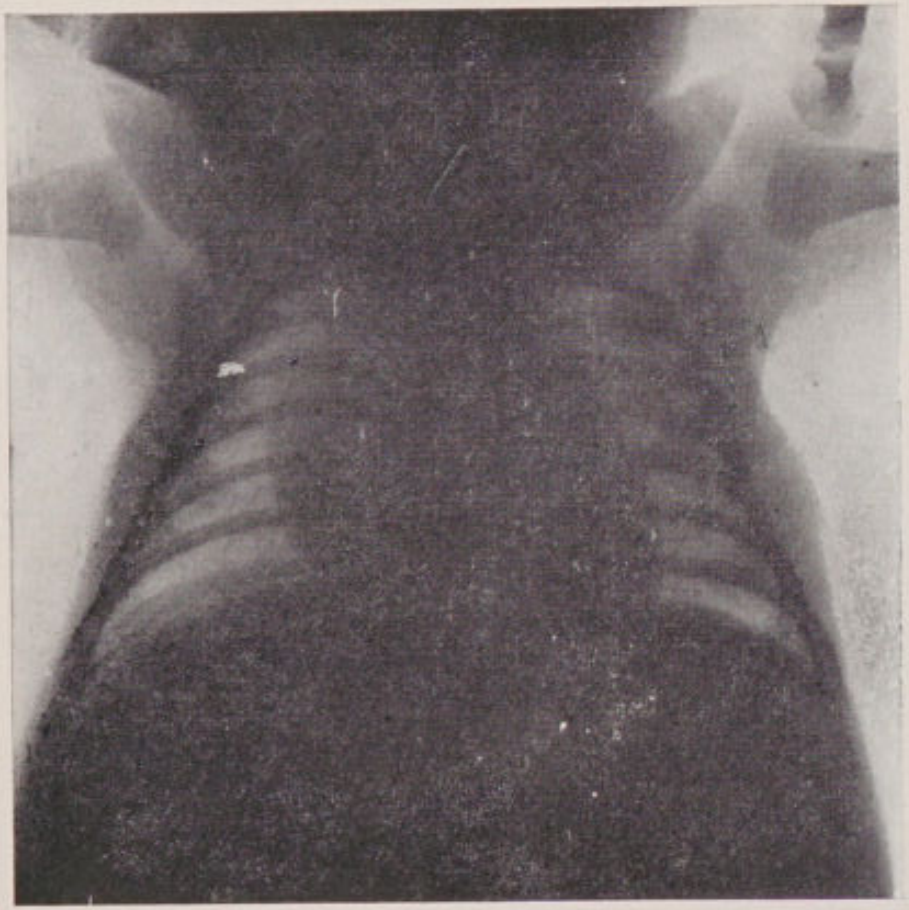


FIG. 54.—CHRONIC PHLYCTENULAR KERATITIS.

Ellen C. Aged 15 years. (*See Example*, p. 155.) There is a calcified primary focus at the intersection of the 6th and 8th ribs and calcified hilar and paratracheal glands on the right. Primary infection occurred 8 years previously.



FIGS. 55A-55C.—ENCAPSULATION OF PRIMARY FOCUS AND GLANDS.

Fig. 55A.—Tom L. May 24, 1941 ; aged 9 weeks. Admitted to hospital, mother phthisical. Moro test positive ; radiograph suggests enlarged mediastinal glands. T. 99° to 101° F.

June 12, 1941 ; aged 11 weeks. Mors from intercurrent infection.

(*See* Primary Focus, p. 47.)

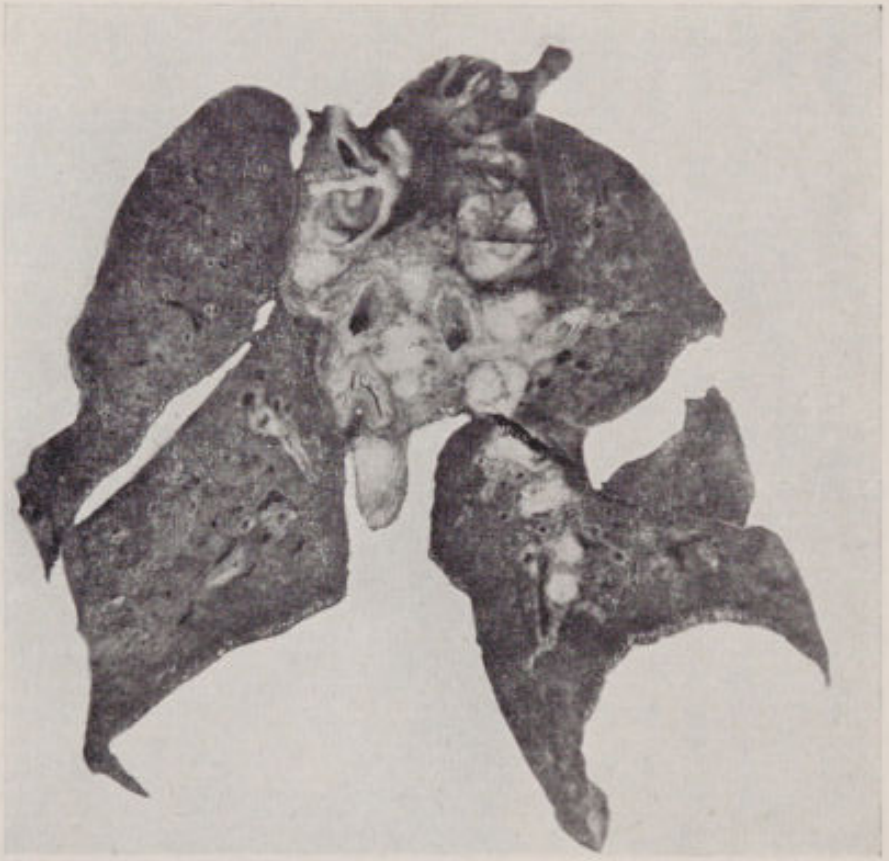


Fig. 55B.—Survey of lungs and hilum, showing massive caseation of hilar glands.
(*By courtesy of Dr. Walter Pagel.*)

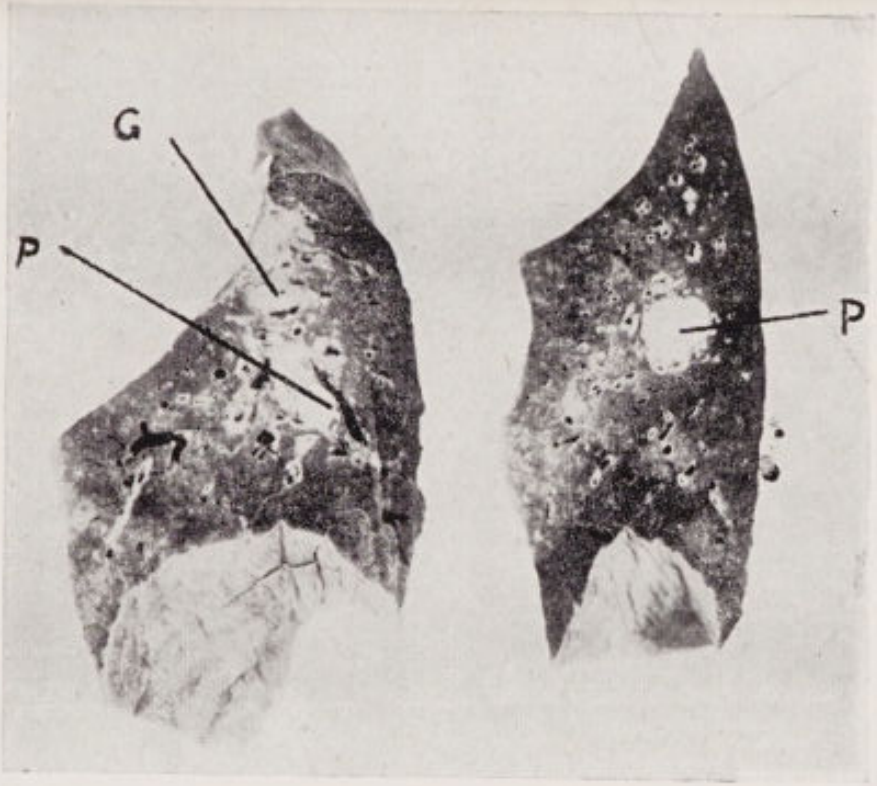


Fig. 55C.—Parrot-Ghon focus (P) in right lower lobe, and intrapulmonary lymph-glands (G).

Dr. Pagel's report on anatomical specimen: "Typical Parrot-Ghon focus in right lower lobe, with involvement of adjacent intrapulmonary and hilar glands. Histological examination shows only a beginning demarcation of glands and pulmonary focus; there is no proper capsule as yet, but there is proliferation of epithelioid cells in the periphery of the caseous masses."

Conclusions.—As the incubation period is 4 to 6 weeks, the focus and glands could only have been fully formed for 5 to 7 weeks. This was a heavy infection, and the building up of the capsule (which usually takes 7 to 10 weeks according to Pagel's investigations) had only just commenced. This case therefore exemplifies the need for prolonged rest therapy in the healing of the primary complex in infants.

(*By courtesy of Dr. Walter Pagel.*)

can be divided into three types, according to their mode of development. First, the *primary gland*, with primary focus in the oro-pharynx, usually the tonsil. Secondly, the *secondary hæmic gland*, where infection is carried direct to the neck glands from some other tuberculous focus in the body by the blood-stream. Thirdly, *allergic glandular enlargement* which occurs in certain cases of primary lung infection, giving rise to a temporary non-specific swelling of the neck glands.

1. **Primary Cervical Adenitis** occurs when the first lodgement of the tubercle bacillus in the child's body takes place in the buccal cavity. This buccal primary complex fulfils all the criteria of a pulmonary primary complex. The bacillus finds non-allergic tissue at the site of implantation, and after a short interval of time the regional lymph-gland becomes infected and enlarged, and the tuberculin test, hitherto negative, now becomes positive. In buccal cases, however, the primary focus is very insignificant; often it is not visible to the naked eye, but may be seen microscopically. Primary lodgement occurs most frequently in the tonsil, or in the nasopharynx, less often in the nose or round a decayed tooth. In the Lübeck disaster, when infants were infected by ingestion of virulent tubercle bacilli, the primary mouth focus was found to be situated most often in the pharyngeal tonsils or adenoids (due to recumbent position), next in the tonsil, and only once in the flesh around a tooth. After primary lodgement, the glands in the line of drainage commence, slowly and painlessly, to enlarge; next the deep cervical group, lying beneath the upper anterior border of the sternomastoid muscle, become involved by lymphatic spread. The glands on the opposite side may also become infected by cross lymphatic drainage, but the original group are more massively enlarged. Occasionally in certain cases there is spread via the lymphatics to involvement of supraclavicular and axillary glands. If the disease remains localized to the glands of the neck, as is nearly always the case, the condition is benign. Danger of dissemination arises remotely, mainly after inopportune operative interference. Cases of primary cervical adenitis are rather rare, but less so in some countries and districts than in others. In Dublin Price found 1·1 per cent cases of primary cervical gland tuberculosis amongst 450 tuberculous children. (For other figures, *see* p. 19.) The bovine type of bacillus is, without doubt, responsible for a great majority of the cases; the incidence

of the disease is low in countries and localities where bovine tuberculosis is rare, and higher in rural than in urban districts. The incidence of bovine type cervical gland tuberculosis is higher amongst infants than amongst older children (*Table XXI*).

Table XXI.—Preponderance of Bovine Type of Bacillus in Tuberculous Cervical Adenitis

Reporter	Area	Age Group	Cases	Bovine
Savage and Griffith	England	0–4 years	21	85·7 per cent
,, ,, ,,	,,	5–15 ,,	54	48·1 ,,
,, ,, ,,	Scotland	0–4 ,,	53	84·9 ,,
,, ,, ,,	,,	5–15 ,,	71	74·6 ,,
Jensen	Copenhagen	0–15 ,,	28	78·6 ,,
,,	Rural Denmark	0–15 ,,	28	85·7 ,,
O'Kelly	Ireland	0–15 ,,	7	2 cases
Mushatt	Eire	0–15 ,,	14	10 ,,

Clinical Picture.—The onset of tuberculous adenitis is gradual and comparatively painless. There may be slight fever during the early stages, but later the condition is afebrile. The tuberculin test is positive, and if known formerly to have been negative, the altered allergic response is diagnostic; it is necessary to follow a negative first test by Mantoux 1–100 before excluding tuberculous infection. The chest radiograph is negative when the cervical gland infection is primary; in this way primary can be distinguished from secondary glands in most cases. The affected glands will be either unilateral, or there may be some lesser degree of involvement on the opposite side, due to cross drainage.

Diagnosis is made by positive tuberculin test, absence of any septic focus in the mouth cavity, slow and painless onset, and absence of other tuberculous focus in the body, including negative chest radiograph. The glands when first seen may be at any stage of development, varying from a single hard node to bilateral caseous masses.

Examples.—

Liam G., aged 1 year 7 months. Fed on T.T. milk until 9 months old, then given non-T.T. unboiled milk. At 19 months he came to hospital with enlarged cervical glands, anterior and posterior deep group on the left, and anterior on the right; no sign of primary focus in the mouth. Hamburger positive, chest radiograph negative.

He was given deep X-ray therapy; the right glands subsided in a month's time, the left also subsided, but one broke down and took 2 months to heal. Cure was eventually complete. Pus from gland showed bovine type of bacillus on culture.

Brigid McS., aged 14 years, complained of "sore tongue". Tuberculin test positive, chest radiograph negative; no focus could be found in the mouth cavity. Fifteen months later she returned with enlargement of a gland in the left anterior deep cervical group. As this was single and discrete, it was removed by operation and proved to be tuberculous. Further search at this time revealed no sign of primary focus in buccal cavity, and chest was again radiologically negative.

2. Secondary Hæmic Glands in the cervical region become infected via the blood-stream from a primary focus or gland elsewhere, usually situated in the lung. The enlargement is often bilateral. The bacillus is often human type. This form of enlargement is mostly seen in young children, associated with other secondary tuberculous metastases common at that age, and with heavy infection of the mediastinal glands. The tuberculin test is positive, as is also the chest radiograph. No primary focus will be found in the mouth, but the tonsils are often enlarged and unhealthy, forming part of the picture of general ill health. The abdomen should be examined if there is no obvious pulmonary lesion. The importance of the recognition of the secondary nature of these glands must be emphasized, on account of the danger of meningitis or dissemination should operation be undertaken; tonsillectomy also should be avoided if possible in these cases.

Examples.—
Jimmy F. (*See* p. 91 and *Fig.* 42.)

James D., aged 2 years. Underwent tonsillectomy without leave during the course of primary pulmonary tuberculosis; an immediate flare-up of the hilar gland activity followed the tonsillectomy, and a hæmic infection of the cervical glands also occurred, resulting in a rather intractable tuberculous cervical adenitis, which did not heal for 2 years—that is to say, long after the mediastinal glands had been cured.

3. Allergic Involvement of the cervical glands may occur during the course of primary pulmonary tuberculosis. The adenitis is not specifically tuberculous; it is an allergic reaction to tuberculo-protein. It is always bilateral. It tends to appear in the exudative or scrofulous child—that is to say, the type that runs a high temperature for slight causes, has a very

active response to the tuberculin test, is inclined to bronchitis, eczema, and asthma, but in whom the tuberculous lesion may not be at all severe, in spite of the somewhat alarming illness picture. The swelling of the glands appears rather suddenly, and remains for only two or three weeks; slight fever is usually present at first, but as the glands recede it falls to normal; there is some degree of malaise associated with the temperature. These glands never harden, caseate, or break down. It is unnecessary to give any specific treatment for the glands, other than general treatment of rest and fresh air for the lung condition. If, however, they give rise to discomfort, mild heat applications may be employed. Price on occasion has seen such allergic glands appear at school age and in infants of one year amongst cases under treatment for primary pulmonary tuberculosis.

Generalized Glandular Tuberculosis.—In this rare condition there occurs simultaneous swelling of practically all palpable lymph-glands, probably tuberculous in nature; neck, axillæ, and groin glands are all involved, and may be associated with enlargement of the hilar glands. The condition is relatively benign. It may or may not be associated with a known tuberculous focus in the body. Sections of such glands removed during life have been found by MacAuley to show typical tuberculous giant-cell systems, and to have followed the usual pathological phenomena of tuberculous infection of the lymph-glands; it is, however, difficult to see any common aetiological basis.

Axillary Glands.—The primary complex may take place at any site where the tubercle bacillus gains entry into the body. This was illustrated by the events of the Ring disaster (1936). Following preventive inoculation against diphtheria at Ring College, Co. Waterford, 25 children, aged 10 to 14 years, developed primary tuberculous lesions at the site of inoculation in the deltoid muscle, with subsequent involvement of the axillary glands. From these primary upper-arm complexes, hæmic spread to other glands occurred in a few cases, namely to mesenteric and mediastinal glands, and in a few cases the supraclavicular and cervical glands were involved by lymphatic spread; in one case generalization occurred with fatal result.

Diagnosis of Tuberculous Cervical Adenitis.—The diagnosis is made on the history of prolonged and painless enlargement, positive tuberculin test, and absence of any obvious chronic

septic focus in the tonsils or teeth, of impetigo, or pediculi in the hair. There may be a history of recent change of milk supply. The temperature is normal except in the presence of abscess formation; there is but slight constitutional disturbance in most cases, and the glands are painless on palpation. The glands will present a varying picture according to the stage at which they are first seen; all tuberculous glands tend to coalesce, and may break down; fixation of the skin depends on the age of the lesion. Scrofuloderma in the surrounding skin is strong evidence in favour of tuberculosis. The chest film may be positive with secondary hæmic glands.

Differential Diagnosis.—

1. *Simple Adenitis*, due to oral sepsis, runs a more acute course, with fever, pain, and general illness picture. The glands usually recede, or they may point and discharge pus, within a week or two.

2. *Streptococcal Adenitis*, due to streptococcal tonsillitis, has a very acute onset, with sore throat and high temperature. The glands remain swollen for a long time and subsequently they present an appearance very similar to tuberculous glands, and their aetiology may only be recognized at operation. Here the history of acute onset, and the tuberculin test, if negative, will be useful in arriving at a correct diagnosis as to the nature of the lesion.

3. *Hodgkin's Disease* (*Lymphadenoma*) has a gradual onset, and glands in other parts of the body may be enlarged also. This disease is usually associated with splenic enlargement, and there is associated anæmia with increased eosinophil count, and often increased leucocyte count. The glands are discrete and the tuberculin reaction may be negative.

4. *Lymphatic Leukæmia* shows a picture of severe illness, often accompanied by hæmorrhages from gums, etc., with high fever and splenic enlargement. The hæmoglobin content is low, and the number of lymphocytes will usually exceed by far the lymphocyte figures found in tuberculosis.

5. *Lymphosarcoma—see* p. 74 and *Fig.* 37.

Treatment.—There has been much controversy about the treatment of tuberculous cervical adenitis. It is impossible, however, to decide on treatment until the case has been definitely labelled as one of primary or secondary involvement; when this has been achieved, which is reasonably possible in the vast majority of cases, the matter of treatment becomes rather

more simple and may be guided by the following rules. Active interference need be considered only in the case of primary infection of the glands.

Allergic glands require no treatment other than that given for the pulmonary condition.

Secondary glands, infected by hæmic spread from another focus, must be treated by conservative measures. Operation in these cases is attended by risk, and the occasional fatalities, such as tuberculous meningitis, following a block dissection provide warning against such interference.

Primary glands, due to mouth infection, may be treated by conservative methods, or by operation, or by a combination of the two.

Conservative Treatment.—This consists of open-air hospital treatment, continued over a period of months, with adequate rest, fresh air, and good food. Immobilization of the neck by splintage will hasten cure. In the presence of a negative chest radiograph, more active measures may be adopted ; for instance, light treatment directed on to the glands ; ultra-violet rays are more universally used, and are very safe as well as satisfactory ; roentgen rays assist in reducing the glands within a short time ; finally, local application of deep X rays provides the quickest cure of all, but it is not advocated except under expert control, as breakdown often occurs. Other therapeutic measures have been tried with varying degrees of success, and various chemicals have been introduced into the glands with good results. Tuberculin therapy has been tried. Some people prefer to do nothing except to place the child under ideal sun and rest conditions, and this works extremely well, but the cure will take some time.

Operative Treatment.—Operative interference is justified only in those cases where the tuberculous process is confined to certain groups of cervical lymph-glands ; thus it is necessary to make a full examination of the child and to X-ray the chest before considering operation. The indications for operation are : (1) If the glands persist after a reasonable period of conservative treatment ; (2) If the glands are in localized groups ; (3) If the general condition is satisfactory.

Time for operation by clean dissection : The glands should be removed when they are still fairly mobile and relatively hard. There must be no periadenitis and no subcutaneous abscess or fluctuation detectable in the glands ; they are preferably

removed long before the skin is involved. These conditions, however, are not always favourable, and therefore the types of operation will be enumerated: (1) Thorough dissection, in ideal cases as described above; (2) Aspiration, when the skin is uninvolved and there is fluctuating localized swelling; (3) Limited incision or excision of sinus, enlargement of fascial opening (collar-stud abscess), and evacuation of contents.

In the operation of thorough dissection, the glands should be dissected out cleanly through a transverse incision, after exposure of the internal jugular vein and the spinal accessory nerve. Bailey (1946) has demonstrated the excellent results obtainable by thorough dissection, followed by an occlusive dressing, in cases even when complicated by extensive skin involvement, and has shown that wide ablation of such skin produces relatively little disfigurement.

Dangers of operation: Apart from avoidable sequelæ, there is a certain risk of spread to the meninges; this is especially to be feared if a hæmic case is inadvertently submitted to operation; the diagnosis between primary lymphatic cases and secondary hæmic ones cannot always be clear, even when all possible clinical investigation is made previous to operation. Cases of miliary tuberculosis of the lungs resulting from operation have been reported.

Cosmetic Results.—The cosmetic results of conservative treatment are beyond question superior to operative results; scars and keloids are avoided, and the condition heals without any apparent residue. If, however, the glands break down spontaneously during the course of conservative treatment, the resulting scar will be more disfiguring than would be a neat operation scar. These questions have to be taken into consideration, but should not unduly influence any decision which has been arrived at by careful examination of the clinical condition and the diagnosis regarding the aetiology of the infection.

Tonsillectomy.—Before undertaking tonsillectomy in any case of tuberculous cervical adenitis, the whole picture of the child has to be considered. The question must be answered: Is this a primary or a secondary infection? Tonsillectomy in a primary infection is a comparatively safe procedure; but in these cases the tonsils seldom show macroscopic evidence of tuberculous infection, and hence tonsillectomy is seldom strongly indicated. It is the case with hæmic infection of cervical glands that presents difficulties; one may be faced with a

condition of tuberculous and mixed infection in tonsils demanding tonsillectomy whilst the pulmonary condition of the child provides a contra-indication to such interference on two grounds—the possibility of a flare-up in the primary complex or the precipitation of further tuberculous hæmic seedings, or both. Each case must be considered on its merits, and the urgency of the condition given due weight. In many instances, it is wise to delay until the lung lesion has become less active.

4. ABDOMINAL TUBERCULOSIS

By D. S. Price and H. F. MacAuley

The term 'abdominal tuberculosis' includes mesenteric gland tuberculosis and also tuberculous peritonitis. There are two types of mesenteric gland tuberculosis: (1) The *ingestion* type, which causes (*a*) tuberculous enteritis, leading to (*b*) tuberculous mesenteric adenitis; and (2) The *hæmic* type due to blood-stream spread from an already existing focus to the mesenteric glands. The three main types of tuberculous peritonitis are: (1) ascitic; (2) fibroid; and (3) fibro-caseous.

Peritoneal infections are due either to blood-stream spread from some other focus or gland, or to direct contact with a breaking-down caseous mesenteric gland. In the early case of abdominal tuberculosis the symptoms are slight and ill-defined, and clinical diagnosis must often be at best but a suspicion; in more advanced cases with mixed involvement the underlying pathological condition cannot be exactly known during life. The diagnosis of abdominal tuberculosis has many difficulties for the physician: bearing in mind, however, the aetiological factors, there will be considered here some practical considerations which may be helpful.

General Remarks.—Abdominal tuberculosis is a very serious condition in young children. The highest incidence is reported to occur in the 1–2 age period, that is, immediately after weaning, when large quantities of cow or goat milk are consumed. As is the case with all adenoid tissue, Peyer's patches are proportionately larger in the infant and young child, and primary entry through the bowel wall usually takes place at this site; the infant is prone to all forms of enteritis, which leaves this region vulnerable to lodgement of the tubercle bacillus; these are some of the factors which predispose the infant under 3 years to abdominal infection by the tubercle bacillus. The

incidence of the disease varies in different localities, low figures depending on a tubercle-free milk-supply. It is agreed that in England about 80 per cent of cases of abdominal tuberculosis are due to the bovine type of bacillus. In Denmark the figure is lower in cities and high in rural districts. The incidence of bovine infection is higher in young children. The human type of bacillus is responsible for abdominal cases due to hæmic spread from a pulmonary focus, or due to ingestion of floor dirt, or to contamination of cups, etc., by phthisical patients; the human type represent about 20 per cent of all childhood abdominal cases, and the majority of these are infected by hæmic spread. Many statistics derived from autopsy material have been published in connection with abdominal tuberculosis; the clinician should beware of drawing conclusions from these as to the actual prevalence of the disease. Figures, both anatomical and clinical, are given on p. 18.

With regard to terminology, in recent publications it has been suggested that the term 'tabes mesenterica' should be discarded. This is indeed necessary if we wish to have a clear picture of the conditions present in abdominal tuberculosis; the following classification is adopted here: (1) Mesenteric gland tuberculosis—ingestion and hæmic types; (2) Tuberculous peritonitis—ascitic, fibroid, and fibrocaseous forms.

1. Mesenteric Gland Tuberculosis

Primary (Ingestion) Mesenteric Gland Tuberculosis.—This lesion is caused by the ingestion of tubercle bacilli which may be contained in milk, less often in butter and cream, or in floor dirt. The bacilli lodge at the lower end of the ileum, the site of selection being Peyer's patches; they then penetrate the wall of the intestine, with, or more often without, macroscopic or microscopic ulcer or scar (Sheldon says that ulceration is present in one-fifth of cases). The bacilli, after penetrating the intestinal wall, infect the neighbouring lymph-node in the mesentery; thus is formed the *abdominal primary complex.* Such a child, having acquired no immunity from previous infection, proceeds in many cases to heavy infection of the glands, with caseation. The latter may involve neighbouring groups of glands by contact, and further groups such as the mesenteric root and pre-aortic glands by lymphatic drainage. In severe infections the pre-aortic group drain their infected contents into the cisterna chyli, whence bacilli may reach the

venous circulation and right heart, resulting in miliary dissemination and meningitis; the latter condition can also result at an earlier stage by the bursting of a caseous mesenteric gland into a branch of a mesenteric artery or vein. A certain small proportion of clinically 'occult' primary tuberculous cases have their primary infection hidden in a mesenteric gland. At times an ulcerating primary focus in the intestinal wall heals by the formation of scar tissue, with resultant cicatrization; should perforation of the intestine occur, which is very rare, adhesions shut off the area from the general peritoneal cavity. In advanced cases, caseous mesenteric glands may infect the peritoneum by contact; further, either by adhesions or by pressure of enlarged glands, complete or partial obstruction of the gut may arise; again, abscesses may form and point at the umbilicus or they may burst into the peritoneal cavity. When primary mesenteric gland tuberculosis involves the peritoneum in children under 4 years the result is often fatal.

Tuberculous mesenteric glands occurring at school age run a more benign course. The older child has much greater resistance than the infant, less milk is drunk in comparison to other diets at this age, and the intestinal tract is less susceptible to all microbic infections. The older the child, therefore, the better the prognosis; yet some cases at school age need strict treatment, and some indeed develop peritonitis. In many instances, however, infection in these glands remains latent, and is first recognized during the operation of appendicectomy, or is discovered in later life by chance, appearing as calcified glands in an abdominal radiograph.

Recently Engel, Stern, and Newns have pointed out the danger of tuberculous meningitis supervening at an early stage in primary mesenteric gland tuberculosis in children under 5 years. Out of 284 autopsies in tuberculous meningitis, they found that 14 per cent were due to primary abdominal glands. Of these 41 cases 68 per cent were aged 1–2 years; the authors stress the fact that a very small lesion in the abdomen can give rise to meningitis; in many cases only one enlarged and infected mesenteric gland was found, with no intestinal ulceration. This careful study gives valuable information; but once again the clinician is warned against establishing clinical prognosis on post-mortem statistics. A question of supreme importance is to know what proportion of early cases of abdominal tuberculosis develop tuberculous meningitis.

In Dublin, clinical experience shows that the infant with abdominal tuberculosis dies of his abdominal lesion in the vast majority of cases; a few exceptions are those which develop tuberculous meningitis as a terminal phase after prolonged illness due to the abdominal lesion.

In their investigation into the Lübeck disaster of 1930, Schuermann and Kleinschmidt have provided an exceptionally careful study of abdominal tuberculosis during the 0–1 age period; it must be remembered that in this case the infecting dose was abnormally massive and virulent, and the resultant lesions were widespread and somewhat atypical. Oral doses of what purported to be B.C.G. preventive vaccine were given to 251 newborns; through a mistake the infants got doses of virulent tubercle bacilli to swallow. A total of 77 died, of whom 71 showed primary abdominal lesions with generalization. In most cases the entry of the bacillus took place in the region of Peyer's patches; the glands most heavily involved were the regional mesenteric and ileocæcal. The infection spread thence by lymphatics to the pre-aortic groups and to other abdominal organs by contagion. In some few heavily infected cases a retrograde lymph-stream spread took place from the abdominal trunks to infect the lower anterior and posterior mediastinal groups.

Hæmic Infection of the Mesenteric Glands.—This occurs through blood-stream spread from an already existing tuberculous focus or gland; the latter is usually mediastinal, but may be a cervical or other primary gland. One of the Ring disaster cases, infected at the site of injection in the deltoid muscle, some months later developed mesenteric gland tuberculosis secondarily to a primary tuberculous axillary gland. One meets a certain number of hæmic mesenteric glands amongst cases under treatment for primary lung tuberculosis. In some the condition is benign, and the abdominal symptoms only of temporary duration; long before the lung lesion has healed, the abdominal symptoms have disappeared. In more marked cases, the abdominal symptoms last for 4 or 6 months; the glands in the right iliac fossa are tender and may be just palpable. Other cases show no regression of symptoms, but, with the lung lesion, they advance to a fatal termination. Here, again, the child's age plays an important role in prognosis; the younger the child the less good the outlook. Certainly the prognosis of a secondary abdominal lesion is not so serious

as that of a primary, and a great number of mild cases recover completely.

Examples.—

Elsie T., aged 15 months. In hospital for primary infiltration of right lower lobe. Some tenderness and palpable gland in right iliac fossa. Anorexia. After two months' open-air bed-rest all abdominal signs disappeared. Left hospital after 8 months' treatment for lungs and has remained well since.

James R., aged 3 years. Primary complex leading to caseous pneumonia with later cavitation and secondary abdominal tuberculosis ; died. (*See Fig.* 26.)

The question of abdominal infection from swallowed tuberculous sputum, as distinct from hæmic spread, must be considered. So far this has not been proved in infants ; the hæmic mode of spread is the more likely at this early age. In older children, however, who are suffering from phthisis, one might reasonably expect to find cases where secondary infection of the intestine is due to swallowed sputum, for in phthisis secondary hæmic lesions do not occur as a rule, whereas the constant passage of tuberculous sputum over certain areas in the intestine could give rise to ulceration.

Diagnosis of Mesenteric Gland Tuberculosis.—

Primary Ingestion Type.—

Positive test, up to 1–100 Mantoux, from negative.
Negative chest radiograph.
No other tuberculous lesion found.
Fever at commencement.
Tenderness in right iliac fossa (more or less).
Constipation or diarrhœa may be present.
Glands may be palpable in later stages.

Metastatic Hæmic Type.—

Positive test.
Other tuberculous focus found, e.g., chest radiograph.
Glands in right iliac fossa may be tender or palpable.

Diagnosis of either type of abdominal tuberculous glands is most difficult in the early stages. If a tuberculin test, known to be negative, becomes positive accompanied by fever, and the chest radiograph is completely negative, then one suspects primary abdominal tuberculous glands. One cannot be certain, although suspicions may be confirmed years later by seeing calcified glands radiologically. There may be some tenderness in the abdomen, particularly in the right iliac fossa ; but unless the glands are considerably enlarged they cannot be palpated.

Repeated examinations in the young child are essential; if the child is crying or if there is distension by flatus, the glands may be missed.

Symptoms of dyspepsia and vomiting may be present, but in infants the appetite is unimpaired. Older children show anorexia and may complain of pain and discomfort; many such suffer from constipation. If the disease is not arrested at this early stage, the glands soon become palpable, the abdomen is distended by flatus, and there is definite tenderness over the affected glands. Later there occurs wasting of the limbs, although the face may remain fat. In the early stages there is usually a rise of temperature, 99 to 101°F. which soon returns to normal and may remain so unless there occurs peritoneal involvement or abscess formation; with the latter, as with all forms of extensive caseation, there is high swinging temperature. In advanced disease diarrhœa is present in varying degrees, and is seldom absent in infants unless obstruction has arisen. Putrid, fœtid fatty stools are present when fat absorption is impeded by faulty drainage, as when the lacteals become blocked through back-pressure or by pressure from without of enlarged glands. Blood and tubercle bacilli are present in the fæces only when there is ulceration of the intestinal wall; this is of infrequent occurrence, and if tubercle bacilli are found in the fæces of young children, one cannot exclude the possibility that they may come from swallowed sputum and not from an abdominal lesion. The sedimentation rate is raised very high in exudative active lesions, less high in caseous lesions.

Differential Diagnosis.—

Appendicitis.—Differential diagnosis of acute and subacute and chronic appendicitis from mesenteric gland tuberculosis is often most difficult. The onset in the first is acute, and vomiting often present; the tuberculin test in uncomplicated appendicitis may be negative, but one cannot always wait to read the test; in many cases both conditions coincide. In doubtful cases it is safest to remove the appendix, and the tuberculous glands are rather improved by this operation; it should not, however, be undertaken lightly in the presence of an active primary lung tuberculosis with secondary hæmic involvement of the mesenteric glands.

Pyelitis.—In pyelitis pain is complained of on both sides of the lower abdomen; it is a frequent complaint with young children. The onset is rather acute. Tuberculin test if negative

will be helpful. Fever is present. Rely on urine examination.

Cœliac Disease.—Differential diagnosis here presents real difficulties, and may be confirmed only at autopsy. The tuberculin test (up to 1–100 Mantoux) if negative is the most important indication that the condition is cœliac and not tuberculous; as most of these cases occur in young children the picture is seldom complicated by a positive test due to some old healed focus. Examination of the stools in both conditions will show an excess of fat, especially neutral fats. Steen remarks that although the fæcal fat percentage is raised in all abdominal conditions in children, yet he finds that in cœliac disease the total output is higher. The skin over the abdomen feels thick in cœliac disease and is thin in tuberculous peritonitis. The small child suffering from abdominal tuberculosis is often very precocious and wise for his age.

2. Tuberculous Peritonitis

Tuberculous peritonitis may occur as part of a generalized miliary tuberculosis, when abdominal organs are also involved. This condition is seen in very young children, and the prognosis is hopeless; some cases are diagnosed only on the post-mortem table. The other types, the *ascitic* and the *plastic*, will now be considered.

The Ascitic Type of tuberculous peritonitis is most usually seen during school age, rather than in infancy; it follows to some extent the age incidence of pleural effusion. The exudation arises from the formation of a few small or large tubercles on the peritoneum, tubercle bacilli having been carried there by the blood-stream from an already existing tuberculous focus, usually in mesenteric glands. The peritoneal fibrinous or serofibrinous exudation has a specific gravity of 1020–1026, and albumin content of 4·5–6·5 per cent; it may be of large or small dimensions. Large exudates are easy to determine by percussion, palpation, and examination for thrill. Small quantities of fluid are much more difficult to detect. The abdomen is tender and resistant, and has a doughy feel. The temperature is raised somewhat above normal; appetite is poor; weight is lost, or if maintained this may be due to the increasing volume of fluid; the child experiences considerable abdominal discomfort, and has increasing embarrassment as the fluid increases in amount. The condition is generally benign; in many cases the fluid is absorbed either with or

without tapping. Certain cases, however, after the fluid has absorbed, pass on to the plastic type of peritonitis.

Plastic, Adhesive, Fibroid, and Fibro-caseous Types of tuberculous peritonitis are more frequently seen in young children and infants than is the ascitic type. The prognosis is very bad. This condition may follow an ascites which has absorbed, or there may be no preceding ascites. Small nodules appear scattered widely over the peritoneum, due to blood-stream spread from some other focus ; in some cases there may be peritonitis due to breaking down of mesenteric glands ; this may be localized or widespread according to the degree of caseation and abscess formation present in the glands involved. Over the whole abdomen are felt hard knobs, the intestines are matted together, and the omentum may be rolled up into the epigastrium where it may resemble an intussusception. An umbilical fistula may be present. The child is very ill, and in great pain ; there is a high swinging temperature ; obstruction, partial or complete, may occur, from adhesions which may cause constriction of intestinal lumen or kinking or matting of intestinal loops.

Examples.—

Lionel P., aged 7 months. Always fed on milk of one T.T. cow. Child was precocious and able to crawl on floor. Abdomen hard, knobbly, and retracted ; partial obstruction. Died 1 month later, and autopsy showed extensive adhesive tuberculous peritonitis and caseous mesenteric glands. Lungs free from tubercle. The mother had a phthisical cavity, and the child had evidently swallowed contaminated floor dust, and developed primary mesenteric gland tuberculosis and later plastic peritonitis from these glands.

Sean C., aged 18 months. Mantoux 1–100 positive (percutaneous negative). Masses of glands felt in lower abdomen, especially on right ; wasting, anorexia, temperature 98°–102° F. S.R. 29. Abscess pointing at umbilicus, but did not burst. Died 3 months later. Autopsy : All abdominal glands were caseous, most pronounced in right iliac fossa. Intestines matted together and bound to peritoneum by adhesions. Glands on pleural surface of diaphragm and right tracheo-bronchial glands were involved.

Treatment in Abdominal Tuberculosis

Conservative.—

1. *Rest.*—Absolute prolonged bed-rest. Fresh air. Warmth to extremities. *Diet.*—Milk and later meat and fish pounded. Low starch. Fats in moderation. *Medicine.*—Iron, cod liver oil in moderation. Ostelin for its vitamin D. Salol for offensive stools. Bismuth for diarrhœa. Vitamin C.

2. *Sunlight.*—Expose legs and arms first, and only later the abdomen, commencing with short exposures (Rollier). Ultra-violet rays are not nearly so beneficial as natural sunlight; again expose limbs first, and abdomen later for short intervals at first; at no time should the skin be reddened.

3. *Tuberculin.*—Advocated by some authorities.

Operative.—

1. *Paracentesis.*—For large collections of fluid. The results are not so good as by laparotomy.

2. *Laparotomy.*—The fluid is evacuated and the abdomen closed again.

Try conservative measures first and operate only in mild cases which are not improving, provided that the lungs are not involved. Obstruction must be dealt with surgically.

5. RENAL TUBERCULOSIS

By D. S. Price and H. F. MacAuley

Tuberculous infection of the kidney in children occurs as two distinct conditions: (1) Acute or chronic generalized miliary disease, involving the kidney; (2) Chronic caseous tuberculosis of the kidney.

1. The Acute or Chronic Miliary Type occurs as a bilateral infection of the kidneys and is associated with generalization of the disease from a pulmonary focus; at post-mortem examination a proportion of cases of miliary tuberculosis show miliary tubercles in the kidneys. Addison reported miliary involvement of the kidneys in 9 out of 14 children who died of tuberculous meningitis. Price found amongst young children that the larger proportion of miliary and meningitis cases did not show infection of the kidney, but more usually of the spleen. A few cases of chronic miliary tuberculosis of the lungs have been described where healing has taken place, and after a lapse of years these cases have yielded radiological evidence of calcified miliary foci in the spleen and, very rarely, in the kidneys.

2. Chronic Caseous Tuberculosis of the Kidney is a rare disease in childhood; nevertheless it represents a condition which demands early diagnosis if cure is to be attempted. The bacilli are carried to the kidney, by the blood-stream, from a primary pulmonary focus (possibly from an abdominal gland in a few cases); lodgement usually occurs as a single focus in the

medulla or papillæ of one kidney. From this focus further infection of the kidney takes place either by direct spread or via the blood-stream or the lymphatics. The focus in the kidney during caseation may rupture into the kidney pelvis, and then the urinary tract will become infected secondarily. Some authorities consider that it is more usual for the kidney capsule to become involved. The infection of a second kidney occurs later either by the blood-stream or by ascending infection from the bladder. Utsvedt (1947) has shown in adults that renal tuberculosis appears many years after primary infection. In many bone and joint cases suspicious signs and symptoms are found to be due to renal calculi, which tend to form during long periods of recumbency, but the possibility of a coinciding hæmic metastasis in the kidney has to be excluded. Harris, in the course of routine examination of urine from 67 children suffering from bone and joint tuberculosis, found that 13·8 per cent showed tubercle bacilli in the urine. Addison, amongst 10 children with tuberculosis of the kidney, found that 7 had other lesions; in only 4 cases was increased frequency of micturition noticed. Tuberculous bacilluria has been reported without disease of the kidneys. Redeker in the course of examining 3000 tuberculous children found only 3 cases of tuberculous kidney; 2 of these had advanced phthisis and 1 bilateral renal tuberculosis. Price, amongst 500 tuberculous children, found only 1 case of renal tuberculosis; this boy, aged 7 years, had bilateral involvement, and the pulmonary radiograph showed a calcifying primary focus in the left upper lobe. The prognosis of renal tuberculosis in childhood is extremely bad. Fortunately the disease is of rare occurrence. In Eire in 1935 no deaths were recorded under 15 years from genito-urinary tuberculosis, and again in 1944 no such deaths were registered.

Signs and Symptoms.—In the early stages of the disease, signs and symptoms are negligible. Indeed, early diagnosis can be made only by routine examination of the urine in all tuberculous children. The first symptom to appear is pyuria, which persists; the condition is perhaps one of frequent and hurried micturition rather than of actual increase in the amount of urine passed. Frequency is more marked at night than by day, but later it is both diurnal and nocturnal. There may or may not be hæmaturia. Pain is not complained of; if present, it should arouse suspicions of stone, especially if the child has

long been recumbent. There is usually no rise of temperature in the early stages of renal tuberculosis; during periods of active spread, however, the temperature may rise to about 100°F. The urine, if examined at an early stage, should arouse suspicion if albumin and pus, with no coliform bacilli, are found in acid urine; these findings are especially to be noticed if the child is suffering from a known tuberculous focus in the lungs. All cases with acid urine, showing pus cells and no organisms, should be further examined for the presence of acid-fast bacilli in the centrifuged deposit, both direct and by guinea-pig inoculation. Hæmaturia is only occasionally present; it renders the demonstration of the tubercle bacilli rather more difficult. A few red cells may, however, be found in the urinary sediment on occasion.

Diagnosis.—Diagnosis can be made only after careful consideration of the case. Positive tuberculin test (two strengths up to Mantoux 1–100), evidence of an active or calcified primary focus in the lung, secondary blood-stream metastases in bone or joint—all these increase the possibility of a tuberculous infection in the kidney. The demonstration of tubercle bacilli in the urine will render the diagnosis certain. As already mentioned, suspicion is first aroused by the presence of pus in acid urine in the absence of *B. coli.* A straight radiograph of the kidney region should be taken first as a routine, although calcification in the kidney of a child is seldom seen. Intravenous pyelography will provide information as to the functioning capabilities of either kidney, and will occasionally exhibit a lesion by showing deformities of the calices. Cystoscopic examination with ureteric catheterization, which in males must be performed under anæsthesia, will in many cases help to localize the focus and find which kidney is at fault.

Differential Diagnosis.—

Pyelitis due to B. coli: In this condition there will be pain on micturition, rise of temperature (at times reaching 104°F.), general constitutional disturbance, and crying and bad temper on the part of a small patient. The finding of *B. coli* in the urine is diagnostic. It must be remembered that it is possible for a tuberculous kidney to become infected by *B. coli.*

Calculus: If a stone is present in the kidney or bladder, there will be pain and hæmaturia, associated with frequency of micturition. It is important to get a straight radiograph.

Treatment.—

Operative: This procedure may be undertaken in unilateral cases provided the other kidney is uninvolved; where there is no active lung lesion; and when, if other secondary involvement in bone or joint is present, these conditions permit of it. There is a distinct risk of the development of generalized miliary tuberculosis and of meningitis after such renal operations. Sanatorium treatment after operation is most necessary.

Conservative: In bilateral infections, or when operation is contra-indicated by the pulmonary or general condition, conservative measures must be tried. Sanatorium or open-air surgical hospital is indicated, with complete bed-rest, open air, and good food. Tuberculin therapy should be tried in all cases where there is no coexistent open lung lesion; the doses used must be very small (*see* TUBERCULOUS EYE CONDITIONS, p. 154).

CHAPTER XIII

TUBERCULOUS LESIONS OF THE BONES AND JOINTS

By H. F. MacAuley

In the earlier portion of this book the mode of entrance of the bacillus, the tissue reactions, and the vicissitudes of both the patient and the bacillus have been described. Of the secondary manifestations, by far the most frequent, other than intra-thoracic lesions, are those caused by involvement of articular structures by hæmatogenous spread—non-articular bone manifestations occur with much less frequency.

Apart from the general factors, such as debility following the exanthemata, which contribute to the dissemination of the bacillus, there is only one known definitely localizing factor and that is trauma. There is practically always a history of preceding trauma in a case, but there is scarcely ever any objective sign of such injury. The injury must, therefore, be of a minor nature; in fact, the presence of any prominent traumatic lesion practically excludes the diagnosis of a tuberculous lesion.

As the relationship of injury to such a lesion is often a question of considerable medico-legal importance, certain postulates are suggested before accepting it in any particular case. They concern the nature of the injury, the interval before symptoms arise, and the extent of the reaction. As we have seen, the injury is always of a minor nature. The free interval is variable, but may be accepted as never less than three weeks. The reaction of the tissues should correspond clinically and radiographically to the stage of evolution one would have expected the lesion to have reached in the period suggested.

Evolution of the Tuberculous Lesion.—This lesion runs a very steady and uniform dehiscence, a point which is significantly emphasized by Calvé. A very large percentage of cases even with adequate treatment run this prolonged but nevertheless entirely satisfactory course. A minority, possibly on

account of huge initial dosage or poor tissue response, but more frequently because of unsuitable or inadequate treatment, are exceptional and deviate from the usual benign course.

It is most important that this normal trend to recovery be recognized, as it is the theoretical basis on which the modern treatment of these maladies should be based.

Treatment in open-air hospitals is a method not intrinsically based on any specific properties of fresh air or sunshine *per se*, but merely depends on the utilization of these agencies to favour and augment the general health of the patient during the prolonged period of immobilization necessary.

In the course of this evolutionary process it is possible to see three distinct phases clearly defined: a period of active dehiscence; a period of removal of diseased tissue; and a period of resolution and recalcification of the affected tissue.

General Pathology of the Lesions.—The bacilli reach the bone or joint involved via the blood-stream from a pre-existing focus, whether such is demonstrable or not. Frequently this mode of invasion is made evident radiographically by the demonstration of wedge-shaped sequestra with the base of the wedge towards the articular end of the bone, or occasionally by complete necrosis of a bone, as may occur in the astragalus, by blockage of the main arterial supply. However, much more commonly, instead of a single large secondary focus, there are multiple scattered smaller foci in the affected area.

While the bone or joint involved is usually a solitary one, occasionally additional bone or joint lesions exist. In fact this is a common occurrence in lesions of the metacarpus, metatarsus, and phalanges, particularly in small infants, in which these latter lesions are especially common.

There is one form of polyarticular arthritis described by Poncet and vigorously discussed from time to time, which has been attributed to tuberculous involvement. As these particular lesions do not ever suppurate they, no doubt, are now to be classed as allergic phenomena.

The more usual lesions due to actual invasion by the tubercle bacilli follow the typical course of tubercle formation, coalescence of minute foci, with destruction of the invaded tissue by disintegration, occasionally formation of small sequestra, undermining and exfoliation of articular cartilage, and joint effusion, often becoming 'purulent'. Occasionally extrusion of the 'pus' from the bone or joint occurs and 'cold abscess' formation

follows, which latter may proceed by penetration of the skin, commonly from ingress of secondary pyogenic organisms, to sinus formation.

Diagnosis.—The initial search in any suspected case must be for the primary focus. The value of the various tests and the methods of demonstrating the primary focus have been fully described in the earlier part of this book.

The demonstration that the particular bone or joint focus is tuberculous is often a matter of considerable difficulty, and in a large number of cases, especially in children, it rests more on deductive reasoning than on actual proof.

Proof that a particular lesion is tuberculous depends on the actual demonstration of tubercle bacilli in the lesion. Where an associated cold abscess is present the bacilli may be demonstrated in the 'pus', but apart from this, such manifest proof is usually absent, and it is perfectly true that a certain percentage (not a very large one) of cases have undergone prolonged treatment owing to an error in diagnosis. In order to put diagnosis on more secure grounds than purely clinical and radiological data, biopsy has had a certain number of supporters.

As regards biopsy on the actual lesion, e.g. joint, it is not to be countenanced in the case of children. The intervention itself is not without danger; the resultant findings, if negative, merely exclude the presence of tubercle bacilli in the actual portion of tissue removed; and after a period of some months at longest in the open-air hospital the diagnosis will be proven or disproven anyway.

Recently it has been suggested to do a biopsy on the draining lymph-glands (Seddon). It is difficult to see why a lymph-gland should be involved in an early lesion. However, as the gland may be removed without difficulty or danger especially from the inguinal group in case of knee-joint lesions, or external iliac group in case of hip lesions—which particular lesions probably lead to the greatest percentage of errors in diagnosis—the minor operation would be well worth doing if it succeeded even in reducing this percentage.

Of course apart from tests of positive value, it is often necessary to exclude other lesions such as low-grade pyogenic infections, congenital syphilitic lesions, etc., by their appropriate laboratory tests.

The value of radiographic examination is, in the extra-articular lesions, of very great importance if closely correlated with the

clinical findings, but the latter usually considerably ante-date the X-ray evidence in intra-articular lesions.

Prognosis in General.—

Prognosis as Regards Life.—As the bone and joint lesions are of themselves of a benign nature unless some complicating factor is introduced, they have no mortality. The danger to life may come either from the primary lesion itself, or from the development of additional new foci, most commonly meningeal.

Dangerous complications associated with the bone or joint lesion are the introduction of secondary infection, leading to prolonged discharging sinuses, and ultimately occasionally to amyloid degeneration, or again calculus formation occurring during prolonged recumbency.

The former is to be prevented by rigid asepsis in the treatment of cold abscess, and particular avoidance as far as possible of all operative procedures within the joint before the stage of regeneration has been reached.

Calculus formation is combated by liberal fluid intake, and by obtaining as free drainage of the kidney pelvis as possible by gravitation. Stone formation mainly occurs in lesions of the hip and spine where patients are kept horizontal on frames for a prolonged period. In hip cases the upper end of the frame should be kept tilted on blocks and the upper end of the cot also tilted up. This is easily done without detriment to the actual joint immobilization in the appliance commonly used, Jones's abduction frame. In spine lesions, as soon as the very active manifestations of the lesion have subsided, the patient should be nursed in the prone position with the thorax and legs tilted backwards.

Prognosis in Regard to the Joint Lesion.—With exceedingly rare exceptions, loss of movement is the price of cure in every established case of joint tuberculosis, and often, indeed, complete loss of movement is the price of complete cure. In the knee-joint especially some minor degree of movement often leads to repeated reactivation of the lesion, and ultimately calls for operative intervention when the child is old enough, twelve or thirteen years of age, to secure complete ankylosis. The position in which the joint is immobilized during treatment is always chosen so that this loss of movement will cause the minimal physical handicap.

Again in every case, other than when exceptionally osseous ankylosis is present, supervision must be carefully maintained

periodically for at least five years, as a joint in good position on the discharge of a patient may gradually lapse into a deformed attitude.

For such cases as come with established deformity these attitudes must be corrected promptly if they are not long present, while old-established deformity will require operative correction.

The duration of treatment naturally varies with the individual case, but in many of the major joints or spine at least two years must elapse before ambulation is resumed. And in lower limb lesions some protective appliance must be used for a considerable period subsequently.

Treatment: General Considerations.—The much discussed question of radical as opposed to conservative treatment, at least in regard to its application to children, may now be taken to be settled. Early radical operation, in the light of the specific evolution of the disease, is based on wrong principles, and experience has shown that it is both impracticable and unsuccessful. Sound conservatism is the only sound basis of treatment, though operative intervention may at times be required as an adjuvant method subserving some special mechanical or physiological consideration, but never supplanting the conservative method.

Any operative intervention must be carefully chosen and take place in that stage of evolution when the reparative phase has been definitely installed, and never in the early or destructive period.

The general lines of treatment can most suitably be considered from certain general principles :—

1. The bone or joint lesion being a secondary manifestation, its treatment necessarily demands the coincident treatment of the primary lesion.

In the majority of instances the primary lesion requires prolonged rest under the best hygienic conditions. Such treatment with heliotherapy is best obtained in open air hospital schools.

If, however, the pulmonary lesion is an open infective one, appropriate orthopædic treatment of the secondary lesion should naturally be carried out in a sanatorium.

2. The orthopædic lesion, whatever its site, requires immobilization and possibly traction during the whole of its period of activity.

3. When under these conditions this lesion is proceeding progressively to cure as shown by general signs of good health, local clinical signs of subsidence of pain, tenderness, swelling,

and spasm, with X-ray evidence of clarification of bone outline progressing to sclerosis, a trial period of at least 6 weeks' freedom from splintage and traction should be given while the patient is still in bed, if the lesion is of the spine or lower limbs.

4. If during this period no sign of activity reappears, such as deformity of a joint, an ambulatory appliance is fitted. The patient may then be discharged from hospital, but periodic inspection must be carried out subsequently over a period of years. Signs of reactivation during this probationary period will require either further immobilization or possibly some operative intervention, the latter usually only in the case of hip or knee lesions which repeatedly relapse, and in an occasional spine lesion, and all only in later childhood.

It is to be noted that before any operative intervention there should not be any discharging sinuses, or clinical abscess formation.

The aim of operative intervention is to aid and promote natural reparative processes along physiological lines rather than to remove diseased tissue. The focal lesion is as little interfered with as is compatible with the proper carrying out of the physiological and mechanical desiderata. For this reason the operation should be an extra- or para-articular one, rather than an intra-articular one. These methods when suitably applied should promote a sound osseous ankylosis, in place of a pre-existing fibrous ankylosis. In practice this usually means that a bone-graft is placed close to the joint joining the bone above to the one below the joint.

In bone lesions—i.e., a tuberculous osteomyelitis or periostitis—only under exceptional circumstances should the conservative method of immobiliizaton be departed from. If, however, the bone focus has formed a subcutaneous abscess or sinus and the general condition is satisfactory, a minor operative procedure to remove debris and stimulate healing is indicated. Also in the case of a bone focus close to a joint, as in the neck of the femur or tibial metaphysis, when the process is radiographically localized, operation should be undertaken in the hope of saving the joint, or limiting its involvement and shortening the period of treatment; otherwise these lesions tend to run an extremely long course.

Treatment of Common Complications.—The common complications encountered are abscess formation, sinus formation, and tuberculous meningitis.

Abscess Formation.—This occurs at two very different periods : (*a*) During the phase of activity, early abscess formation ; and (*b*) Late, when the activity has subsided and a mild form of abscess appears.

a. Early abscess : This presents the familiar features of a fluctuant collection of ' pus ' without any attendant sign of inflammation other than swelling. In this ' pus ' no organisms except tubercle bacilli are demonstrable ; even then they are demonstrated only by careful and prolonged search in any large percentage of cases. In spite of its other chronic features, it is remarkable that the swelling often becomes clinically evident quite suddenly (though many abscesses demonstrable by radiographic examination in a deep-seated focus never become clinically demonstrable at all). Presumably the explanation of its sudden obtrusion is the escape of tensely confined ' pus ' within a joint or similar lesion from within the capsule and its rapid expansion in the more capacious fascial spaces.

Frequently this escape of confined ' pus ' is accompanied by relief of clinical symptoms, mainly the disappearance of a slight rise in the temperature chart, and relief of pain.

Such a swelling must be very carefully watched from day to day (facility for this inspection is one of the advantages of the skeleton types of splints over plaster-of-Paris fixation), and more active treatment initiated only when the swelling is well circumscribed and relatively tense, but long before the overlying skin has become thinned and reddened. At this well chosen period aspiration should be carried out. A special syringe with a wide-bore needle having terminal and lateral openings and with a well-fitting stylet of the type of Gauvain's should be used. The skin should be adequately prepared and the main operation carried out with rigid aseptic precautions. Local anæsthesia by freezing with local ethyl chloride spray is sufficient. The skin puncture should be made outside the periphery of the swelling and the needle thrust obliquely into the abscess, thus preserving a good extent of healthy tissue between the skin puncture site and the actual abscess cavity.

When the contents are evacuated the puncture mark is sealed and a pad with pressure applied. This procedure is repeated, if necessary, at intervals of a week or longer until the abscess cavity is obliterated.

A blunt probe is used, if the needle after insertion is clogged with caseous matter, to clear the needle lumen with the needle

in place—the proper calibre probe is supplied in the equipment with the syringe.

Occasionally the contents are too thick to be successfully aspirated or the site too dangerous for blind puncture; it is then advisable to empty the abscess by incision, followed by careful suture of the subcutaneous tissue and fascia. The use of modifying fluids setting up a graded inflammatory reaction with liquefaction of the thicker masses is popular in France, but the difficulty of grading the reaction makes the method of incision preferable.

b. Late or residual abscess: This remains indolent for considerable periods, and usually clears up after one or two aspirations.

Sinus Formation.—When sinuses are formed early in the evolution of the lesion, they are usually troublesome and long protracted. It is therefore advisable to temporize with repeated aspirations of the preceding abscess as long as possible, as in this way the reparative power of the tissues is allowed to get under way and even if a sinus does occur, if treated aseptically, it heals up firmly and solidly and does not protract the treatment necessary for the uncomplicated lesion. It is, however, essential not only to take great care to prevent superadded secondary infections but also to continue without relaxation the primary treatment of the original lesion.

Infected sinuses, the result of injudicious operative intervention, or improper treatment of abscesses, through which secondary infection has reached the deep-seated lesion, are extremely difficult to get healed, and occasionally persist long enough to cause amyloid degeneration.

Tuberculous Meningitis.—Tuberculous meningitis is the commonest cause of death in tuberculous lesions of the bones and joints. It of course heralds a fatal prognosis. However, its diagnosis should always be fortified by laboratory examination of the cerebrospinal fluid, in the hope—sometimes not vain—that the causal organism is other than the tubercle bacillus, and the meningitis capable of treatment and recovery.

General Management of Cases in Orthopædic Hospital.—1. General examination, clinical, radiographic, and laboratory, to establish with exactitude the nature of the lesion of which complaint is made. Apart from investigations to establish positively the lesion as tuberculous, such as the demonstration of a primary focus, tuberculin tests, etc., other lesions are to

be excluded such as low-grade pyogenic lesions by blood-counts, etc., and syphilitic lesions by the Wassermann reaction.

2. On admission to the hospital, the patient should be Schick tested and Dick tested, and immunizing measures, if necessary, carried out.

3. Careful examinations should be carried out periodically for evidence of new lesions, such as renal tuberculosis.

4. Apart from immobilization, and day-to-day supervision of splintage, temperature charts, etc., the progress of the case should be watched by periodic X-ray examinations, carried out at three-monthly intervals. In addition, interval examinations of the sedimentation rate may give some indication of progress.

LESIONS OF INDIVIDUAL JOINTS

Tuberculous Lesions of the Spine

Method of Examination.—

Clinical.—For the detection of the *early lesion*, the patient must be completely stripped, and placed prone on a firm wooden couch. The spinous processes are carefully palpated and the distances between the individual processes roughly estimated by running the index and second fingers two or three times down the whole length of the spine; any unduly prominent one is also demonstrated thus. The spine is then carefully hyperextended and any sectors not moving with their normal facility noted. In the lower dorsal and lumbar regions the manœuvre is carried out by grasping both ankles with the right hand and palpating the spine with the left. As well as loss of mobility, spasm with rigidity of the erector spinæ muscles is carefully sought. In the case of the cervical and dorsal regions, active movements are the best guide if the child is old enough to carry out instructions. Passive movements of rotation and flexion and extension in the upper cervical region are carried out, with particular delicacy of touch on account of the grave danger of any forcible movements on a softened atlas or axis; also of the cervical spine generally and the upper dorsal spine by extension of the neck or chest with the left hand while the right hand carries out the palpatory movements along the spinous processes.

Suspected areas are as accurately localized as possible for the purposes of X-ray requisition.

Abscesses are then carefully sought for in the usual sites.

Radiographic Examination.—Antero-posterior and lateral views are always requested.

Symptoms.—Unfortunately the lesion is usually only too obvious from the presence of angular deformity. Pain in the back and stiffness in stooping and turning are common complaints. Occasionally the simulation of wry-neck in cervical cases, and in lumbar cases abdominal pain, or an obvious limp due to a flexed hip from psoas spasm, mislead the unwary.

A description of the distorted attitudes of advanced cases is unnecessary.

Signs.—A prominent spinous process or processes, unequal intervals between the processes, decreased flexibility of the spine, and spasm of the adjacent erector spinæ sectors of muscle, are all obvious on clinical examination.

The deformity is median and angular, except when unilateral vertebral destruction, which is unusual, occurs.

Radiographic Diagnosis.—The lateral view shows in early cases narrowing of the intervertebral disk, and later infiltration and compression anteriorly of one or more vertebral bodies, resulting in wedge-shaped bodies. This appearance occurs in the usual type of lesion, which is an osteomyelitic one and central. More rarely, owing to the focus or infection being situated on the anterior surface of the body, a semilunar excavation is demonstrated in that area. In the latter case more extensive longitudinal spread occurs underneath the anterior spinal ligaments and a considerable length of the spine tends to become involved, but without gross gibbus formation.

Frequently also a paraspinal abscess is demonstrated as a more or less rounded swelling surrounding the vertebral focus, but occasionally an elongated swelling extending parallel with the vertebral column is shown.

The radiographic diagnosis is distinctive; only two conditions are liable to cause confusion—namely, Calvé's disease or a septic vertebral lesion.

In Calvé's disease, a vertebral body is vertically compressed and hypercalcified, and takes up a lamellar shape, which is infrequent in a tuberculous lesion. Furthermore the adjacent disks are never involved in Calvé's disease.

In a septic vertebral osteomyelitis, in a chronic stage with sinus formation, apart from deductions from the clinical history, blood-counts, etc., the usual bone proliferation accompanying

infection with septic organisms is manifest, and in this particular area is shown by ossification occurring under the anterior longitudinal ligament, and bridging together the anterior surfaces of two or more vertebræ.

Complications.—Two are of importance : (1) Abscess formation ; (2) Paralysis.

1. *Abscess Formation.*—Sites of election :—

Cervical.—

a. Retropharyngeal : This must be distinguished from the acute abscess due to pyogenic organisms, which is situated more superficially, close to the mucous membrane and anteriorly to the buccopharyngeal fascia. The distinction is made by clinical examination of the upper cervical spine, supplemented by radiograph.

This form of abscess is, of course, not suitable for aspiration and must be treated by formal incision externally through the skin.

b. Lower cervical : Abscess is uncommon at this site. It is directed by the cervical fascia towards the sternomastoid muscle, of one or other side. It frequently results in wry-neck appearance.

Thoracic.—Abscesses in this area may track around the ribs and make their way to the skin around the intercostal arteries, or descend the posterior mediastinum to become psoas abscesses. However, much more frequently they form globular shadows around the vertebral focus, and are only demonstrable radiographically during the entire course of their evolution. They thus usually remain closely pent up in immediate proximity to the lesion, and this is the reason for the much greater incidence of paralysis in this region.

The treatment of these abscesses will be discussed under PARALYSIS.

Lumbar and Lumbosacral.—These are the common sites which produce psoas and iliac abscesses. They form collections of tuberculous ' pus ' in the right and left iliac fossæ, and only when neglected do they descend underneath Poupart's ligament into the thigh, or down into the pelvis to discharge via the rectum or to form ischiorectal abscesses. But with greater frequency they project posteriorly, especially in the case of children undergoing recumbency treatment, and are palpable in the loin.

Iliac abscesses lie outside the peritoneum and iliac fascia, and displace the viscera inwards, and they are therefore easily

aspirated. Aspiration should be carried out only when a well-defined collection of 'pus' has formed. It is carried out by inserting the trocar well below the anterior superior spine and thrusting it obliquely upwards and backwards into the collection. The left hand thrusts the viscera medially and at the same time presses the abscess in the direction of the penetrating needle. Even though it is necessary to wait for a well-defined swelling, aspiration should not be deferred until the 'pus' becomes subcutaneous, as secondary infection and sinus formation are then very probable sequelæ.

In addition, a certain degree of hydronephrosis is often associated with such a collection, and evacuation should on that account also be performed at the earliest safe opportunity.

Abscesses so situated give rise to psoas spasm and hip flexion, but differentiation from tuberculous hip disease is always easy, as the only movement restricted is that of extension, whereas in hip-joint disease in its usual forms there is some degree of restriction of movement in every direction.

2. *Paraplegia.*—For fuller information on this subject the work of Seddon and Butler should be consulted. The treatment of paraplegia is essentially the treatment of the originating spinal lesion. But when a radiographic abscess is present and no decided improvement is occurring after three to six months' adequate fixation, the simple operation of costo-transversectomy should be carried out to relieve the cord from pressure and prevent irremediable damage. For a successful outcome the abscess should be tense, and the operation performed at a stage when the 'pus' is fluid and not thick and solidified.

For paraplegia due to other factors, there is little prospect of improvement by operative measures. In the late forms occurring some years after the original lesion, the treatment should be a return to recumbency and fixation. The outlook is poor, and the aim should be prevention of this form of paraplegia by adequate and sufficiently prolonged treatment of the initial lesion.

Treatment of Vertebral Lesions.—

1. *Plaster Bed.*—This should be reserved for very active and painful lesions. A bed of plaster is made to encase the patient from the crown to the soles, and fitting the posterior half of the body. It should be padded with two longitudinal strips of felt on each side of the prominent spine, the knees should be kept slightly flexed by padding behind the tibial heads,

the feet maintained at right angles by the plaster, and a space adequately cut out at the buttocks for nursing purposes. Wooden blocks placed at intervals underneath raise the plaster bed from the underlying mattress or board. An anterior plaster shell should be made for turning purposes so that the skin of the back may be carefully tended.

In this, as in any fixation apparatus when the patient is in dorsal decubitus, the upper end should be kept tilted on a higher block than the lower end, to facilitate drainage of the kidney pelvis and prevent calculus formation.

2. *Whitman Frame.*—This is the most generally useful form of apparatus. It is a rectangular frame, longer and narrower than the body, and fitted with a tightly laced corset, body bands, and shoulder straps. Each frame is supplied with foot-pieces, usually knock-knee bars, and, for lesions above the mid-dorsal region, head-piece and forehead-occiput extension bands. For lower lesions extension strapping may be applied to the legs and tied to the end-piece of the frame or run over pulleys to weights. The tubular steel frame can be bent to develop secondary curves above and below the lesion, but it must be sufficiently rigid not to sag under body weight.

The usual precautions are taken to attend to the skin and to keep it dry by raising the frame off the bed-board on blocks so that there is a free air space between the splint and the bed or carriage. For turning purposes use anterior plaster shell.

The knees are kept slightly flexed by transverse pads just below the line of the knee-joints, and the child is encouraged to thrust the feet down against the foot-pieces night and morning, in order to keep the joints free, and preserve the muscle tone of the limbs.

When the lesion is commencing to heal and recalcify, the frame is reversed, and the patient, instead of lying supine on the convexity, is placed prone in the concavity. In this position the back muscles are developed and strengthened, and better kidney drainage secured.

When it is thought from the clinical and radiographic examination that quiescence and stability have been reached, the usual tentative period of freedom from fixation is allowed, and if satisfactory, the prone position is largely maintained and the child gradually prepared for further activity.

Ultimately short periods of ambulation are allowed daily, and in increasing periods, in a well-fitting posterior spinal

support, and with boots tilted $\frac{1}{4}$ in. on the inner side of the heel.

Examination at periodic intervals should be continued over a period of years.

3. *Operative Treatment.*—The indications for this line of treatment, by means of fusion operations, are significantly sparse. Operation is only indicated, even when the general postulates of quiescence, etc., are fulfilled, in unilateral vertebral collapse.

Tuberculous Lesions of the Hip

These may be conveniently divided into two primary classes : (1) Intra-articular ; and (2) Extra-articular.

1. *Intra-articular Lesions.*—Common intra-articular lesions are : (*a*) Acetabular ; (*b*) Synovial ; (*c*) Marginal neck lesions.

a. Acetabular : Easily diagnosed radiographically by widening of the Y cartilage. Clinical diagnosis easy and typical. Prognosis is fairly good, as healing occurs usually without much destruction, and ultimately sound and persistent ankylosis.

b. Synovial : Here marked and obvious clinical symptoms occur without distinctive radiographic findings, at least for a considerable time. In the early radiograph there is *slight rarefaction* as compared with the sound side, both hip-joints being taken antero-posteriorly on the same film. A pulpy thickened synovial membrane may be delineated. Frequently also a slight effusion may be detected, if the film is carefully examined, by an increased width of the joint space between the radiographic U and the outline of the femoral head inferiorly, whilst superiorly the joint space is slightly decreased.

Here, as in all types, with the passage of time the radiographic picture becomes typical and shows the characteristic features of erosion and destruction. It is worth noting that this grosser radiographic picture corresponds for the most part to a stage of favourable clinical progress. The earlier radiographs with no loss of bony outline correspond to the stage of invasion and activity, and the later ones, showing eroded areas, to the pathological stage of removal of grossly involved tissues.

The primary synovial type of lesion is slow to sclerose and prone to relapse. The contact of opposing articular surfaces is poor, and cartilage denudation minimal, and so there is a poor response to extra-articular operative fusion.

c. Marginal neck lesion : Here the usual clinical phenomena are demonstrated early, associated with early and distinctive radiographic appearance of a semilunar deposit in the outer side of the femoral neck, the base of the crescent abutting on the outer neck margin.

Abscess formation is frequent, though subjective symptoms are slight. The lesion is relatively benign, and when the infiltrated area is removed there is a very broad area of bony surface in contact. It therefore responds very well to operative fusion.

2. *Extra-Articular Lesions.*—These are (*a*) Femoral ; (*b*) Iliac.

Both lesions are liable to give rise to intermittency of symptoms due to ' sympathetic ' synovial effusion, and from the clinical standpoint are therefore confusing, but the X-ray picture is characteristic at an early stage. In the latter stages this position may be reversed, especially in the cervical type. In this type nature's demolition of the infiltrated areas often presents bizarre X-ray appearances, particularly suggesting cystic changes, while the clinical picture is typical.

a. Femoral : As the cervical type is very intractable to treatment after joint involvement, if detected prior to joint invasion the focus should be evacuated operatively by subtrochanteric tunnelling.

Though this method of procedure is not generally approved, still a goodly percentage of cases thus treated do completely recover without joint involvement, and in any case no detrimental effects follow. If the operative procedure is carefully carried out, the operative approach would appear to be justified. Of course the conservative treatment by splintage, etc., is carried out without any relaxation or remission, whether operation is undertaken or not.

b. Iliac : Unlike the preceding type, this tends to heal without any joint involvement, and should undergo the usual methods of fixation treatment.

Symptoms.—Pain and limp constitute the cardinal symptoms. The former is frequently referred to the knee. It is usually of no great severity, unless when there is considerable tension within the joint or when erosion of cartilage is occurring. In the latter it occurs with severity at night, waking the child from sleep, constituting the ' night cries ' of hip disease.

Limp is a prominent symptom, though occasionally it may show intermittency.

Signs.—

Tenderness.—This varies from case to case and at various epochs in the evolution of the malady. Its subsidence is some evidence of quiescence. It should only be tested for with extreme delicacy as a diagnostic aid.

Joint Thickening.—This is of considerable diagnostic importance in children, and is estimated by comparing the thickness of the joint on the two sides by palpation between the index finger placed posteriorly and thumb anteriorly over the femoral neck, using both hands simultaneously, the left for the right joint and the right for the left joint.

Muscle Wasting.—Its degree and rapidity of development is particularly well marked in tuberculous lesions. It should be sought not only in the quadriceps group but also in the gluteal mass.

Glandular Involvement.—Enlargement of the iliac group of lymph-glands is emphasized by French observers, but the author finds himself unable to place much reliance on this sign. Biopsy has been carried out on some cases by Clarke in the diagnosis of doubtful lesions.

Limitation of Movement.—This particular examination is of the greatest importance, and must be carried out with exactitude. To do this, and avoid getting false results from pelvic movements, either the pelvis must be fixed by keeping one hip fully flexed while the other is examined, or by feeling the anterior superior spines between the fingers of one hand while the leg flexed at the knee and grasped above the ankle is manipulated with the other.

With the patient on his back on a firm couch the movements of flexion, extension, abduction, and adduction are compared on the two sides, but hyperextension and rotation are best carried out with the patient prone. The pelvis is fixed, and the lower spine palpated with one hand, while the movements mentioned above are carried out by grasping the lower leg above the ankle with the knee flexed to about 90°.

All movements are restricted once the interior of the joint is involved in this as in all cases of an arthritis, but in the extra-articular lesions there may be little or no restriction of any movement, and diagnosis depends mainly on the radiographic examination.

Muscle Spasm.—For this the controlling hand is removed from above, and palpates the muscles of the upper thigh,

buttock, and lower abdomen while gentle movements are carried out. The palpation of the abdominal recti is particularly useful, especially when the limb is carried into internal rotation, as a test for quiescence (Gauvain).

Deformity.—The grosser and more advanced forms of lesion are accompanied by obvious deformity, easily demonstrable by placing the anterior superior spines on the same transverse plane, by visualizing the lumbar hollow when the legs are placed extended and flat on the couch, and by measurement.

Radiographic Diagnosis.—In extra-articular lesions this is the main objective criterion, and deposits of usually diminished density are shown delimited from the surrounding bone.

In the true articular lesions, when primarily bony, the X-ray diagnosis is again distinctive, but when primarily synovial, radiographic diagnosis is difficult. In the latter the clinical diagnosis frequently antedates the radiographic one. However, with a little care, in early synovial lesions some important changes are to be seen, if sought, in the radiograph, particularly : (1) the signs of effusion previously described, and (2) rarefaction of the articular ends.

In all cases, of course, it is necessary that antero-posterior views of both hips, well centred, should be provided for examination.

Occasionally, if one hip is held slightly flexed from spasm, an epiphysis with an apparently diminished upward convexity is shown, and to the casual observer it may suggest Perthes' disease. However, the ossification of the epiphysis is regular and rarefied, if anything, not hypercalcified, and the coincident changes of Perthes' disease in the neck and acetabulum are absent, for in the latter lesion the neck is thickened, and the outline of the acetabulum is that of half a lemon and not of half an orange (Calot).

Radiographic examination is of the greatest importance in forming an opinion as to the progress of the lesion. The 'fuzzy' blurred outline of the acute hip, with favourable progress becomes later replaced by a clearly demarcated bony outline, even though areas of infiltration are replaced by areas of obvious erosion.

Complications.—

Deformity.—In early lesions the joint assumes the position of flexion, abduction, and external rotation. Later the position becomes one of flexion, adduction, and internal rotation. In

late cases (though exceptionally an early dislocation occurs with joint distension) erosion of the acetabular rim may occur superiorly, and the femoral head may move outwards and upwards from the acetabulum (wandering acetabulum).

Abscess.—This most frequently occurs in front or on the outer side of the thigh, occasionally behind the femoral vessels, or in the adductor region, and rarely in the iliac fossa, pelvis, or ischiorectal fossa. And, as elsewhere, abscesses may form sinuses in these varying locations.

Differential Diagnosis.—

Simple Coxitis.—A lesion to which attention has been drawn by Fairbank occasionally occurs following the exanthemata, particularly chicken-pox. Its differentiation may be difficult in the early stages, though the history may be suggestive, but it clears up absolutely and permanently after a few weeks' fixation.

Perthes' Disease (Pseudo-coxalgia, Coxa plana).—Clinically the presence of this lesion is suggested by the age of the patient (five to ten years), who is usually a boy; there is a very obvious limp with marked intermittency, little pain or tenderness, some broadening of the buttock, and generally preponderance of limitation of movement in the directions of abduction and internal rotation, with usually little or no spasm. Radiographic examination shows distinctive appearances in the acetabulum and neck, if flattening, hypercalcification, and fragmentation are not already present in the epiphysis of the femoral head.

Coxa Vara.—Here the lesion is again suggested by the clinical findings, and the diagnosis is made precise by radiography.

Epiphysis of Infancy or *Acute Suppurative Arthritis.*—In this lesion the history dates back to infancy; usually a scar of spontaneous or operative evacuation is visible, and a dislocation or subluxation is present. Movements are free, and the gait simulates that of unilateral congenital dislocation of the hip. The radiograph shows a clear bone outline, and absence of the epiphysis, or a stunted epiphysis and femoral neck, together with displacement of the articular surfaces.

Congenital Dislocation.—The history, the limp, freedom of movement, absence of femoral head from its usual situation behind the femoral vessels on clinical examination, and distinctive radiographic appearance prevent any confusion in diagnosis.

Treatment.—

1. *Uncomplicated Lesions.*—The following methods may be adopted: (*a*) Pugh's method of fixed traction; (*b*) Weight and pulley traction; (*c*) Jones's abduction frame; (*d*) Plaster spica; (*e*) Operative treatment.

a. Pugh's method: Two broad bands, which are wide enough to encircle the thigh, of adhesive strapping are applied; the lower end of the bed is tilted high on blocks, and the adhesive ends tied to a fixed upright projecting above the end of the bed. No weight or pulley is used. The sound leg is left free, but prevented from rotating by a foot-piece, or wooden bar nailed transversely to the heel of a boot. The shoulders are restrained by a transverse band of webbing running through two axillary loops. This is a very useful method in young children and in cases with much flexion due to spasm.

b. Weight and pulley: Extension strapping is placed in the usual way from the top of the thigh to just above the malleoli, and passed through slots in a wooden foot-piece which also acts as a spreader. The extensions are then tied to a small wooden block, which carries the extension cord. The weight attached is usually equivalent in pounds to the age of the child plus 1 pound.

If deformity is present, traction is carried out in the line of such deformity, using an adjustable inclined plane, if necessary, to support the leg.

Counter-extension is made by tilting the lower end of the bed, with, in addition, perineal bands attached to the top end of the bed. A suitable foot-rest, or wooden boot, is fixed to the sound foot to prevent foot-drop.

This method is usually only applied as a temporary measure to correct deformity due to spasm, and to get the leg in the desired position of abduction, extension, and mid-rotation.

c. Jones's abduction frame: When the position has been correctly adjusted (if any adjustment is necessary) by gradual traction by the preceding method, or by immediate gentle reduction under anæsthesia, the leg is placed in this frame. The frame must be correctly fitting, and the side-bars adjusted, particularly the pelvic bars, by wrenches.

Extensions are applied to both legs and the feet supported at right angles in foot-pieces. Knee pads are placed behind the tibial heads, and perineal straps adjusted and tied so that the buttocks are prevented from sagging down below the saddle

of the splint. Then the extensions, which are continued by lamp wick from the adhesive ends, are tied tightly to the end-pieces of the splint, and a windlass left in place for further tightening. The wing-pieces of the frame are bent closely to the trunk and the knees bandaged to the knock-knee bars.

Nursing is continued in this appliance till quiescence is reached. This is a charge of much responsibility and requiring much assiduous care, particularly in regard to the perineal straps and leg extensions, the posterior knee pads, and the skin.

When clinical and radiographic examination shows quiescence the usual trial period of freedom from fixation is allowed. If no relapse occurs, ambulation is allowed preferably in a short walking plaster spica rather than in a walking calliper splint. This splint must be longer than the leg, so that when the child is erect the heel of the foot does not reach the bottom of the boot, and therefore the body weight is transferred from the pelvis through the irons to the ground, and thus weight-bearing through the leg is greatly reduced. If no shortening is present, a rise or patten is added to the boot, still keeping the affected side $\frac{1}{2}$ in. shorter than the sound one.

The usual periodic examination is carried out at three-monthly intervals over a period of years.

d. Plaster spica: This is only used in exceptional cases. It is applied in the customary way and extends from the thorax to the toes on the affected side, made closely fitting and well moulded to the body and leg. It is, of course, necessary to have the hip in the optimum position for fixation, i.e., moderate abduction without rotation, and the knee slightly flexed with the foot at right angles.

For prolonged fixation this method is now little used, but a variation of it is occasionally very useful for reduction of prolonged flexion (Hunt). Here the affected hip is flexed until the lumbar lordosis is fully reduced, and a long plaster spica is applied to the sound side from thorax to toes. This fixes the body, and strong traction can be efficiently applied to the affected side. When reduction has occurred, then the abduction frame is again put into use.

e. Operative treatment: When deformity persistently recurs after each tentative period of freedom, or relapsing deformity occurs in the ambulatory period, operative fusion of the hip finds suitable application.

Here a bone-graft is either turned down from the ilium (Wilson), up from the trochanter (Hibbs), or a free tibial graft is used ; often indeed a combination of these methods is employed to secure ankylosis. The graft is placed to bridge the interval between the trochanter major and the ilium, and care is taken to encroach as little as possible on the actual site of the disease.

The operation can be very suitably done under low spinal anæsthesia. At its conclusion a large plaster is applied, which includes the thorax, abdomen (here the plaster is cut out centrally), the affected leg completely, and the sound thigh. A cross-bar of plaster between the two thighs gives added security against breakage.

After a period of at least three months, the graft should be solidly enough in place to allow of ambulation in a short plaster spica. A few months later, with favourable progress bony ankylosis should commence to occur across the actual joint space.

If flexion or eversion is present, it may either be corrected by osteotomy at the time of the fusion operation or subsequently. Another method of hip arthrodosis, described by Tumble and elaborated by Brittain (1942), in which the graft is inserted across femur into ischium, possesses obvious architectural advantages, especially as regards prevention of adduction deformity.

The operative results in well-chosen cases are very good. They are especially good in cases where there is a large area of head and neck in contact with a wide expanse of ilium.

In cases relapsing during the trial period of freedom, a period of ambulation in a short spica should be practised, and an interval thus placed between the period of recumbency and the time of operation. This allows for general rehabilitation, improved circulatory tone from exercise, recalcification of the skeleton generally, and, of more importance, recalcification of the great-trochanteric area and the ilium, so necessary for their function in the grafting procedure.

2. *Treatment of Complicating Dislocations.*—

Preventive : The maintenance of adequate abduction during the acute and subacute phases.

Corrective : *Early dislocation*—reduction under anæsthesia. *Late dislocation*—general splintage of the lesion and, at a suitable period subsequently, extra-articular fusion and osteotomy.

Tuberculous Arthritis of the Knee

Clinical Examination.—Apart from ordinary methods, particular attention should be paid to the temperature at the joint level and adjacent limb areas above and below the joint. Calvé has drawn attention to the reversal of the normal physiological comparative temperatures in these areas in this lesion. In health the joint itself feels cooler to the palpating hand than the adjacent parts above and below.

As regards passive movements, it is essential to test flexion with the patient prone. In this posture a slight limitation of flexion, which would otherwise escape notice, is easily detected.

Thickening of the synovia should be sought not only in front but all around the joint area.

Biopsy.—Glandular enlargement in the groin is stressed by French observers, but in general there is little difference to be made out between the two sides. Whether histological and other apposite examinations of an excised inguinal gland are of any utility as a diagnostic procedure is still under discussion.

Radiographic Examination.—In every questionable case antero-posterior and lateral films of each side must be presented. The X-ray findings in the early synovial case depend mainly on the delineation of a thickened synovial membrane; a change, usually an increase, in the size of the joint space; and an enlargement of the upper tibial epiphysis on the affected side. If the lesion is primarily osseous, the X-ray picture is generally definitive.

Types of Lesions.—

1. *Synovial.*—Here there is palpable thickening of the synovial membrane, and the supporting X-ray findings described above.

2. *Articular Erosion.*—Easily demonstrated radiographically.

3. *Metaphysial and Epiphysial Lesions with Sequestrum Formation.*—A wide wedge-shaped area of bone is demarcated, the apex of the wedge in the metaphysis, and the base in the epiphysis or sometimes actually abutting on the articular cartilage. The involved area runs uninterruptedly across the epiphysial line, unlike septic lesions which stop abruptly at that line.

Complications.—

Abscesses.—These may occur in proximity to the joint, or loculated collections may occur actually in the joint, especially

in the suprapatellar pouch. In either situation they should be treated by aspiration in the usual way.

Deformity.—Flexion is the usual deformity, in late cases associated with posterior subluxation of the tibial head.

Differential Diagnosis.—

Simple Chronic Synovitis.—This is rare in children, and a lesion such as this should be treated as a tuberculous one until such time as the latter can be definitely excluded.

Syphilitic Synovitis.—This is a common lesion and is usually suspected from the absence of any considerable synovial thickening, and relative painlessness in a much swollen knee. Not infrequently the opposite knee is simultaneously involved, or soon also becomes involved. Other stigmata may be present, particularly interstitial keratitis, and the Wassermann reaction is positive.

Hæmophiliac Knee.—Its occurrence in boys, the history and the occurrence of periodic increase in size with further attacks, even when obvious hæmorrhages are not apparent elsewhere, should make the diagnosis clear.

Treatment.—Tuberculous knee lesions are very persistent and require prolonged treatment.

The Thomas knee splint, with well-fitting ring, adhesive traction, and supporting bands behind the thigh, tibial head, and calf, and foot-pieces, is an excellent method of treatment.

Fixation should be continued until X-ray films show clear-cut bone outline and approximation of the articular ends, and the usual trial period of freedom from fixation shows no tendency to a relapse. Ambulation in a walking calliper splint is then permitted under the usual supervision.

Operative treatment is rarely indicated in the usual synovial or articular lesions, but in the juxta-articular lesions with sequestrum formation, extra-articular evacuation should be carried out; otherwise the latter lesions run a most prolonged course with persistent sinuses. It is in such as these that amputation becomes necessary to avoid amyloid degeneration. Very occasionally in osseous lesions, as distinct from the foregoing, the epiphysis forms practically a free sequestrum, when it should be removed and a modified arthrodesis carried out as well.

Arthrodesis, even in spite of relapsing deformity, should be postponed, if possible, until the child is at least twelve years old. The desired position both after splintage and operative treatment should be one of full extension in children.

Treatment of Flexion Deformity.—This deformity is always preventable if the case is seen early enough. Early in the disease it is easily corrected by supporting the head of the tibia, and applying traction in the knee splint. If more resistant, the thigh is put on an inclined plane, and traction applied by weight and pulley both in the direction of the thigh, and in the direction of the lower leg by separate weights and extensions. When a posterior subluxation has persisted for some time and the other methods fail, it is extremely difficult to correct, but an attempt should be made to straighten the leg in wedged plasters. A long plaster is applied, including the pelvis, leg, and foot, with thick padding in front of the knee; this is then divided at the level of the knee-joint, half way around the circumference of the leg, and wooden wedges of increasing width are inserted at intervals of a few days.

Tuberculous Arthritis of the Ankle

From clinical and radiographic examination the lesion is generally obvious, and does not merit any description.

The lesion may be: (1) Synovial; (2) Erosive; (3) Juxta-articular—exactly like knee lesions.

Treatment.—For juxta-articular lesions operation is indicated; in the other types splintage.

Either a skeleton splint may be used with right-angled foot-piece, and posterior calf and thigh troughs, or a plaster cast. Only in severe lesions is it necessary to carry the cast above the knee—when this is done the knee should be kept slightly flexed. The foot itself is maintained at a right angle to the leg.

In the presence of sinuses it is advisable to use a fenestrated cast.

Tuberculous Tarsitis

This lesion is obvious on clinical and radiographic examination —when generalized—with pulpy swelling, loss of movements of abduction and adduction of the foot, and rarefaction and blurring of the bony outlines in the radiograph.

Practically the only lesion causing confusion is Köhler's disease of the tarsal scaphoid, but here the hypercalcification and compression of the scaphoid bony nucleus in the radiograph, as well as its limitation to boys between three and six years, makes the distinction obvious.

In circumscribed lesions confined to an individual tarsal bone, clinically it is possible to detect thickening and some swelling of the adjacent soft tissues, often associated with superficial abscess or sinus formation, together with definite X-ray changes.

Treatment.—The generalized form is treated on the same lines as the ankle lesion. The localized form, if confined to a single bone, is often best treated by total excision of the affected bone, whether it be os calcis, astragalus, or other, followed by immediate application of a plaster cast.

Early ambulation can be obtained in lesions of the ankle and tarsus by the insertion of a walking iron in the plaster cast.

Tuberculous Lesions of the Upper Limb

These lesions are much less frequent than those of the lower limb. They, however, also run a protracted course and require prolonged splintage. Fortunately they can be treated with the child ambulatory, except in the early and more acute phases.

Tuberculous Arthritis of the Shoulder.—This lesion manifests the typical restriction of movements in every direction characteristic of every arthritis. There is scarcely any other lesion with which it can be confused.

Abscess formation is relatively uncommon, and as articular erosion is usually well marked, ankylosis with complete loss of movement is usual. On this account care must be taken that the arm is kept in the most useful position, that is, in right-angled abduction with the elbow slightly in front of the body and the arm slightly externally rotated.

Treatment.—The desired position may be maintained by a plaster cast embracing pelvis, thorax, arm, and hand. This cast is suitably cut out anteriorly over the thorax and upper abdomen, and keeps the upper arm in the desired position—the elbow flexed, and the wrist fully dorsiflexed. A plaster bar from the elbow to the side of the pelvis gives additional firmness.

An abduction arm splint may be used instead.

Splintage is maintained until the arm can be voluntarily held abducted without the supporting appliance and without sagging at the shoulder.

Tuberculous Arthritis of the Elbow-joint.—This lesion presents all the classical features of tuberculous lesions and its

diagnosis offers no difficulty. The lesion is associated with considerable destruction of the articulating bones, and sinus formation is common.

Treatment.—

1. *Collar and cuff*: The joint is kept flexed by this means at an angle of about 40°, the restraining bands at the neck and wrist being so secured that the child cannot of his own accord remove the fixation. It is a suitable method for the early case.

2. *Plaster casts*: This method of fixation, in which the cast extends to the shoulder and down to the palm, with the wrist held in the usual dorsiflexed position, is suitable for cases with acute symptoms and those with considerable swelling or deformity or sinus formation. If the joint is not in the optimum position its position is corrected under anæsthesia and the plaster immediately applied.

Tuberculous Osteomyelitis

1. Lesions of the Short Long Bones.—The common examples are lesions of the metacarpals, metatarsals, and phalanges. These form a very composite group, occurring as they do with great frequency in infants, because of the multiplicity of lesions, and associated as they are with bone hypertrophy. The only difficulty in diagnosis arises from dactylitis occurring in congenital syphilitic lesions. The history, presence of other stigmata, and Wassermann test provide the distinguishing criteria.

These lesions are mainly of importance as evidence of wide hæmatogenous spread, and general therapy is therefore a primary indication.

The patients should be confined to bed, general therapy and open air treatment instituted, and small plaster casts or small skeletal splints applied.

Surgery finds no place in the treatment of these lesions except for the removal of grossly distorted digits with sinus formation.

When such lesions occur in older children, while the general measures are similar, surgical measures may be more often employed. Thus in the stage of subactivity of a localized lesion, grossly affected bone may be completely excised subperiosteally in the metacarpus or metatarsus, but the epiphysis is spared, if possible, and in other instances removal of sequestra and curettage of the focus may suffice to hasten the healing of sinuses.

2. Lesions of the Long Bones.—

Types.—(*a*) Metaphysial lesions with wedge sequestration; this type generally involves the epiphysis and neighbouring joint; it has already been described in the case of the individual joints which are most frequently involved. (*b*) Small scattered cavities, with little surrounding sclerosis and inconspicuous subperiosteal bone proliferation. (*c*) General osteomyelitis involving the whole or greater part of the diaphysis with subperiosteal bone formation and sinuses.

Small Scattered Cavities.—In this type the affected portion of the limb with the joints immediately above and below the lesion should be adequately splinted, and general therapeutic measures carried out. Most of the lesions will gradually sclerose and a fine limiting subperiosteal regeneration of bone will ultimately consolidate the cure.

If some of the cavities break down spontaneously to form sinuses, these usually heal under the treatment outlined above. With persistent sinuses, and the general condition of the patient good, healing may be hastened by freshening the sinuses by curettage.

Hypertrophic Tuberculous Osteomyelitis.—Before this diagnosis is decided upon the Wassermann reaction should always be taken. The distinction from chronic septic osteomyelitis is not of so much importance, as the treatment is similar.

If seen before the formation of sinuses, it is wise to provide splintage and open-air therapy. If later, the diaphysis must be freely opened up the whole length of the area involved, and the cavity treated either by Orr's method of vaseline gauze drainage and plaster-of-Paris cast, or a skeleton splint applied with open dressing of the wound. Frequently in the latter case dressings may be soon dispensed with and the granulating wound stimulated by exposure to the air.

Certain cases show a very grossly and solidly thickened bone with little or no medullary cavity. They are extremely irksome to treat and do not lend themselves at all favourably to any attempt at radical intervention. General therapy and only minor surgical measures are to be advised.

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