A REVIEW OF PRIMARY COMPLEXES IN THE DEPARTMENT OF PAEDIATRICS OVER A TEN-YEAR PERIOD

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The primary complex in children is known as the primary complex because it is the first time that the child comes in contact with the tubercle bacilli. The primary infection itself is a benign condition. Many primary infections heal during the first stage and progress no further. Every child at some stage or other has to pass through a primary complex and such a healed primary focus confers a certain degree of immunity towards tuberculosis.

SITE OF INFECTION

The lung is the most common portal of entry in human beings. The most common mode of infection is by inhalation. Lincoln (1963) states that between October 1930 and January 1947, 964 consecutively seen children with roentgenographic evidence of primary tuberculosis were observed in the wards of the Children's service in Bellevue Hospital. During this period, a diagnosis of extrapulmonary primary tuberculosis was made only fourteen times. Statistics reported by Ghon and Kudlich on the basis of autopsies show that the pulmonary route is still by far the most common path of infection in pulmonary tuberculosis. This is so in our Department where the primary lung infection was present in 95% of our cases. There were only two cases of lupus vulgaris showing the characteristic lesions of the skin, and middle ear infection due to the tubercle bacilli was present in four cases.

THE PRIMARY PATHOLOGY

When the tubercle bacillus is inhaled it reaches the end of a small bronchiole and lodges in the wall of an airspace at the periphery of the lung. At the site of lodgement is formed the primary focus called the Parrot-Gohn focus and it is a sub-pleural lesion. The right side of the lung is more commonly involved than the left lung because of the more direct route of the right bronchus. Around the primary focus there occurs an exudative non-caseous tissue reaction (primary infiltration) allergic in nature which varies in size of a pea to involvement of a whole lobe. Drainage from the primary focus takes place by lymphatic vessels which lie around the bronchiole to the corresponding lymph node at the hilum. This two-fold infection of the lung focus and the lymph node is known as the primary complex. Usually an interval of six weeks elapse between the implantation of the tubercle bacillus and the development of allergy as manifested by a negative tuberculin test becoming positive. The end of the period of allergy is manifested by certain allergic features, namely phyltenular conjunctivitis, a positive Mantoux test, and the presence of erythema nodosum. Phyltenules are small greyish nodules usually on the limbus accompanied by injection of blood vessels of the adjacent conjunctiva. The presence of phyltenules suggests the possibility of an active tuberculous infection. In our series of cases phyltenular conjunctivitis is fairly common, and it will not be possible to give the exact percentage

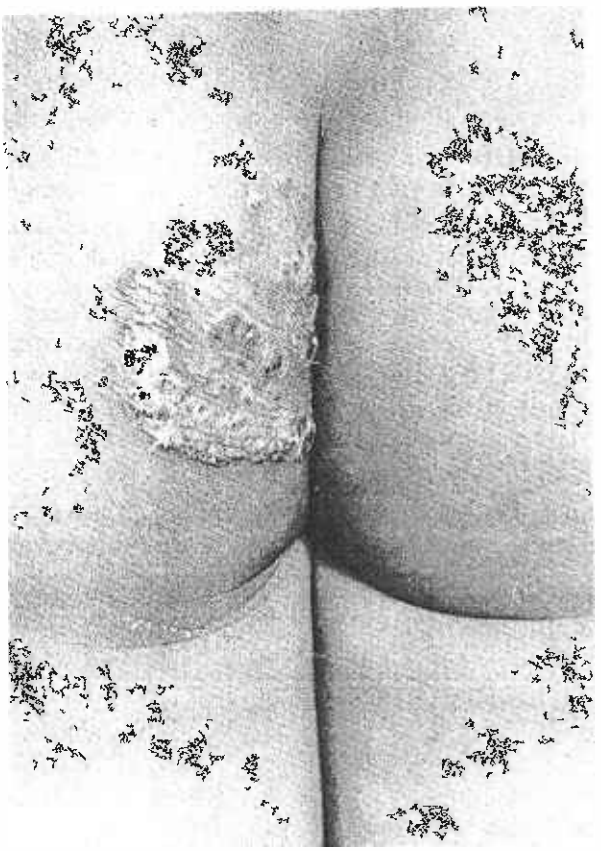

Fig. 1. Lupus vulgaris of the left buttock in a Chinese child.

Year	Primary Complexes	Total Number of Admissions	Total Number of New Outpatients
1956	213 cases	6,473 cases	2,357 cases
1957	177 cases	7,217 cases	2,268 cases
1958	133 cases	9,697 cases	2,055 cases
1959	101 cases	10,517 cases	3,871 cases
1960	105 cases	10,712 cases	4,240 cases
1961	77 cases	10,480 cases	4,680 cases
1962	87 cases	11,922 cases	4,851 cases
*1963	53 cases	6,731 cases	4,047 cases
1964	20 cases	6,249 cases	2,936 cases
1965	10 cases	6,528 cases	6,074 cases

* From 1962, the Unit was divided into Government and University parts and the figures represent the figures admitted to the University side of the Paediatric Unit.

in our cases because of its transitory nature, and many cases will not be reported to the mother, unless the doctor specifically asks for a red eye. Wallgren (1935) in Sweden found 96% of 800 cases gave a positive tuberculin reaction. Among 300 children suffering from erythema nodosum he found radiological evidence of tuberculosis in 90% of the one to five year group, 85% of the five to ten year group, and 56% of the 10 to 15 year group. In our series of cases we certainly do not find such a high incidence of erythema nodosum associated with tuberculosis, and our cases of erythema nodosum are more commonly due to streptococcal sore throat infection, rheumatic fever and the administration of drugs.

INCIDENCE OF PRIMARY COMPLEXES

The number of cases of primary complex in children seen in the Paediatric Unit also show a steady decline. It must be remembered that most of the cases of primary complex are followed as outpatients and are not admitted, except in the event of miliary tuberculosis, or bronchogenic tuberculosis or pleural effusions. The number of children with primary complexes seen in the Paediatric Unit will not reflect the number of cases seen on the island, as the Tan Tock Seng Hospital for tuberculosis, the School clinic, and the Singapore Association for Tuberculosis also follow their own contact cases of primary complexes.

FATE OF A PRIMARY COMPLEX

In 90% of cases there is progressive healing of the primary complex. If the child's resistance is low, and he comes in contact with an overwhelming dose of tubercle bacilli, then complications occur and these can be divided into two groups:—

a) Parenchymal complications.

b) Complications occurring to the lymph node component.

a) **The Parenchymal Component:** The commonest type of lesion that is encountered here is segmental collapse of the lung. In this type of lesion there are very few clinical signs and the segmental collapse can only be demonstrated clinically. The child in such cases looks relatively well. Segmental lesions can often be mistaken for pyogenic ones and in doubtful cases it is best to treat the child with ordinary antibiotics to see if the lesions clear with antibiotics for a pyogenic lesion.

b) The second type of parenchymal lesion one gets is pleural effusion. Pleural effusion is an early complication of a primary infection and both Walgren (1937) and Lincoln and Sewell (1962) in their series of cases found that two-thirds of the cases of pleural effusion developed within six months of the onset of primary tuberculosis. In the Bellevue Hospital where

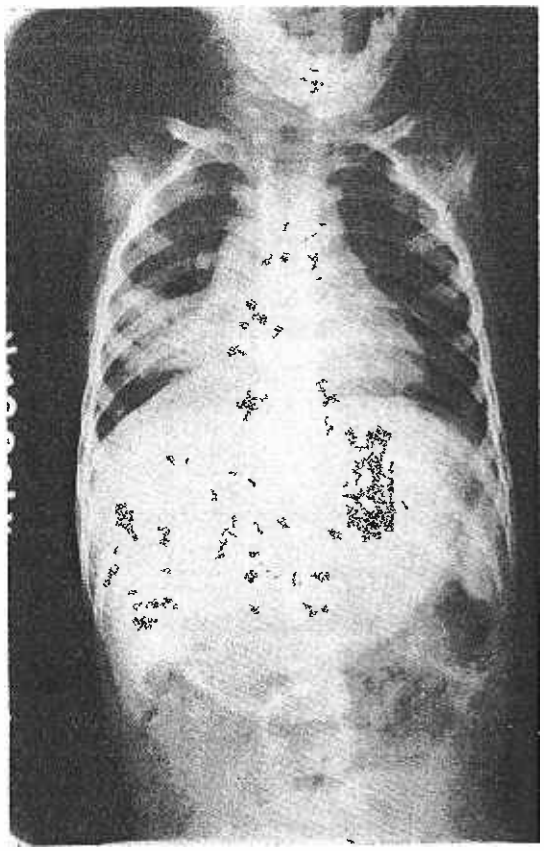

Fig. 2a. Consolidation of the right middle lobe in the T.B. Meningitis patient.

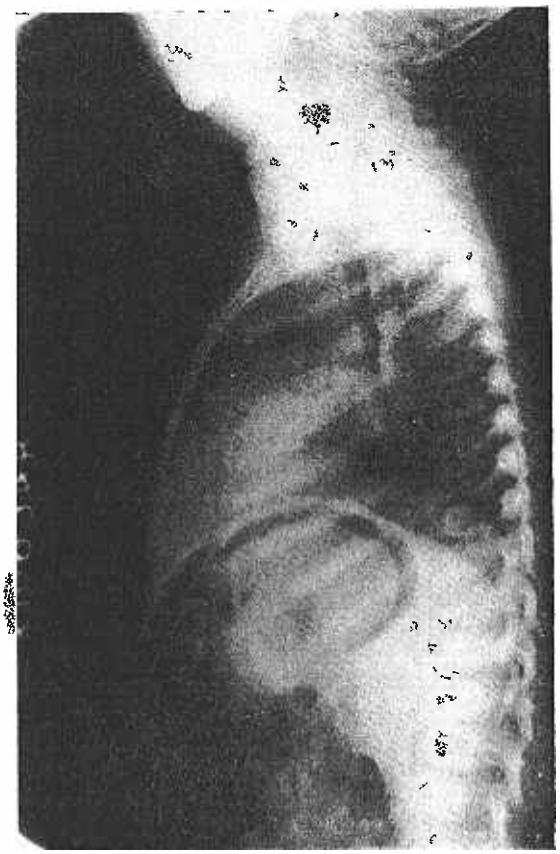

Fig. 2b. Lateral view of consolidation of right middle lobe.

964 children with pulmonary primary tuberculosis occurred primary effusion was diagnosed in 8% of cases. Lincoln and Sewell (1962) state that in Scandinavian countries pleurisy was found more frequently, where the incidence of erythema nodosum was as high as the pleural effusion. The commonest cause of fluid in the pleural cavity in Singapore is pyogenic infection *i.e.* an empyema thoracis but if the fluid is serous then the commonest cause of the pleural effusion is tuberculosis.

When one considers the total number of primary complexes one sees yearly in the Department, pleural effusions are relatively uncommon, and this is supported by the figures below from our Unit.

Year	T.B. Pleural Effusions	Primary Complexes
1956	12	213 cases
1957	9	177 cases
1958	11	133 cases
1959	16	101 cases
1960	10	105 cases
1961	10	77 cases
1962	6	87 cases
1963	1	53 cases
1964	2	20 cases
1965	4	10 cases

PATHOGENESIS OF PLEURAL EFFUSION

A tuberculous pleural effusion may arise in many ways:—

a) Rich states that extension of a lesion from a subpleural focus of tuberculosis is responsible for most cases of pleurisy with effusion. In adults, it has been shown by Stead that a focus of active tuberculosis has been found on the pleura on the same side as the effusion.

b) Lincoln and Sewell (1962) state that effusions may occur during the course of an otherwise asymptomatic pleural primary tuberculosis and in children it is most commonly found on the same side as the primary parenchymal lesion.

c) Pleural effusion may arise as a manifestation of the allergic state of the pleura to the primary infection. Wallgren (1939)

suggests the possible importance of hypersensitivity to tuberculin in the pathogenesis of a pleural exudate.

Many workers seem to attach little importance to its allergic nature because of the fact that the Mantoux is not always strongly positive, yet its rapid absorption with corticosteroid without doubt seems to justify the concept that all or part of the mechanism is due to hypersensitivity. The onset of pleurisy with effusion is occasionally insiduous with fever remaining high for a period of one to two weeks. Unlike empyemas, children with pleural effusions due to tuberculosis do not often have a high swinging temperature and are not very toxic or distressed, unless the collection of fluid is excessive. Constitutional symptoms like lassitude and anorexia are common. The main physical signs are in the involved chest with a dull percussion note and diminished air entry with a shift of the mediastinum. With antituberculous treatment it may take about one to two weeks before fever and constitutional symptoms abate, and about 3 months, before all radiological evidence of fluid disappears. However, an opacity in the costophrenic angle may remain for long periods due to thickening of the pleura. This pleural thickening is well illustrated in the following case report:—

Case No. 1: The patient was a four-year old Chinese Mongol child admitted with fever and cough occurring over a period of two months. Physical examination revealed that the child was not too ill, although radiologically and clinically the effusion was extensive. Physical examination revealed that the temperature was 100°F with a dull percussion note over the right chest and diminished air entry below the inferior angle of the scapula. On aspiration straw-coloured fluid was obtained and on direct smear acid fast bacilli were seen. This child was on antituberculous drugs for over two years and although clinically the child looked well, radiologically there was extensive pleural thickening.

Case No. 2: The second case of pleural effusion was a seven-year old boy who was admitted with a history of seven days fever and cough. Clinically the physical signs were those of an obvious pleural effusion on the right side. The Mantoux test was strongly positive and radiologically the film showed a pleural effusion. The child was put on injections of Streptomycin together with oral isoniazid and Prednisolone and within a period of ten days there was marked clearing of the chest radiologically (Figs. 4 (a) & 4 (b)).

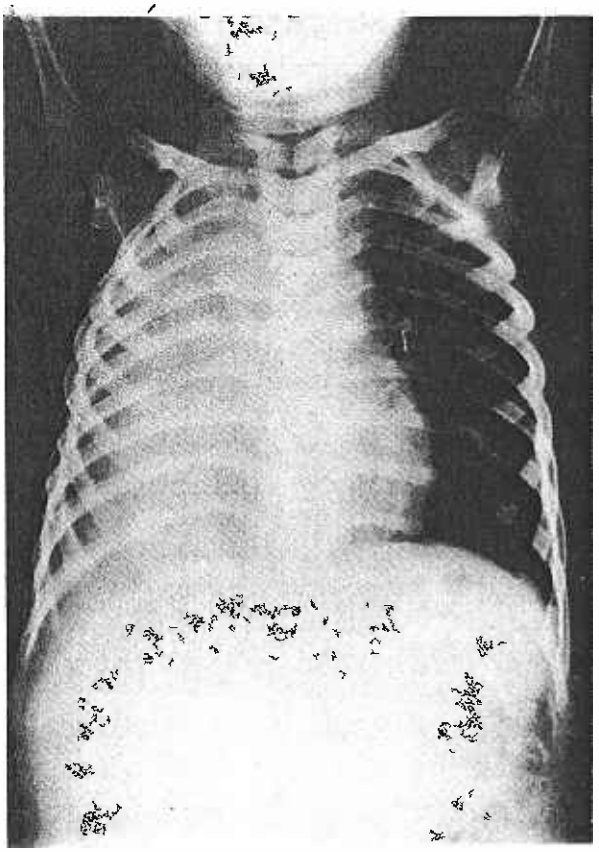

Fig. 3a. Radiograph of the chest to show extensive pleural effusion.

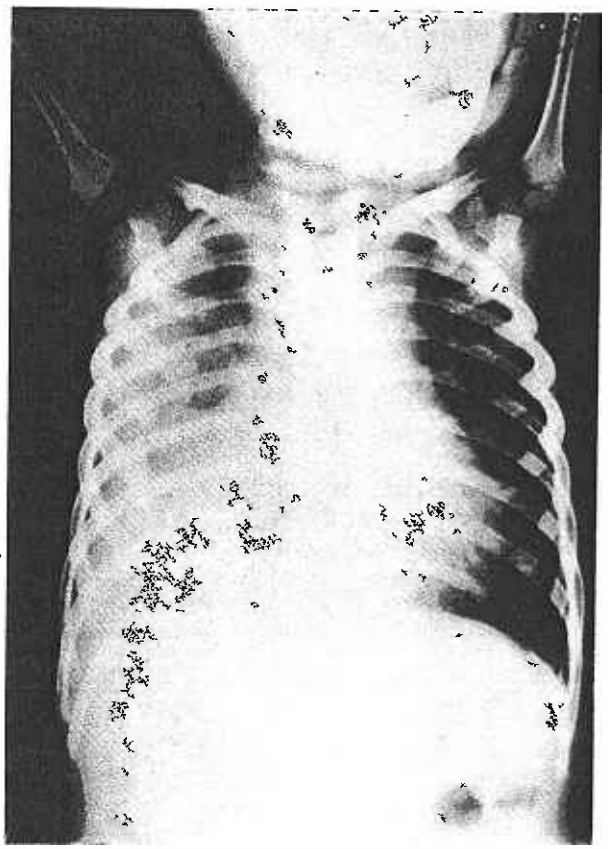

Fig. 3b. Radiograph of the chest to show very little clearing after 1½ years of antituberculous therapy.

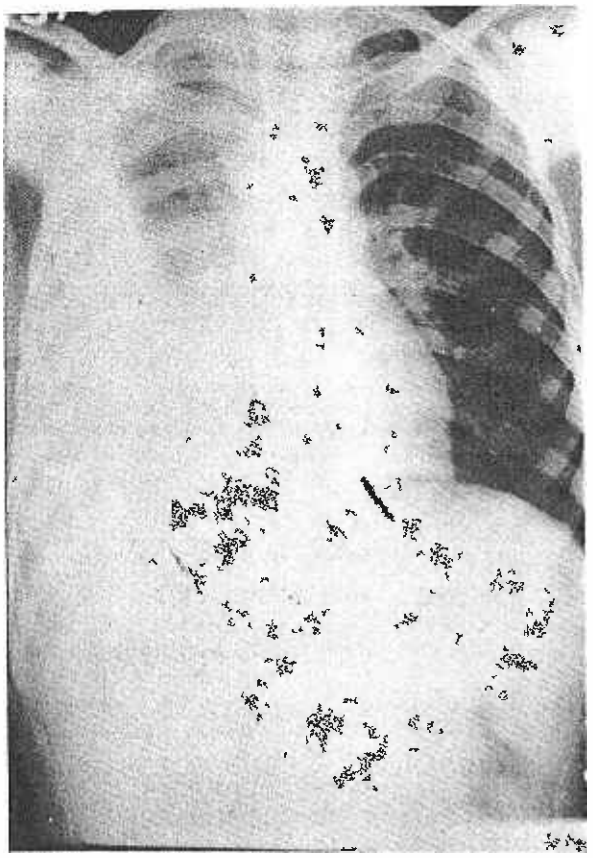

Fig. 4a. Radiograph of the chest to show a pleural effusion before therapy.

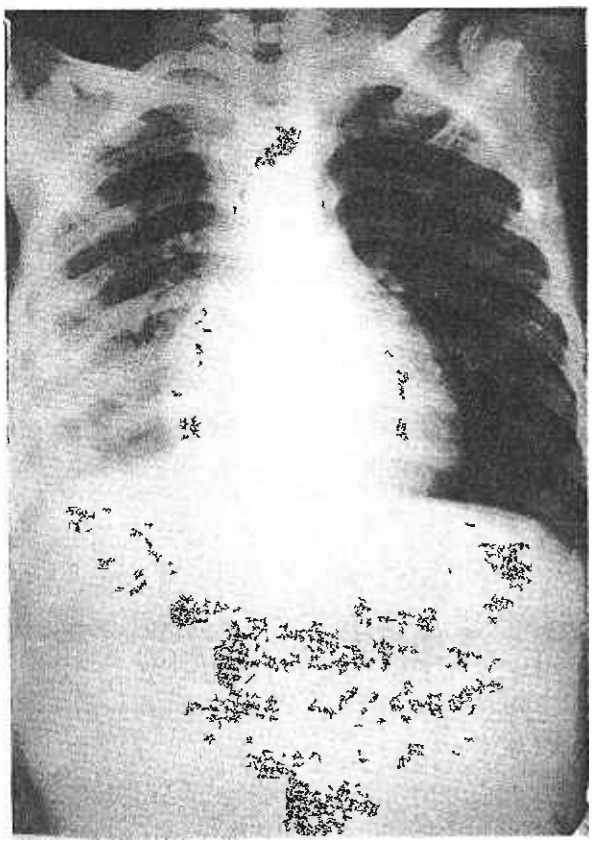

Fig. 4b. Radiograph of the chest to show clearing of the opacity due to a pleural effusion with antituberculous drugs and steroids over a period of ten days.

When the steroids are used simultaneously with antituberculous drugs the constitutional symptoms abate in 2 to 3 days, and radiological absorption of fluid can be evidenced by one to two weeks. The action of steroids indicates that much of the pathology and symptoms are due to a hypersensitive reaction due to the tubercle bacilli. However, the use of corticosteroids does not seem to affect the long-term results. The present line of treatment is the same as for any primary tuberculosis with Streptomycin and isoniazid for three to six months, and isoniazid and para-amino salicylic acid 3 to 6 months. With this treatment full recovery without any sequalae is the final outcome.

MILIARY TUBERCULOSIS

It is generally accepted that widespread dissemination of bacilli occurs in the first few months after a primary infection. The term "fine disseminated tuberculosis" is confined to those lesions seen in the lung on radiograph or at autopsy rather than generalised tuberculosis when widespread dissemination is implied. The term "miliary" is derived from the Latin word "miliarus" or the size of the millet seed. A millet seed has the average diameter of less than 2 mm. The mottlings seen on radiograph may be so small that they are barely visible. It is well-known that miliary tubercles may be of a certain size and density before they can show up on radiograph and not uncommonly one can get miliary tubercles at post-mortem without necessarily showing up radiologically.

AETIOLOGY

The mottled pulmonary lesions are due to bacilli getting into the capillaries and infecting the lung parenchyma. Lincoln and Sewell (1962) state that bacilli can get into the lung capillaries in the following ways:

a) from a caseating focus invading the adjoining lymph node, which invades the child's blood vessels and is made easier than in adults because the blood vessels of children have fewer elastic fibres in the walls than those of adults and are, therefore, more vulnerable to invasion by tubercle bacilli.

b) The disseminating focus may be a tubercle formed by bacilli in the intima of the blood vessels during the course of occult dissemination. In general, they are found at the junction of small pulmonary veins.

INCIDENCE

The following figures illustrate the number of cases of miliary tuberculosis seen amongst our cases of primary complexes. As seen below there is a rapid decline in the incidence of miliary tuberculosis in the Department of Paediatrics.

Year	Miliary T.B.	Primary Complexes
1956	23 cases	213 cases
1957	10 cases	177 cases
1958	13 cases	133 cases
1959	14 cases	101 cases
1960	11 cases	105 cases
1961	15 cases	77 cases
*1962	4 cases	87 cases
1963	4 cases	53 cases
1964	5 cases	20 cases
1965	2 cases	10 cases

* From 1962 onwards, the Paediatric Unit was divided into University and Government Departments and the figures represent the figures in the University Department.

Fever was present in 100% of our cases and most of the cases presented as a pyrexia of unknown origin. The symptoms were insiduous in onset with loss of weight, anorexia, and the patients were on the whole very ill and toxic. None of the patients was dyspnoeic, and no physical signs were audible in the chest. Hepatosplenomegaly was found in 80% of our cases. Choroidal tubercles were invariably present if one made a really good look for the tubercles. Miller (1963) found them in 11 of the 52 cases, while Illingworth and Wright (1948) reported them in 25 of the 43 children with miliary tuberculosis and tuberculous meningitis, while Illingworth and Lorber (1956) reported their presence in 70 of 99 children with radiological evidence of miliary tuberculosis or without meningitis. A positive culture for T.B. bacilli was found in 25% of our cases with miliary tuberculosis where a gastric lavage was done. The outcome of miliary tuberculosis has completely reversed since the advent of chemotherapy. Formerly nearly every patient died within three months of diagnosis. In Singapore, Haridas (1946) found that the mortality for childhood tuberculosis was nearly 100%. Nowadays, with antituberculous therapy the prognosis is good. With Streptomycin and isoniazid, resolution of the mottlings takes place in 4 weeks, and complete clearance of the mottlings does not occur until 3 months later. Below is the radiograph of the chest of a six-year old girl who presented with fever over a period of 3 weeks. The lungs were riddled with tubercles and radiological clearing with Streptomycin and isoniazid took about 3 months (Figs. 5 (a) & 5 (b)).

Nowadays, with the addition of steroids together with the antituberculous drugs, the clearing is much quicker. Below is the radiograph of a seven-year old Malay girl, admitted to the Unit as a pyrexia of unknown origin with hepatosplenomegaly. The Mantoux reaction was negative and a radiograph of the chest showed soft miliary mottlings (Fig. 6a). The cerebrospinal fluid was normal, and the fundus showed choroidal tubercles. The patient was put on Streptomycin and isoniazid and Prednisolone and within a period of five days, there was marked clearing of the soft tissue shadows (Fig. 6b).

This case illustrates the usefulness of steroids in reducing the anti-allergic reaction round each tubercle. Speiss (1962) in animal experiments has shown that on autopsy and on radiological examination of these animals, that the allergic reaction was considerably reduced, but the essential lesion such as the primary focus did not show much more rapid healing while on steroids compared to those without steroids. However, there was no deterioration of the lesion as long as antituberculous drugs were given in adequate dosage. From the work of Speiss, it can be said that steroids allow the antituberculous drugs to penetrate the lesion and allow their action against the tuberculous bacilli. Furthermore, the toxicity due to allergic reactions such as fever, anorexia, discomfort can be dramatically reduced by the concomitant exhibition of steroids. In the case described above when the lesions showed marked clearing, steroids were used for a period of one month only.

TUBERCULOSIS OF THE BRONCHI

The lymph nodes are the cause of endobronchial disease in primary tuberculosis. In children, the nodes that drain the primary focus always become tuberculous. The infections may be limited to the outer bronchial wall, but often the infection progresses inwards through the wall of the bronchus to the mucosal lining. Practically

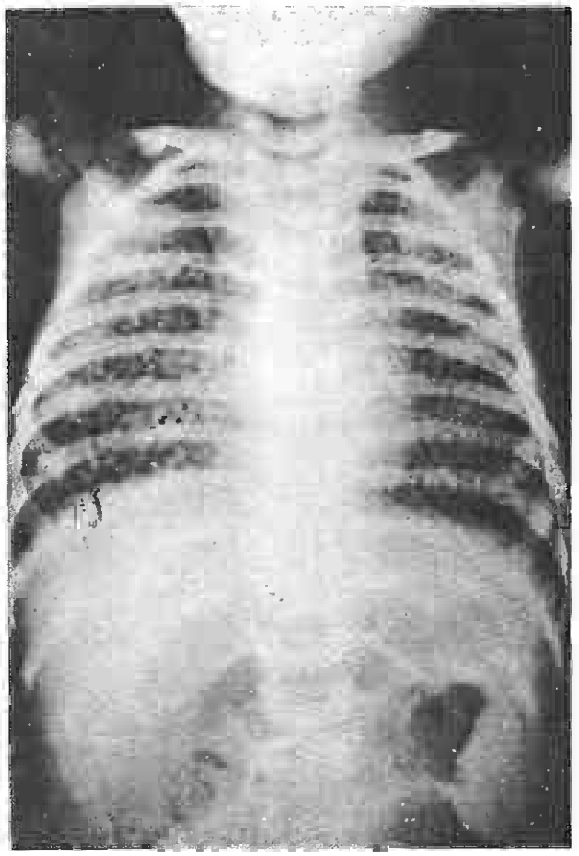

Fig. 5a. Note the widespread miliary tubercles with hepatosplenomegaly.

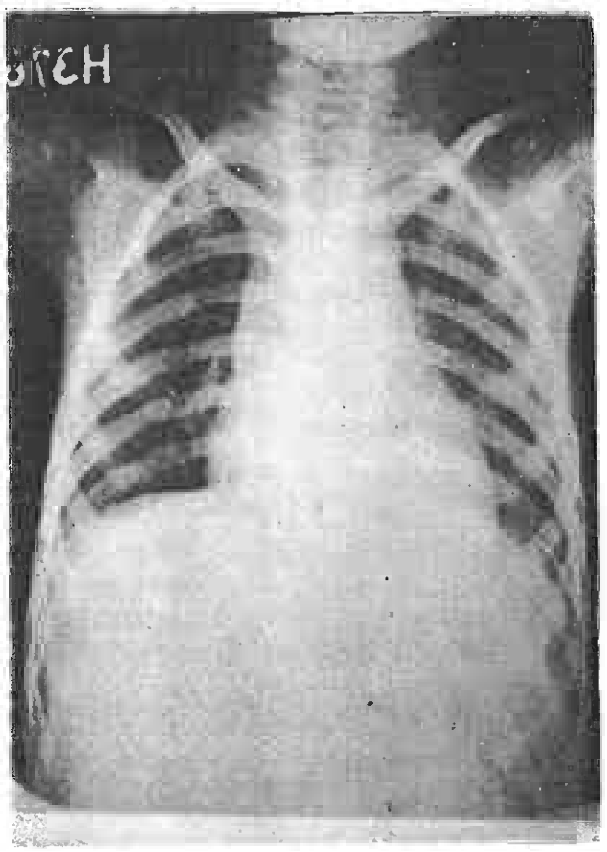

Fig. 5b. Note clearing of the miliary mottlings with Streptomycin and isoniazid after 3 months.

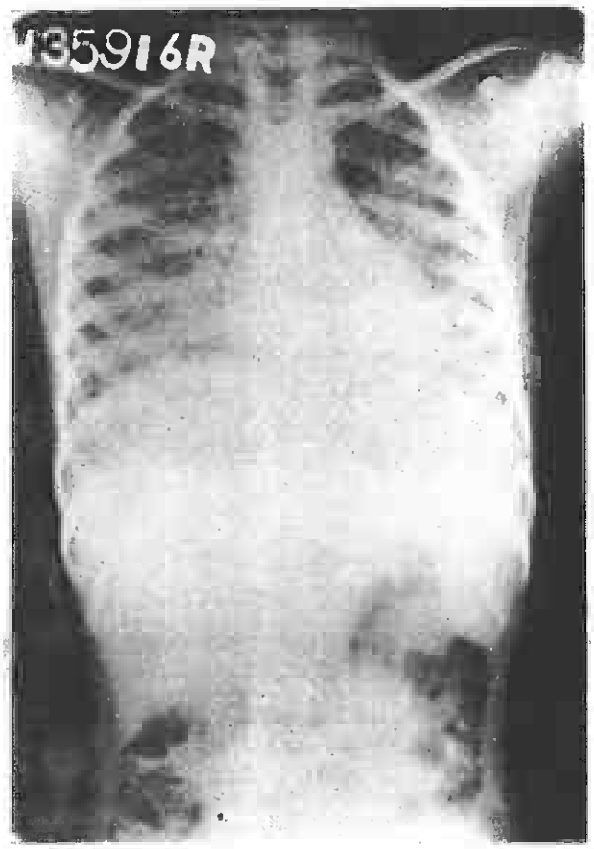

Fig. 6a. Note the soft miliary shadows of the chest in a 7-year old Malay. Note the hepatosplenomegaly due to tuberculosis.

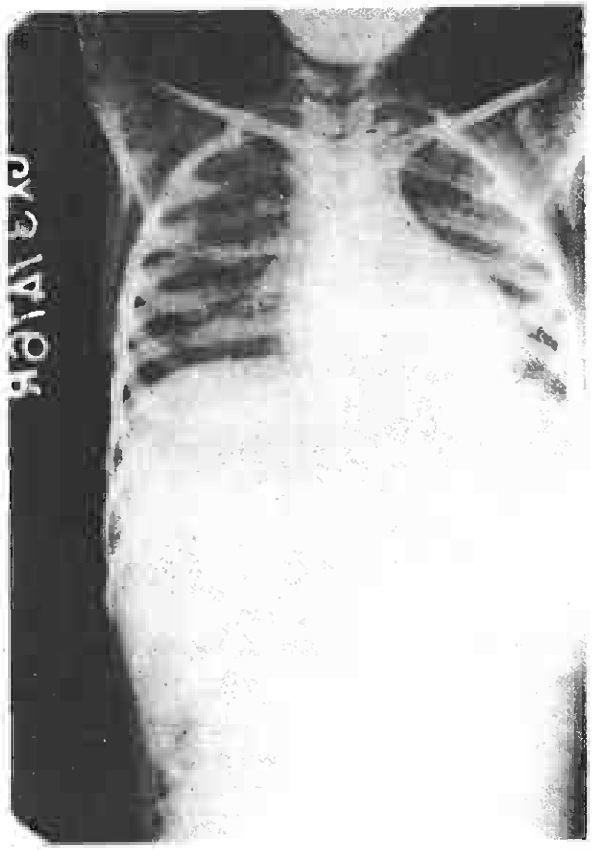

Fig. 6b. Note the clearing of the chest with Streptomycin, isoniazid and Prednisolone at the end of five days of steroid therapy.

every child with pulmonary primary tuberculosis has some involvement of the bronchi, even if this never progresses beyond the first stage, fixation of the node to the outer bronchial wall. Therefore, endobronchial tuberculosis is considered an integral part of primary tuberculosis. If the infection in the bronchial wall progresses, ulceration of the mucosa develops and granulation tissue forms. Complete obstruction to the bronchus by caseous granulation tissue will give rise to collapse of the lung. Sometimes the bronchial lesion acts as a check valve. It allows air to go in but during expiration the bronchus narrows and air cannot come out. When large areas of air are obstructed by this mechanism, one gets an obstructive emphysema with displacement of the heart and mediastinum. Obstructive emphysema is not common, and in our Unit during the past four years, we have had only four such lesions. Miller (1963) states that although a temporary phase of incomplete obstruction is not uncommon, and degrees of emphysema are seen on radiographic examination of the children under investigation, it is rare for a clinical illness to present with symptoms caused by the emphysema. Walber (1955) studying 538 children with primary tuberculosis of the lungs found only 7 cases with obstructive emphysema whereas nearly half had segmental lesion. The following is a case report of two cases of obstructive emphysema from our Unit.

Case No. 1: T.B.A., aged two years, was followed by the Outpatient Department because of a primary complex in the form of superior mediastinal lymph node enlargement due to tuberculous lymph nodes. While on Streptomycin and isoniazid, after 3 months of therapy, the patient developed sudden onset of dyspnoea with cyanosis. Physical examination revealed ballooning of the chest with a shift of the trachea to the right. The percussion note was hyperresonant and the air entry was diminished over the left chest with a few rhonchi. As the child was acutely ill and dyspnoeic an emergency bronchoscopy was necessary, and at bronchoscopy tuberculous granulation tissue was found growing into the left main bronchus. The latter was removed though the bronchoscope and a repeat film of the chest taken soon after showed reexpansion of the lungs (Figs. 7a & 7b). There was however, mediastinal enlargement due to enlarged tuberculous lymph node. This child has been followed for over two years and the child is clinically well and radiologically quite well.

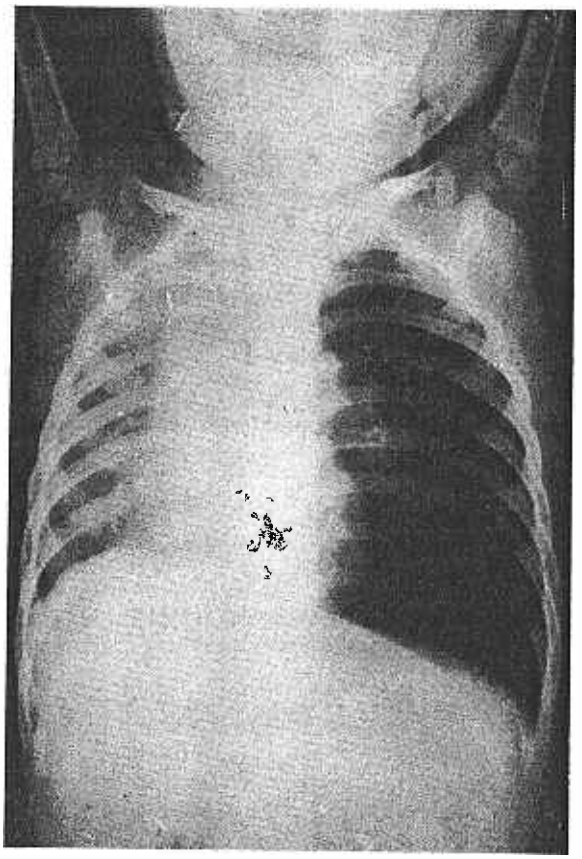

Fig. 7a. Note the radiolucent area on the left side due to an obstructive emphysema with mediastinal shift.

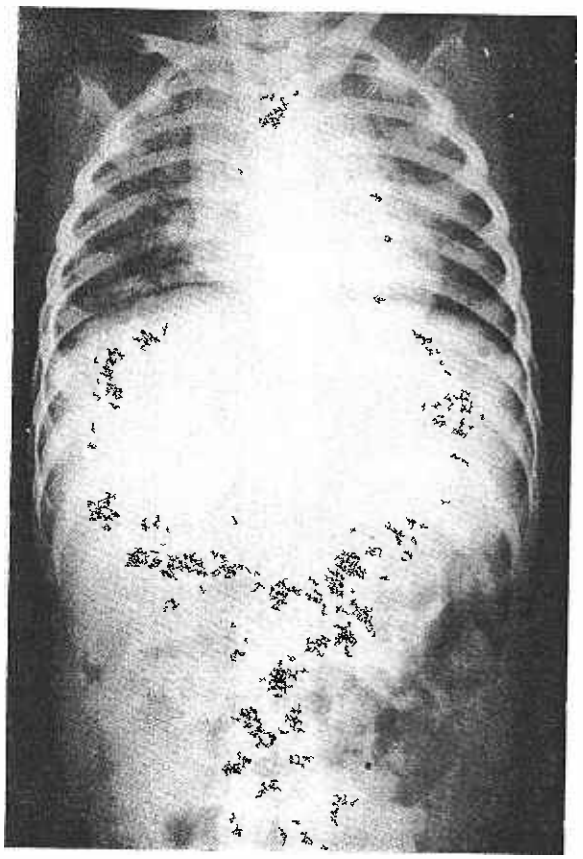

Fig. 7b. Re-expansion of the lungs after bronchoscopy.

Case No. 2: The second patient was a case of obstructive emphysema who was not acutely dyspnoeic but clinically showed a hyperresonant percussion note with diminished air entry over the left lung. Radiologically the chest showed an obstructive emphysema of the left upper lobe with a compression of the left lower lobe (Fig. 8). No emergency bronchoscopy was required in this case, and the child responded well to antituberculous drugs after a period of two years.

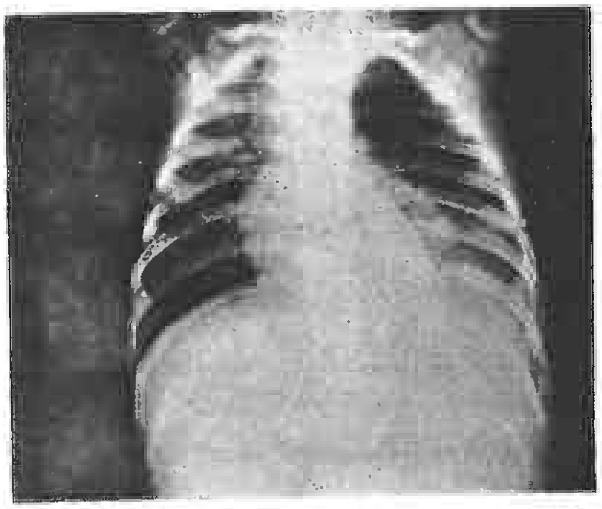

Fig. 8. Left upper lobe constructive emphysema with left lower lobe collapse. Right paratracheal lymph nodes enlargement.

CAVITATION OF LUNG

Unlike adults where cavitation of the lung is common in the upper lobes, in children cavitation is rare. We have had only 3 such cases of primary tuberculosis with well defined cavities. The following radiograph was from a four-year old with persistent cough and a positive sputum on culture with bronchogenic tuberculosis of the lung. There was persistent bronchial breathing and the child responded after 2 years of antituberculous Streptomycin and I.N.H. (Figs. 9a, 9b).

CERVICAL LYMPHADENITIS

Lymphadenitis is an early complication of primary tuberculosis, occurring within the first six months of infection. The most common mode of infection is by way of lymph nodes. The right hilar lymph nodes are the first group to enlarge and this is followed by enlargement of the tracheobronchial lymph nodes and cervical lymph nodes so that when one sees a child with cervical adenitis, one is reminded of the fruits of a tree whose roots are deep below in the hilum. In the Bellevue Hospital for tuberculous children,

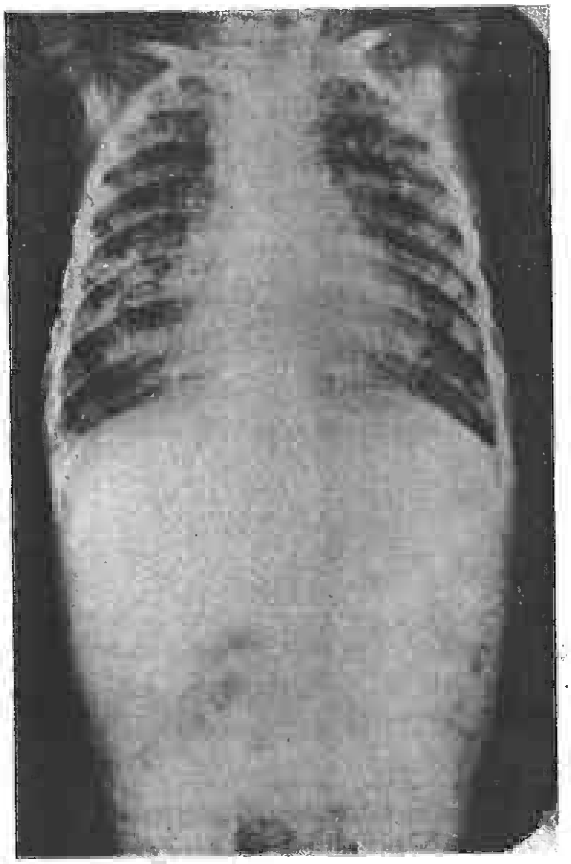

Fig. 9a. Bronchogenic tuberculosis with cavitation of the right lower lobe in a 4-year old child.

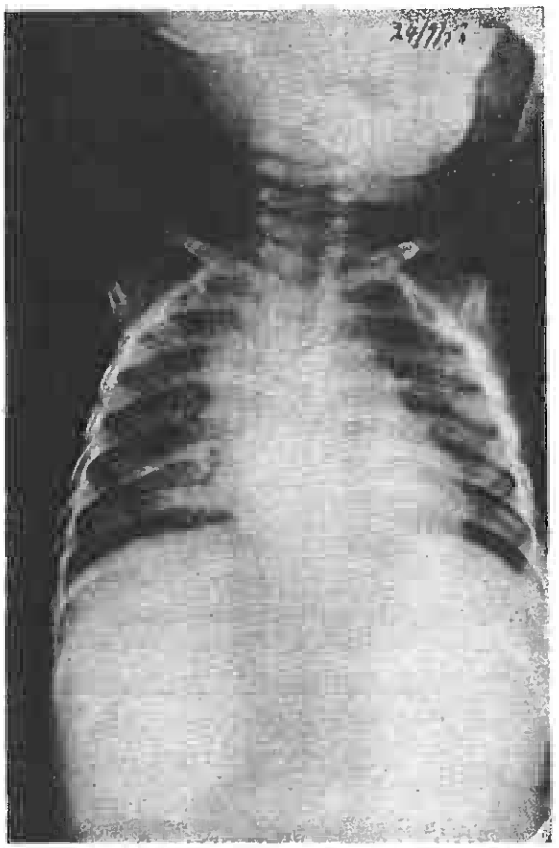

Fig. 9b. Clearing of the cavity after 2 years of antituberculous therapy.

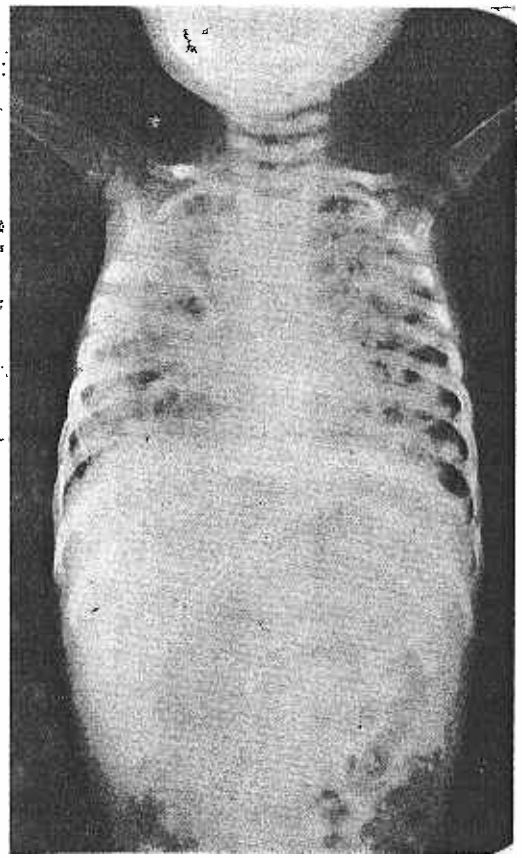

Fig. 10. Advanced bronchogenic tuberculosis in a two-year old child.

cervical adenitis was noted in 5% of their cases of primary complex. The diagnosis was confirmed by culture, biopsy, pathological examination and later development of calcification. The lymph nodes in the posterior triangle are most commonly involved. Tuberculous enlargement of the supraclavicular lymph nodes, axillary lymph nodes and submandibular lymph nodes are less frequent. The following are the figures which show the number of cases of cervical tuberculous adenitis, seen in our Department here.

Year	Tuberculous Adenitis	Primary Complexes
1956	19 cases	213 cases
1957	15 cases	177 cases
1958	10 cases	133 cases
1959	29 cases	101 cases
1960	26 cases	105 cases
1961	32 cases	77 cases
1962	15 cases	87 cases
1963	3 cases	53 cases
1964	7 cases	20 cases
1965	3 cases	10 cases

The mode of presentation varied. In some cases the child was well with no symptoms, in others the glands were noted only accidentally, while in others the glands were painless and firm. If the infection is recent, then the child is usually quite ill with a temperature of 104°F. The lymph nodes of the neck become easily noted because enlargement is rapid. The lymph nodes are tense and painful but not as painful as the lymph nodes of a pyogenic infection. There is a lot of periadenitis and oedema so that the lymph nodes often fuse together. This mode of onset is due to a hypersensitive reaction. With this mode of onset, early abscess formation is the rule. Most of the cases of tuberculous adenitis were treated with isoniazid and para-amino salicyclic acid as an outpatient unless the child is very ill when Streptomycin and isoniazid are used when the patient is warded. In recent years we have used corticosteroids to reduce the oedema especially the recent infection with acute onset group. The regression in size is remarkable. This is another example of the anti-allergic effects of corticosteroids in tuberculous therapy (Figs. 11 & 12).

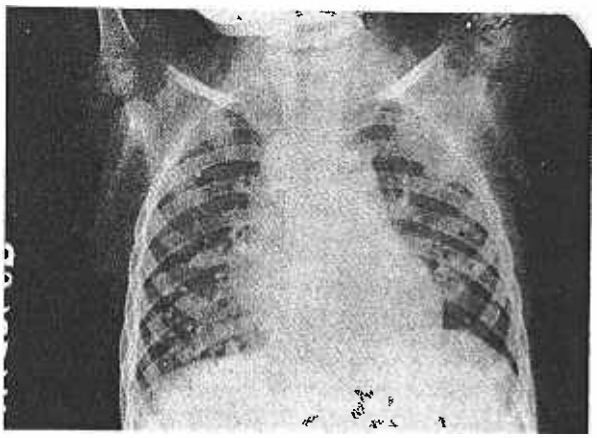

Fig. 11. A case of tuberculous lymph node enlargement of the neck, superior mediastinum. Note the large calcified lymph node in the superior mediastinum.

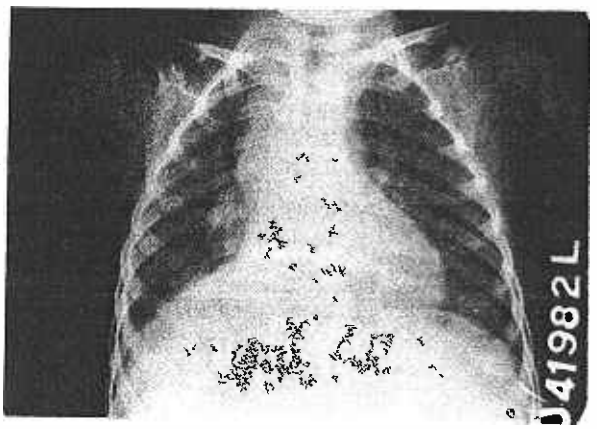

Fig. 12. Note the mediastinal enlargement in a 3-year old child due to tuberculous lymph nodes and collapse of the right lower lobe.

TUBERCULOSIS OF THE KIDNEY

Tuberculous infection of the kidney always arises by haematogenous spread from a distant active focus during the early stages of primary infection, or later from an active pulmonary or other lesion. It is also well recognised that tubercle bacilli can be recovered from the urine of children with recent primary infection although they do not show any clinical signs of tuberculous disease of the renal tract. Munro (1944) found tubercle bacilli in the urine of 37 of 189 children admitted to a children's sanatorium, and he considered that minute lesions must have been present in each case. Tubercle bacilli can also be recovered from the urine in many cases of miliary tuberculosis. Yet localised lesions of the kidney or urinary tract in children is rare, and this is because the incidence of renal tuberculosis is small compared with the number of children undergoing primary infection, and secondly, the symptoms of renal tuberculosis do not appear till at least 5 to 7 years after primary infection and are not likely to be seen very often before 10 years of age. From 1928 to 1946 Miller (1948) saw only 56 cases of tuberculosis of the kidney in 10,000 tuberculous children and only ten of them present as renal tuberculosis. In the records analysed at Newcastle there were only 9 cases of renal tuberculosis in 12 years among 1,200 children treated with chemotherapy. In our Department during the last 10 years we had only 4 cases of renal tuberculosis. All 4 cases were mistakenly admitted as acute nephritis and required further investigations because of persistent haematuria. Below is the radiograph of one of them which shows marked deformity of the right kidney due to tuberculosis. There were miliary mottlings of the lung. The urine on culture grew the tubercle bacilli (Figs. 13a & 13b).

CONCLUSION

Thus we see in spite of the varied clinical pictures of primary complexes that we see in the Unit, the incidence of primary complexes is rapidly on the decline. Factors which are responsible for the reduction of childhood tuberculous meningitis are also responsible for this reduction in childhood primary complexes, namely the introduction of B.C.G. vaccination, the mass X-ray campaign of adult tuberculosis, the introduction of potent antituberculous drugs like Streptomycin, isoniazid and para-amino salicylic acid and a very useful adjunct, Prednisolone and improved living and social conditions during the last few years.

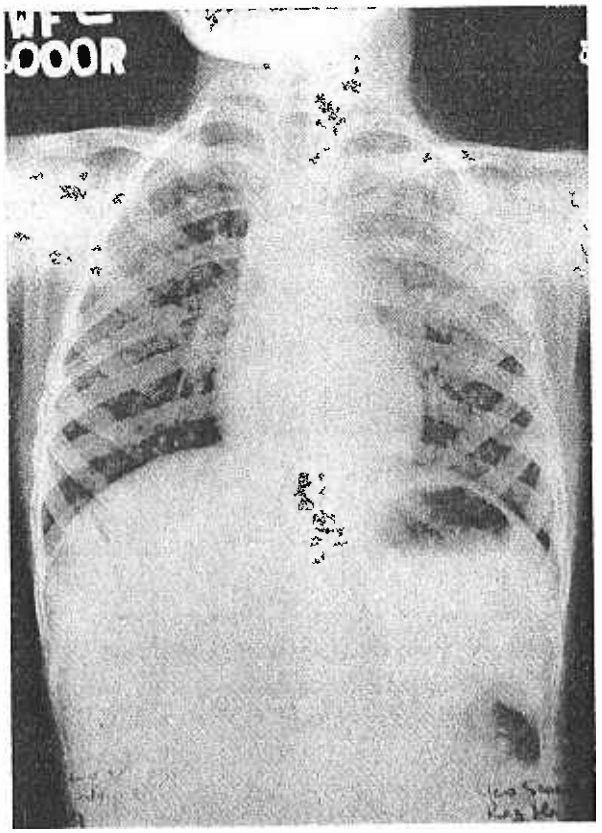

Fig. 13a. Radiograph of the chest in a case of renal tuberculosis. Note the miliary mottlings of the lung.

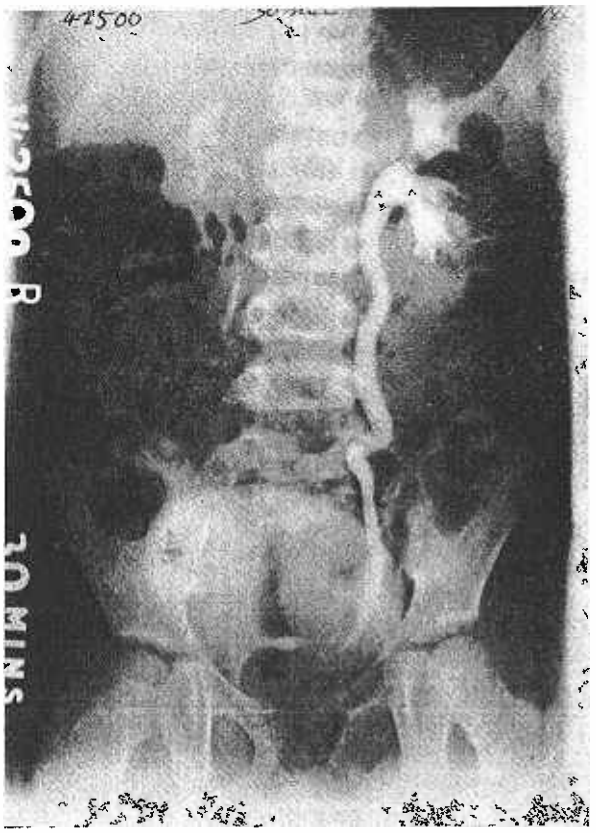

Fig. 13b. Picture of an intravenous pyelogram to show complete destruction of the right kidney due to renal tuberculosis.

REFERENCES

1. Illingworth, R.S. and Wright, T. (1948): "Tubercles of the Choroid", Brit. Med. Journ., 2, 365.
2. Lincoln, E.M. and Sewell, E.M. (1956): "Tuberculosis in Children", McGraw-Hill Book Company Inc., London.
3. Miller, F.J.W., Seal, R.M.E. and Taylor, M.D. (1963): "Tuberculosis in Childhood", J. & A. Churchill Ltd., London.
4. Muller, R.W. (1948): "Uber die Nierentuberkulose in kindersulter", Stuttgard Thieme; Quoted in Lancet, 1, 1,078, (1950).
5. Wallgren, A. (1937): "Uber das Ansteckungsvermogen der Kindertuberculose", Acta Paedia., 22, 230.