Tuberculosis in children in India-I

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INTRODUCTION

Tuberculosis is different in children. It involves many organs, instead of being the predominantly respiratory disease that it usually is in adults. Fortunately, it readily responds to treatment–if you diagnose it early enough and treat it for long enough! This is the problem. Unfortunately, tuberculosis causes such non-specific symptoms and signs, and you are so seldom able to isolate bacilli, that you may never be sure of the diagnosis. Even experts sometimes disagree. In India particularly, it is a disease of the poorest of the poor, but even in them it causes only a small proportion of their burden of morbidity. The great problem is to reach those infected.

Of every thousand Indians, seven children and about twenty adults have active tuberculosis, and five of these adults are sputum positive. Only about half the 9 million in the community at any one time are ever diagnosed, and of these only about 13% complete their treatment, so there is a huge pool of infectious cases, half a million of whom die each year. Fortunately, the incidence of tuberculosis among children reporting to hospital is slowly decreasing, probably largely due to improved coverage with BCG.

TUBERCULOSIS AND THE VILLAGE HEALTH WORKER

Children are infected by adults, not by one another. Tuberculosis in children is therefore due to the failure of present programmes to control the disease in adults, each of whom commonly spend thousands of rupees on private medical care. When a patient is eventually diagnosed, it is usually with a Rs 60 X-ray, and without his sputum being examined. Those patients who do seek government care are likely to be faced with a long journey to a primary health centre which has restricted hours and an irregular supply of drugs.

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Can village health workers do any better? The Mandwa and Malsiras projects show that they can. Even if these workers are only half literate, they can be taught the five early cardinal symptoms of tuberculosis, and can conveniently and successfully treat patients at home in the village. They can also give streptomycin injections safely, and there is no better health educator than a convinced village woman. In view of the dismal failure of the government programmes, village health workers probably hold the key to the successful control of tuberculosis in India—if we can only give them the chance!

THE PRIMARY COMPLEX AND ITS COMPLICATIONS

Inhaled bacilli usually lodge in the periphery of a child's lung where they cause a tuberculous lesion with giant cells and lymphocytes. They also spread to his hilar lymph nodes. This combination of a peripheral lung focus and enlarged hilar nodes is the 'primary complex'. It frequently causes no symptoms or signs, although you can often see enlarged nodes on an X-ray. Bacilli are also carried in his blood to distant sites, where they may cause miliary or meningeal disease immediately; or-they may lie dormant for years before they destroy his bones, joints or kidneys (Fig. 1).

His lung lesion usually heals and calcifies without causing symptoms, although a few bacilli may remain to become active in later life. But if he is unlucky his primary lesion fails to heal and any of the complications (Fig. 2) may follow. These are the children we are concerned with here.

Tuberculosis and malnutrition affect each other disastrously

Tuberculosis impairs a child's nutrition and may precipitate protein-energy malnutrition. Malnutrition depresses his tuberculin sensitivity and lowers his resistance to infection, so that he is at greater risk of disseminated disease. It also renders him more liable to other infections and lessens his inflammatory response. The younger and more malnourished he is, the more intercurrent infections he has, and the more likely it is that his tuberculous infection will progress and perhaps kill him.

THE DIAGNOSIS

It is usually easy in an adult who coughs up bloody sputum laden with bacilli. It is much more difficult in a child, and his history is all important. He becomes 'ill' over weeks rather than days, he does not eat, play, or smile normally, he loses weight and the curve on his weight chart flattens or falls. If he is lucky you see him at this stage. Usually you don't see him until he has fever and loss of weight, and often not until he has a cough, *but without bloody sputum*, which is very rare in children. Sometimes, he

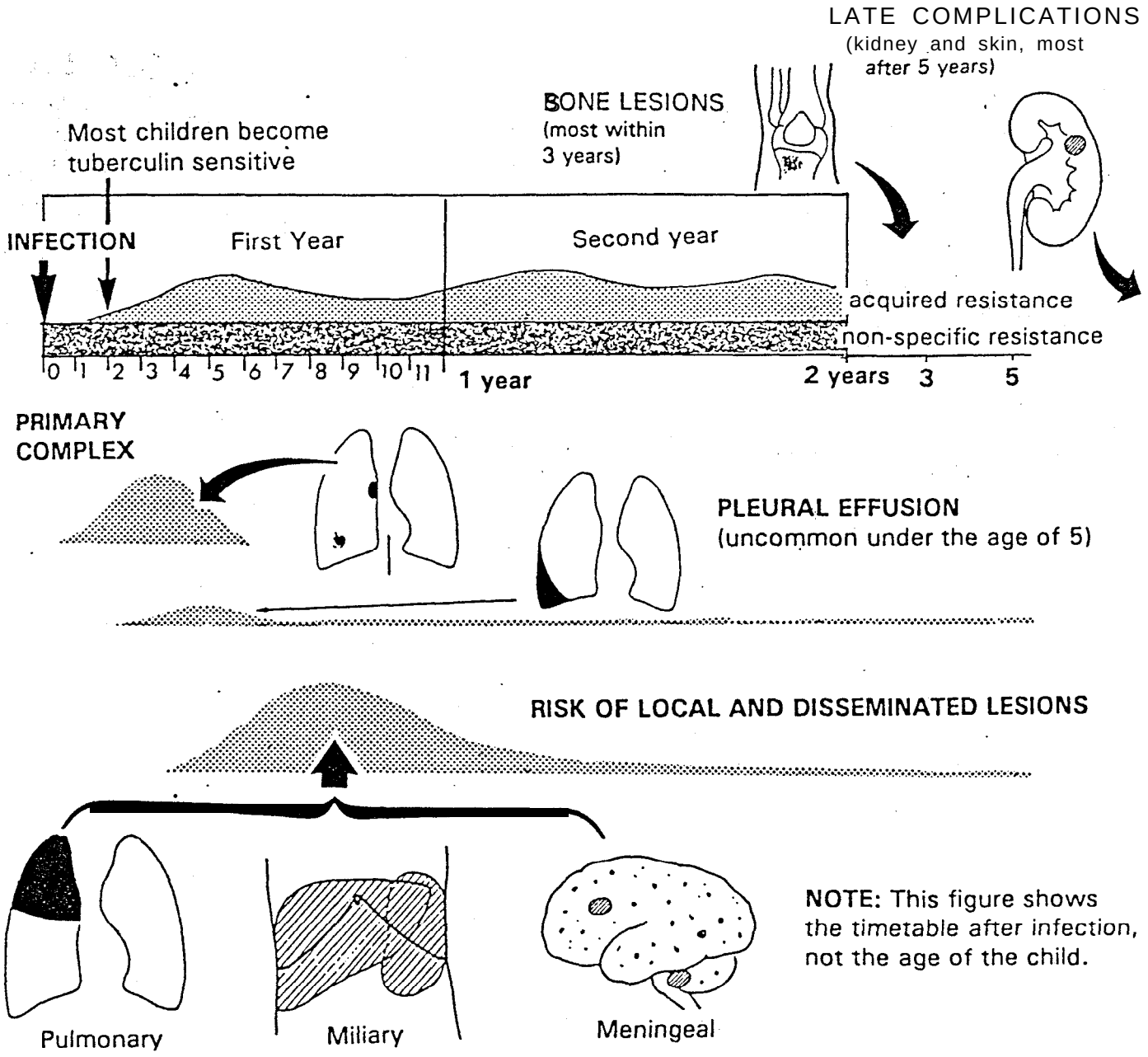

FIG 1. A timetable of primary untreated tuberculosis. Although pleural effusions are uncommon they often occur earlier than other local and disseminated lesions. If these are going to occur, 90% of them do so in the first two years.

merely 'fails to thrive'. Two extrapulmonary events: erythema nodosum, and phlyctenular conjunctivitis (see below) greatly assist the diagnosis. Erythema nodosum is sometimes said to be rare in India; although the redness of the nodules is not so easily seen in a dark skin-you will find it if you look.

When a child has tuberculosis, there is always an adult somewhere with a chronic cough. His grandmother perhaps? Do a tuberculin test (cheap) and, if possible, X-ray his chest (more expensive). You will find a tuberculin test and perhaps a BCG test so useful that anyone seeing sick children must be able to do both of them. The persistence of suspicious symptoms which fail to respond to symptomatic treatment, in the presence of a positive tuberculin test, a suspicious family history, and the absence of a BCG scar should make you suspicious enough to start treatment.

You will find that you have very few confirmed cases of tuberculosis, in whom the history, the physical signs and the X-rays make the diagnosis almost certain, and many more probable and suspected ones. Often, all you can do is to make a good guess and resign yourself to the fact that you may never be quite sure. Use the criteria described later, and always keep tuberculosis in mind. Remember it *in any child with malaise or altered behaviour,* and try to treat him early and adequately. Try to walk the uneasy tightrope between unnecessary deaths and unnecessary treatment. If a child you suspect of having tuberculosis recovers, he may be responding to treatment, or he may be recovering spontaneously from another disease.

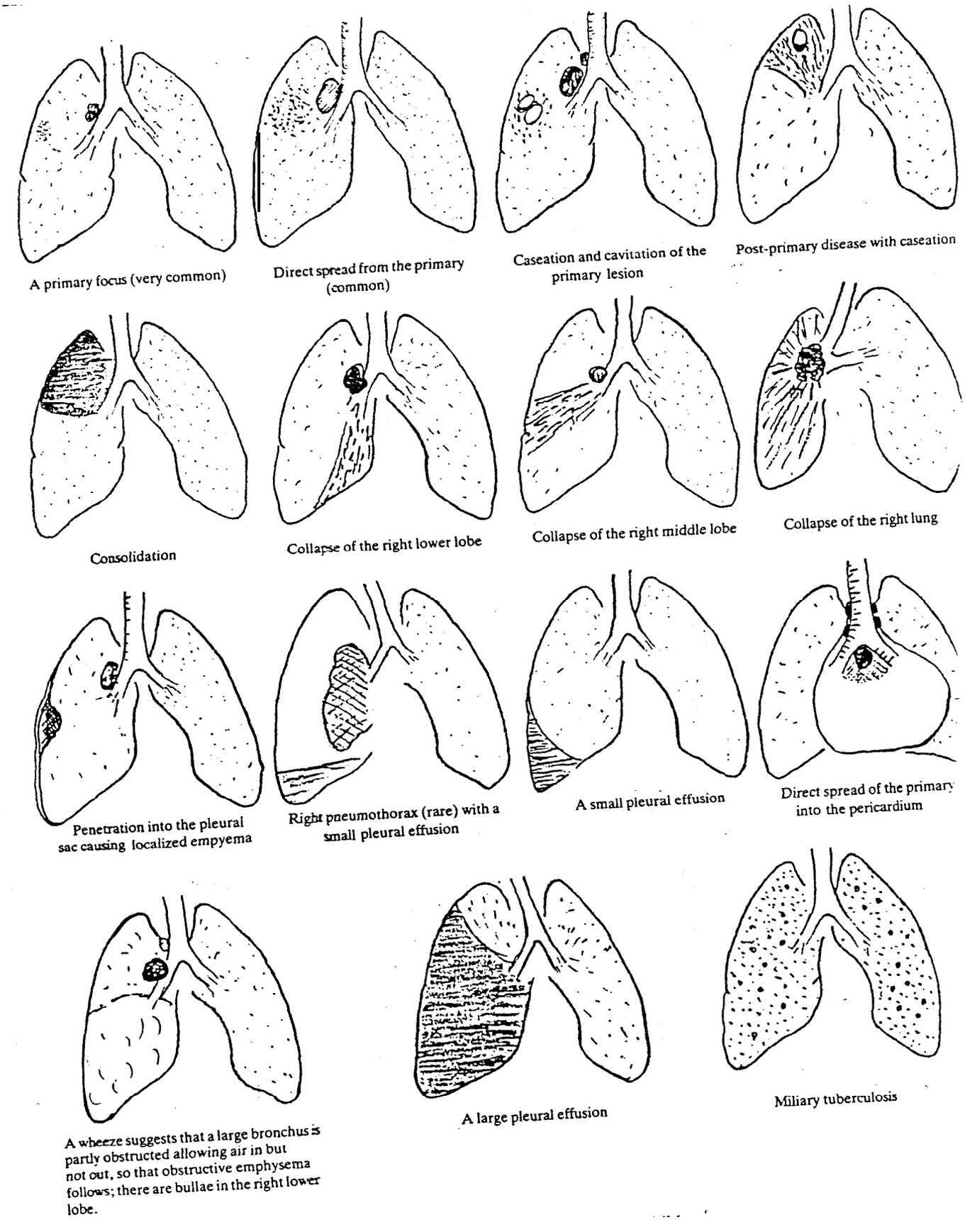

FIG 2. Pulmonary tuberculosis in children

Often, you will never really know. Even if he does not have it, you have not harmed him-provided you have not overlooked something even more serious. Chemotherapy usually allows you to treat him as an outpatient at home.

Miliary tuberculosis and tuberculous meningitis

These are the two most severe forms of the disease and need intensive treatment with three or four drugs. Some children have both and it is common, for example, for a child with tuberculous meningitis to have a few tubercles on his choroid. Miliary disease resembles uncomplicated pulmonary tuberculosis, except that the child is more ill and has miliary lesions on his chest X-ray; often he also has an enlarged liver and spleen and choroidal tubercles-if you can find them. If he is very ill, suspect miliary spread, even if this is not very obvious. Rarely, miliary disease persists for months.

Tuberculin sensitivity

Six to eight weeks after a child's primary infection, an increase in cell-mediated immunity makes him sensitive to tuberculin which you can test for by using the Mantoux or Heaf tests. Unfortunately, this sensitivity is reduced by malnutrition and infection. In both tests an extract of tubercle bacilli is injected into his skin which is observed for an inflammatory response 48-72 hours later. A reaction to the tuberculin test means that he has been infected by tubercle bacilli; *it does not necessarily mean that he has active tuberculosis.*

Unfortunately, there is no certain way of distinguishing tuberculin reactions caused by BCG vaccine from those caused by tuberculosis, but the stronger a tuberculin test is the more likely it is to be caused by tuberculosis. So you will have to use other criteria to decide, particularly his family history (see below).

The Mantoux test uses dilute PPD (purified protein derivative) and is more sensitive. Its sensitivity can be varied by changing the dilution of the PPD, and it gives you more information. But it is more difficult to do properly, and the PPD for it needs to be refrigerated and diluted; once diluted it has to be used within a given time. The Mantoux test also needs skill and practice, so it is mainly used in specialist centres and is less suited to a generalist in a busy clinic.

The Heaf test uses concentrated PPD and a metal gun with several small needles; some models have a magnetic head and a detachable metal plate with small pointed blades. A Heaf gun is much easier to use, and it is less important to refrigerate the PPD for it. The Heaf test is commonly used by paediatricians, and should be used by all general practitioners. Unfortunately, Heaf guns are imported and expensive (East Healthcare, Sandy Lane, Littlemore, Oxford, O4 5JT, UK, Rupees 3750 plus carriage). We hope that this article will stimulate an Indian firm to make these guns cheaply, and more Pharmaceutical companies to distribute the PPD for them.

The BCG test

If you inject a child who already has a tubcrculous infection with BCG, it produces an accelerated (earlier) reaction at the site of injection which is distinctive enough to be useful for diagnosis. Although it is only 50% specific, it is 50–80% sensitive and can be positive even when there is malnutrition, overwhelming infection, measles or whooping cough. It is often positive when the Mantoux test is negative, and is particularly useful in children under 5 years.

The BCG test is still controversial, since adequate trials have not been done. It works by giving a much larger dose of antigen (the equivalent of about 20 TU) than a standard tuberculin test (1 IU). *It does not replace the tuberculin test* and should only be done when this is negative. A child needs to be seen three times, so its value in outpatients is low. If you do it in place of a tuberculin test, you will get too many false-positives and some strongly positive children will get a severe reaction. Its value in diagnosing tuberculous meningitis is unknown. If a child has AIDS there is also the possibility of disseminated BCG infection.

TO PREVENT TUBERCULOSIS IN CHILDHOOD, FIND AND TREAT ALL ADULTS

PREVENTION depends on finding and effective/y treating open adult cases. The value of BCG vaccine is controversial: (1) It will not protect a child if he has already been infected by a known case (he needs prophylaxis, see below). (2) It may not protect him from an overwhelming source of infection, although there is good evidence that it provides a useful degree of protection against miliary and meningeal disease.

HISTORY. A child with tuberculosis can present in several ways: (1) He may become 'ill' over weeks, rather than days, with loss of weight and sometimes a cough, fever or a wheeze. (2) He may have pneumonia which fails to respond to ordinary antibiotics. (3) He may have recurrent *non-wheezy* respiratory infections (recurrent wheezy infections suggest asthmatic bronchitis, especially if there is weight gain, rather than tuberculosis). (4) He may fail to recover and gain weight after measles or whooping cough.

More specific clues are less common: (1) A persistent spasmodic cough suggests tuberculous bronchial involvement or bronchopneumonia. (2) Shortness of breath suggests that a large volume of his lung has collapsed or is displaced. (3) A persistent wheeze suggests that one of his bronchi is partly obstructed, so that it lets air in but not out; a few such children progress to obstructive emphysema. (4) Persistent cough, fever and weight loss suggest post-primary disease or miliary spread.

HIS FAMILY HISTORY is crucial, so look for a contact. Tuberculosis has a social stigma, so persist in your questioning. Examine the sputum of any suspicious contacts and, if possible, X-ray their chests.

EXAMINATION often shows nothing except that he is not well. Look for: (1) Signs of malnutrition? (2) Chest signs? Is his trachea central? Does his chest expand equally? Unequal movement? Dullness to percussion (if this is marked it suggests an effusion). Signs of pneumonia in his middle or lower lobes? Listen for bronchial breathing (consolidation), crackles and wheezes. (3) Enlarged, non-tender, firm, multiple, matted, supraclavicular or cervical nodes, especially with satellite glands round a larger one, and sometimes with fluctuation, skin involvement and a discharge? Biopsy is seldom practical, but is desirable if enlarged nodes do not respond to treatment or grow (see below). (4) Fever? (5) Choroidal tubercles? Look for these if he is more than mildly ill. They lie near blood vessels; are paler than his retina, and are yellow and later white. As they heal a black ring of choroid appears. Search his whole retina; you may find a single tubercle far out at the periphery. If necessary dilate his pupils and give him a brief anaesthetic to complete the examination. (6) Enlargement of his liver and spleen suggests miliary disease, but is only marginally useful because this has so many other causes. (7) A BCG scar makes the diagnosis of tuberculosis less likely, but does not exclude it.

Two early but uncommon extrapulmonary events at the time of the primary infection which considerably strengthen the diagnosis are:

Phlyctenular conjunctivitis causes a 1–2 mm red spot at the junction between his conjunctiva and cornea. This becomes pale during a few days, and then ulcerates at its centre. A group of dilated vessels forms near it on his conjunctiva. Similar nodules may appear on his cornea. If his conjunctiva is involved, he only feels irritation. If his cornea is involved, he feels pain, he cannot look at a light and keeps his eye closed. Unless there is obvious local sepsis, assume that he has tuberculosis and treat him for it. You can give him local steroids for his painful cornea, if you are sure he does not have herpes simplex or a bacterial ulcer.

Erythema nodosum causes tender red swellings on the front of his legs. He can walk without pain, but does not like the swellings being touched. Streptococcal infection is usually the commonest cause, but tuberculosis probably comes second. Accept it as the cause if: (1) He has no clear history of a sore throat. (2) He has any abnormality on his chest X-ray. (3) His Mantoux is > 8 mm or becomes so 3 weeks later. He may be so sensitive to tuberculin that he gets a severe local or general reaction when you test him, so start with a tenth of the normal concentration.

WEIGHT CHART. He is usually underweight and has a flat or falling curve. If he does not have a weight chart, start one.

TUBERCULIN TESTS

THE MANTOUX TEST. There is great confusion about tuberculin units (TU) and international units (IU), but the volume injected is always 0.1 ml. 1 U of RT23 is equal to 3 IU of PPD-S (both are purified fractions of tuberculin). The original diagnostic dose was 1 U of RT23 and this is still the standard dose in India. All reagents are diluted in a dilute solution of Tween 80 which prevents the adsorption of the reagent onto the glass. Use a 1 ml glass non-leaking tuberculin syringe with a short, fine 26-gauge needle.

Avoid previous test sites. Place the needle, bevel uppermost, flat on the front of the child's forearm on the opposite side from his BCG immunization. Slowly, and as superficially as possible, inject 1 TU in 0.1 ml intradermally, while slightly stretching the skin in the opposite direction. Don't touch the plunger until the eye of the needle is inserted. Raise a 6-9 mm wheal like the skin of an orange. Three to four days later run your finger over his forearms. Preferably read the tests at 48 hours as well; if they increase from 48 to 72 hours they are more likely to be positive. Use a small ruler to measure the largest diameter of the palpable induration *(not the erythema);* this ranges from 0 to 30 mm.

CAUTION! (1) Keep tuberculin solutions refrigerated. (2) Use a special syringe for Mantoux testing only. (3) Record the diameter of the induration in two directions and average them; the harder the induration the more likely it is to be important. (4) Inject the tuberculin *intradermally* to raise a wheal (small lump), not subcutaneously. (5) Do the test precisely. (6) When you read and interpret the test look for a BCG scar on his left shoulder. (7) If you repeat the test, allow for an earlier one increasing the subsequent response.

THE HEAF TEST. Use a Heaf gun and PPD 2 mg/ml. Refrigerate the PPD and use it within a month of opening an ampoule. Use a small glass rod to place a drop of the reagent on his forearm and spread it over a circle of 1 cm. Cock and fire the gun through this. The six small needles will carry a little PPD into his skin. Read the test in 72 hours and report its grade as in Fig. 3. Heaf grades I and II correspond to Mantoux reactions of less than 6 mm. Grades III and IV correspond to reactions of more than 6 mm. The traditional method of disinfecting the head was to press it onto a spirit soaked pad and flame it. Since this may not be sufficient to kill HIV, disposable end-plates should now be used; unfortunately these are more expensive.

THE RESPONSE TO TUBERCULIN IS INCREASED BY: (1) Increasing the dose of tuberculin. This is not a linear relationship; doubling the dose less than doubles the induration. (2) Previous exposure to non-tuberculous mycobacteria. This is common in many areas but produces only small responses of less than 8 mm. (3) Previous BCG vaccination. Under 3 years this may produce a Mantoux reaction of 6-8 mm, over 3 year: it is usually 8-12 mm.

> 20 mm , severe 3+. This is followed by a pustule at 5-8 days which heals with a scab at 10 to 15 days. Some workers consider induration of >10 mm in 24-48 hours as positive.

Normal or negative test. Normally, when a child does not have tuberculosis, there is no reaction at 48-72 hours, a papule with induration at 2 weeks, a pustule at 4-6 weeks, and healing with a scab at 7-10 weeks.

CAUTION! (1) The acceleration of the reaction is important, not its size. (2) Previous BCG immunization can give a 1+ reaction, especially within one year. (3) This is a useful additional test but not confirmatory by itself.

X-PAY CHANGES can be difficult to interpret, and often do not correlate with a child's tuberculin test, so do not rely on them too much. He may have X-ray changes, but no symptoms or signs. If he has symptoms and signs he will probably have X-ray changes also, but these may be minimal. *Persistence of a shadow for 2 to 3 weeks is more useful than a shadow on a single film;* most pyogenic pneumonia will have started improving by then. If a shadow persists and a child is ill, the sooner antitubercular chemotherapy starts the better, especially if he is young.

You may see various combinations of: (1) Loss of translucency of his lung fields (infiltration, consolidation or collapse). (2) Hilar adenitis (almost always, but often difficult to interpret). (3) The common combination is hilar adenitis with infiltration, consolidation or collapse: this is the progressive primary complex. (4) Mediastinal adenitis (less common but more serious). (5) Increased translucency in part of a lung feld (unusual, a difficult sign) due to obstructive emphysema, often accompanied by hilar adenitis. (6) Infiltration with a cavity, and typically a positive sputum, is unusual before puberty; this occurs earlier in girls who have cavities at a younger age. (7) Widespread fluffy opacities (bronchopneumonia). (8) Miliary mottling of his lung fields (unusual) may be difficult to see and the film must be good; look carefully. Take a penetrated (very black) film and shine a bright light (a powerful pocket torch will do) through it, looking especially at the lateral intercostal spaces. If his immune response is depressed, he may be unable to develop enough inflammatory response to disseminated disease to enable you to see it as miliary mottling on his chest X-ray, although you should be able to see larger lesions. If you are inexperienced, the only X-ray lesions that you can say with any certainty are tuberculous are a primary complex and miliary mottling. Usually, his X-ray will merely look 'suspicious'.

CAUTION! (1) A normal X-ray does not exclude a primary complex (the lesion may be hidden behind his hear-t), nor does an abnormal X-ray always confirm it (the abnormality may not be tuberculous). (2) Not all cavities or pleural effusions are tuberculous.

BACTERIOLOGY is more difficult in children, but if you can send specimens for examination, do. Gastric lavage is unpleasant even in trained hands, so only do it when absolutely necessary. Aspirate his stomach *early in the morning as he awakens,* before a meal and before the secretions that have entered his stomach during the night have passed onwards. If they are not going to be processed immediately, neutralize them with a buffer, or his gastric acid will destroy the bacilli.

DIAGNOSIS. Most children show the major criteria together with a varying number of the supporting ones.

The major criteria are the following symptoms not responding to non-tuberculosis treatment, and lasting more than a fortnight or even a month: (1) fever, (2) loss of weight or failure to gain weight for 2-3 months, (3) anorexia, and (4) repeated respiratory infections.

The supporting criteria are: (1) A strongly positive tuberculin or BCG test (see above). (2) A positive family history. (3) A cough (not marked in babies, see below). (4) A history of measles or pertussis at the start of symptoms. (5) Severe malnutrition. (6) No previous immunization with BCG (this does not protect completely). (7) Enlarged lymph nodes.

If he is severely malnourished and fails to respond to an adequate diet, consider the possibility of tuberculosis, regardless of his tuberculin test, which may be negative. Try a therapeutic trial. *This is a very important presentation.*

If he is an infant, the diagnosis is particularly difficult, because he may be too young to develop a proper cough.

If his chest X-ray is negative, but he has symptoms suggestive of tuberculosis and a positive tuberculin test (more than 10 mm), treat him.

THE MAIN DIFFERENTIAL DIAGNOSES are the many causes of cough, weight loss and fever in children. Most of these are short lasting. If his symptoms persist, and especially if they last more than a week or two, in the presence of the supporting criteria, suspect tuberculosis. If he has a pyogenic infection, his chest X-ray will usually be normal at 2-3 weeks, so be sure to X-ray him at this time. Tuberculosis does not cause diarrhoea (unless it is almost terminal), whereas many of the differential diagnoses do. If AIDS is common, it is an important differential diagnosis: enquire about diarrhoea and ask after his mother's health.

The differential diagnosis of X-ray changes include: pneumonia, pyogenic lung abscess, empyema, spontaneous pneumothorax, inhaled foreign body and tropical eosinophilia.

THERAPEUTIC TRIAL. If you still suspect tuberculosis after two weeks of investigation, and perhaps treatment for other diseases (commonly pneumonia needing 2 weeks treatment with an antibiotic such as penicillin and chloramphenicol), start a trial of antituberculous chemotherapy. Most deaths in hospitals occur in children who are already moribund on admission, so a delay of 2 weeks before starting to treat tuberculosis is reasonable.

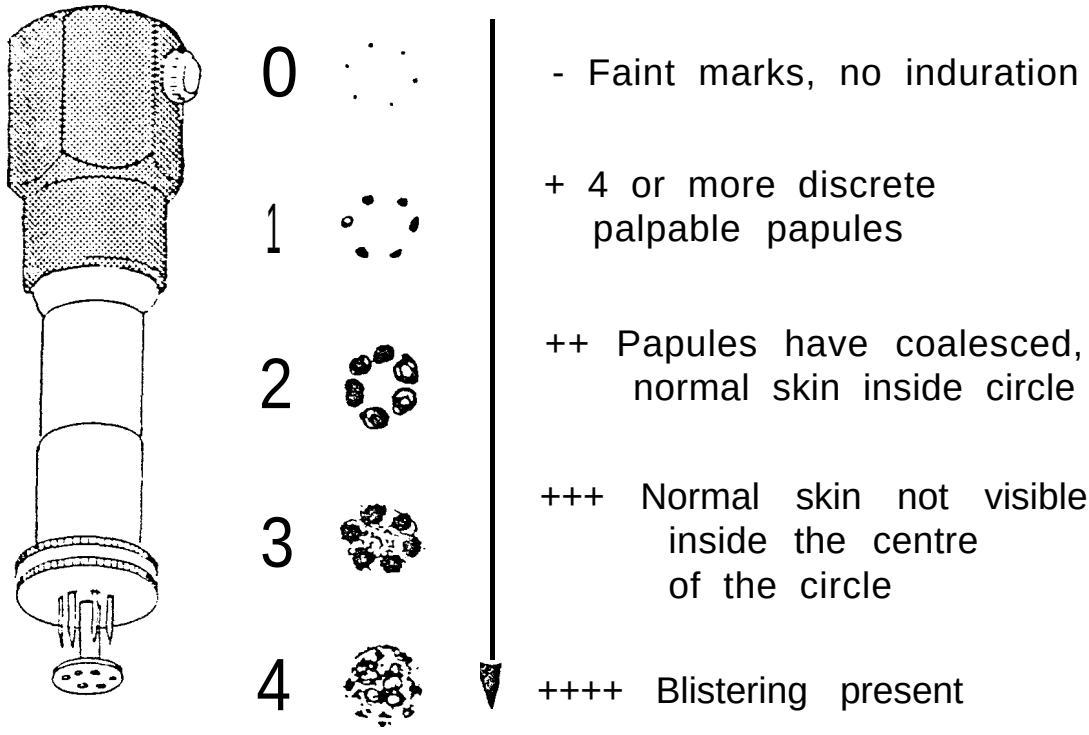

FIG 3. The Heaf test is a practical way of doing the tuberculin test. Read the test in 72 hours. Heaf Grades I and II correspond to Mantoux reactions of less than 6 mm. Grades III and IV correspond to reactions of more than 6 mm

THE RESPONSE TO TUBERCULIN IS REDUCED BY: (1) Current or recent measles, and to a small extent, by a recent measles vaccine. (2) Malnutrition, particularly severe malnutrition; kwashiorkor depresses it greatly. Even so, nutritional status and tuberculin sensitivity are often poorly correlated. (3) Overwhelming tuberculous infection, especially miliary or meningeal tuberculosis. (4) Prolonged or high dose steroid treatment. (5) HIV infection. (6) improper technique, such as massaging the injection site, injecting too little, or injecting subcutaneously.

INTERPRETING THE TUBERCULIN TEST. Look for a BCG scar. There is no reliable way of distinguishing tuberculin reactions caused by tuberculosis from those caused by BCG. So consider: (1) The child's age; the younger he is the more useful the test; any positive test under the age of 5 is very important. (2) His nutritional status. (3) His previous illnesses, especially recent measles and perhaps malaria. (4) Steroid treatment reducing reactivity. (5) Previous BCG vaccination. (6) The dose of tuberculin you inject. (7) The size of the reaction, the clinical setting (is he so ill that he might have miliary or meningeal tuberculosis?) and his contact history.

If he is well nourished, a Mantoux reaction of: (1) Less than 7 mm is negative and is probably due to non-tuberculous mycobacteria or BCG. (2) 8-15 mm is doubtful positive and could be caused by tuberculosis *or* BCG. It is not diagnostically helpful unless he is very young (<3 years) and you are, already suspicious for other reasons, when even a moderate positive may mean that he has active tuberculosis. (3) 16-20 mm suggests infection if he has not had BCG, and is of doubtful importance if he has. It is more valuable if he is young. (4) More than 20 mm suggests tuberculosis whatever his BCG status, especially if he is young.

If he is severely malnourished, a Mantoux reaction of more than 5 mm suggests tuberculosis. If he has kwashiorkor a response of any size may be very significant.

If his tuberculin test is negative, he may still have tuberculosis because: (1) Your technique may have been poor. (2) You may have tested him too soon after exposure. (3) He may be immunodeficient because of: (a) severe protein-energy malnutrition, (b) measles up to 5 weeks after the disappearance of the rash, or some other infections, (c) overwhelming infection (miliary or meningeal disease), especially if he is moribund, (d) steroid treatment, (e) AIDS. Repeat the test, he may take a year to convert. If you repeat the test too often, testing will itself increase positivity.

CAUTION! (1) A negative tuberculin test does not exclude tuberculosis, nor does a positive one by itself prove active disease! (2) Don't overdiagnose tuberculosis. The significance of a strongly positive test in an otherwise healthy child. is described below. (3) Tuberculin tests are almost valueless in diagnosing tuberculosis in adults. (4) The ESR is useless in tuberculosis.

THE BCG TEST. One of us would only do this if a child is Heaf or Mantoux negative, if he is positive he may get a severe reaction to BCG. Inject BCG as usual. See the child at 48-72 hours. Read the test as follows:

Accelerated or positive test. Induration of 5-9 mm, mild 1 +. Induration 10-20 mm, moderate 2+. Induration

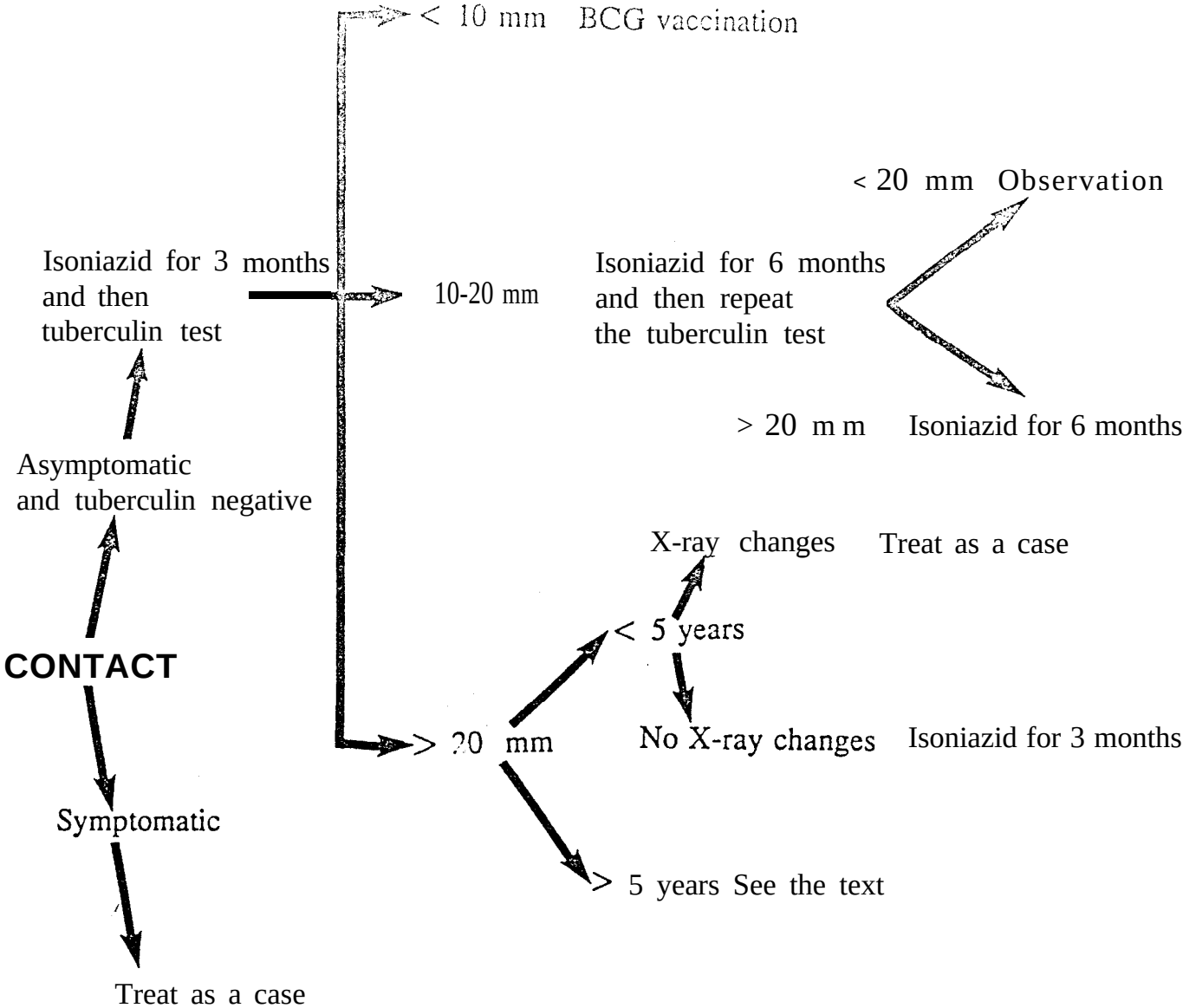

FIG 4. Managing the contacts of an infectious case of tuberculosis. Chemoprophylaxis is most important, especially if he is young, became it will almost certainly ensure that he remains well. Unfortunately, 60% of poor Indian children may not complete even 3 months of prophylaxis

Don't start a trial lightly, and don't do it too often. Monitor his temperature and activity closely during the trial. Assess his response by his weight gain, improved appetite and activity, and remission of fever. If he is not much improved in a month reassess him.

CAUTION! There is no substitute for careful assessment and follow up.

MANAGEMENT. You can usually treat him at home. Admit him if he is very toxic and ill, you are unhappy about compliance, and also perhaps to start treatment and educate his parents. Do your best to improve his diet. If he is anaemic, try to correct this.

CHEMOPROPHYLAXIS. If a contact of open tuberculosis has symptoms, he is a case (see below) and needs the full treatment. If he has no symptoms and is at high risk, he has a good chance of developing them, so he needs chemoprophylaxis. Give him isoniazid 5 mg/kg/24 hrs for several months, if possible for 6 to 9 months.

There is little point in giving him a second drug unless you can supervise him daily and there is much isoniazid resistance locally. If so, give him isoniazid and rifampicin 10 mg/kg/24 hrs for 3 months, then on alternate days for 3 months, then twice weekly for 3 months.

Chemoprophylaxis is most important, especially if he is *young* (<3 years), because it will almost certainly ensure that he remains well. Unfortunately, 60% of poor Indian children may not complete even 3 months of prophylaxis, but what little they do get may still be useful. In some parts of India 30% of patients have primary isoniazid resistance and there is also some primary thiacetazone resistance. Other drug combinations are

often impractical, so the choice may be isoniazid or nothing. Fortunately, if he is symptom-free, there is little risk of resistance to it emerging.

If you diagnose tuberculosis in one child, others may have it too. So ask his parents about any other children who may also have been infected. Do they have BCG scars? Only some may be ill, but all should have prophylaxis for at least 3 months. Start it immediately, even if you cannot immediately examine them all. You can often find sick children by questioning their parents. Tuberculin test all child contacts.

If a contact is asymptomatic and is tuberculin negative, he may be infected, but his tuberculin test may not yet have had time to become positive. Monitor him and especially his weight carefully. Ideally, give him chemoprophylaxis for 3 months. X-ray him and tuberculin test him again at 3 months. Unfortunately, few healthy children return for investigation, so the following suggestions are ideal rather than practical. If at 3 months he is still tuberculin negative or is only weakly positive (<10 mm), give him BCG and stop the isoniazid, or it will kill the BCG. If he is intermediate (10-20 mm) give him isoniazid for a further 6 months and repeat the tuberculin test. If he is strongly positive (>20 mm) see below.

If a contact is asymptomatic with a clear chest X-ray, but is strongly tuberculin positive, what you do depends in theory on his age: (1) If he is under 5 give him chemoprophylaxis with isoniazid for at least 6 months. If he has X-ray changes, treat him as a case, even though he has no symptoms, because miliary and meningeal spread are so common at this age, especially if he is malnourished. (2) If he is over 5, the risk of miliary and meningeal spread is less. So observe him and only treat him if he develops tuberculosis. If his family is sensible and advantaged, they can watch and weigh him and bring him at the first sign of trouble. Chemoprophylaxis is not usually recommended over the age of 5.

If a contact is symptomatic and especially if he has **no BCG scar,** investigate him. He may well be an active case and need chemotherapy (Fig. 4).

CAUTION! (1) Daily isoniazid with some other drug for a year is the only proved regime for subclinical tuberculosis; but many experts are satisfied with 6 months treatment. (2) Can you find and treat the adult who infected him?

THE EXPLANATION to the family is vital; try to win their support. Explain to his mother and father, and his grandmother, that: (1) Tuberculosis is not a disease that car: be quickly cured by a few injections. (2) He may need treatment for at least a year. (3) He needs *their continued help during this time.*

(The concluding part of this article will appear in Vol. 5, No. 6.)