THE DIAGNOSIS AND TREATMENT

— OF —

EARLY PULMONARY TUBERCULOSIS

— IN —

CHILDREN.

N.A.COWARD.

THESIS.

"The Diagnosis and Treatment of Early Pulmonary Tuberculosis in Children".

Recent investigations, conducted both in the post mortem room and by means of the various Tuberculin tests on the living make it apparent that Tuberculosis in Children, is a much commoner form of disease, and occurs at an earlier age, than was formerly imagined.

Harbitz shewed in 1905 in his pathological statistics for Christiana that 43% of all children who died, had foci of tuberculosis. Hamburger and Sluka's figures for Vienna were 40%; Comby's for Paris 38.5%.[1].

In 1907, Wolff Eisner described the method for using the eye reaction to Tuberculin as a test for Tuberculosis - modified by Calmette - and now known as "Calmette's test". This was soon followed by Von Pirquet's cutaneous and Moro's Inunction tests.[1].

The application of these to large groups of children was the means of shewing that a much larger proportion is infected with Tuberculosis than had hitherto been suspected from post mortem findings. More careful search was then made by means of the microscope, and by the inoculation of guinea pigs, in order to elucidate the matter further.

Hamburger records figures shewing the prevalence of Tuberculosis in children according to post mortem findings, in the case of patients whose deaths were not due to Tuberculosis. Thus he found that between 5 - 6 years 34% shewed signs post mortem of Tuberculosis, between 7 and 10 years 35%, and between 11and 14 years, no less than 53%.[1].

Hamburger and Monti in a diagram representing the result of the cutaneous, reinforced by the subcutaneous, test on 509 school children in Vienna, shew that positive reactions were obtained in over 90% of children in the years 10 - 14, the line rising steadily from birth, at 3 years of age over 30% being positive, and at 6 years over 60%.[2.]

These figures have been confirmed by observers elsewhere, by Mantoux for Paris, by Nothmann for Dusseldorf, and by Gangliopner for Prague.[1.]

Hamburger and Monti shewed too that the mortality rate of Tuberculosis among children during the first 2 months of life was 100 per cent, at the end of the first year 50%, at the end of the second year 20%, and from the fourth year onwards much less. Thus with regard to Tuberculosis children may be said to be very susceptible, but excepting the first two years of life, not so liable to fatal results.[2.]

The question therefore of the early diagnosis of Tuberculosis in Children is of added importance in the light of these investigations. Robert Hutchison says "Careful hospital statistics taken in various parts of the world, show that about one third of all children who die in hospital, die of Tuberculosis in one form or another; and further, in about 12% of the remaining cases, tubercle is present in a latent form."[3.]

We have seen that many cases who die from other cause than Tuberculosis, shew the signs of that disease on post mortem examination. These tubercular foci are in many cases found to be healed lesions, being small masses of fibrous or calcareous material. At one

time these healed lesions have been foci of active tubercular disease, only by the resistance of the tissues the disease has been eradicated, and healing induced.

The early detection of Tuberculosis in Children is essential, owing to its extremely rapid and fatal dissemination in some cases. With few signs during life, post mortem examination reveals tubercles in the liver, spleen and kidneys, and ulcers in the intestines, though the symptoms may have only pointed to pulmonary disease. There is, too, always the danger of Tubercular Meningitis and generalised Tuberculosis setting in.

Pulmonary Tuberculosis is in many cases insidious in onset, its symptoms often slight, its signs too often almost negative. Diagnosis, therefore, in such circumstances cannot be other than difficult. For a proper understanding of the disease, and as an essential toward diagnosis, we must study it with special regard to its

(a) Protecting influences and Predisposing causes.
(b) Sources of infection and ways of dissemination.
(c) Symptoms, with differential diagnosis.
(d) Signs, with methods of investigation.
(e) Methods of Treatment.

Queen Mary's Hospital contains 850 beds; of these over 500 are occupied by cases of Tuberculosis - 300 Surgical, 200 pulmonary. The material for this thesis has been obtained from 50 cases of Early Pulmonary Tuberculosis in one of the female phthisis blocks. Figures have been taken also from the material supplied by the other wards in the hospital.

The children admitted must be under 16 years of age. They are drawn from the various districts in the London County Council area. Children of the poorest class in the land, of parents who live in most unhygienic and in some cases insanitary surroundings whose family histories are of the worst, in them Tuberculosis could with good reason be expected. They are sent down with often very indefinite signs and symptoms, and a diagnosis is often only arrived at by a process of exclusion, after continued observation and investigation.

Age. While formerly Tuberculosis was a disease almost unsuspected before puberty, it is now regarded as practically the commonest disease affecting childhood. It is well established both from clinical observation and from experiments on animals that a foetus can be born of a tuberculous mother shewing well marked signs of tuberculosis, but so exceptional is such an event that for practical purposes congenital tuberculosis can be excluded altogether.[1]

Hamburger shewed from records of post mortem examinations made on children dying from other causes than tuberculosis, that from 0 - 6 months 0% contained evidence of that disease, from 7 - 12 months 45%, 1 - 2 years 17%, and 3 - 4 years 30%. There is thus evidence to shew that even in infancy tuberculosis is commonly found. According to the same observer the mortality rate among children infected during the first two months of life is 100%, at 12 months 50%, at the end of the second year 20%. These figures do not tell us the percentage of pulmonary infection, as compared with lesions in other parts.

We know that many infants die from general abdominal and meningitic Tuberculosis, but the pulmonary type found in adults is very rare. Even tuberculosis of bronchial glands in very young children is by no means a common occurrence; at least that is the experience from post mortem examinations made on children at this hospital.

In the series of 50 cases of Early Pulmonary Tuberculosis selected, the oldest were 15 years of age, and the youngest 2. The average age was 9 years 1 month, only 1 case was 4 years old, but there were 10 cases 5 years of age.

From statistics taken from the other phthisis wards, of 217 cases admitted as Pulmonary Tuberculosis, the average age was 9 years 10 months. The youngest was 1 year 8 months - this child had a very marked Von Pirquet reaction with crackling rales all over the lungs; the next youngest with a similar condition was 2½ years old, and there was one of 3 years old with definite signs of pulmonary tuberculosis.

The numbers with their respective ages were

2	cases	- 4	years of age	on admission.
10	"	5	"	"
14	"	6	"	"
18	"	7	"	"
14	"	8	"	"
25	"	9	"	"
17	"	10	"	"
29	"	11	"	"
22	"	12	"	"
22	"	13	"	"
21	"	14	"	"
18	"	15	"	"
2	"	16	"	"

These numbers refer only to the ages on admission; it is impossible to say at what age symptoms of the disease were present, but in many cases so indefinite were the symptoms and the signs, that it is fairly safe to conclude that the disease was not of very long standing. Therefore the figures are to some extent of value. It will be seen from them that Pulmonary Tuberculosis is rare before the age of 5, and after this eleven and upwards are the most common years.

These figures compare very well with those of Hamburger. As seen before, from post mortems made on children whose deaths were not due to Tuberculosis, he found signs of this disease, 34% in patients 5 - 6 years of age, 35% in those 7 - 10; 53% in those 11 - 14, though of course his figures did not allude to pulmonary tuberculosis in particular.

Protecting Influences.

Pulmonary Tuberculosis in children seldom occurs where another tuberculous lesion is present, such as tuberculosis of the spine, hip joint, knee joint, or ankle joint, and though in some cases the lungs may be affected secondarily to glands in the neck, in others the latter act is a protection to the lungs, and on their removal pulmonary disease has been known to ensue. The presence of such lesions acts as a protection against pulmonary disease by auto-inoculation, whereby the tissues learn to defend themselves against the organism and thus acquire immunity.

Among the 200 patients with Pulmonary Tuberculosis in hospital at present, only one is the subject

of surgical tuberculosis. This case is one of spinal caries in the cervical region and the disease appears to have spread directly from the spine to the lungs. Again, among the 300 odd cases of Surgical Tuberculosis, including cases of Tubercular hips, spines, knee and ankle joints, dactylitis and lupus, there is only the solitary case quoted, that has signs of pulmonary disease. There are also many cases of Otorrhoea, tubercular in origin, that is to say those with positive Von Pirquet reactions who clear up with tuberculin, who are not included.

Morbus Cordis has long been held to be antagonistic to pulmonary tuberculosis, due to the increased venosity of the blood in the pulmonary system.[4] Dr. Kidd says "All diseases of the heart which bring about a passive congestion of the lungs confer a certain degree of protection against pulmonary tuberculosis, but in the words of Peacock this opposition certainly in no degree amounts to an ancompatibility". Cases have been recorded by various observers where the two diseases have been coexistent, but the condition is undoubtedly rare and there is not at the present an instance among the patients in this hospital.

Predisposing Causes.

The influence of heredity has of late years been somewhat called in question. The transmission from mother to foetus, though capable of demonstration in animals, is extremely rare in man. The number of cases who can give a definite family history of phthisis has been said to be 30%.[5] It is probable that the direct inheritance of the disease is of decidedly

subordinate importance to extrauterine infection, however acquired, that is to say children become infected directly by their parents or relatives, rather than inherit the disease. It is possible, however, that the children of phthisical parents have lessened powers of resistance in their blood to the tubercle bacillus, and therefore are more likely to contract disease if they come within range of infection. This fact, coupled with the likelihood of infection when living in contact with phthisical relations, would magnify the danger of tuberculosis for such individuals. It is difficult to explain otherwise how children of tubercular parents living in different parts of the world, have repeatedly all become infected by the tubercle bacillus, and it would seem to point to some inherent weakness, or lessened powers of resistance.

Among etiological factors, those of great importance are, overcrowding, ill ventilation, insanitary and unhygienic surroundings improper and insufficient feeding; all of these presuppose lack of sunlight, fresh air, and general cleanliness, and consequently by lowering the body's resistance render the child more liable to infection by the tubercle bacillus, or to other illnesses which pave the way.

Measles, whooping cough, and influenza, have long been held to be predisposing causes to pulmonary tuberculosis, measles and influenza may either light up an already existing lesion, or by causing certain changes in the bronchial and pulmonary epithelium so lower their power of resistance as to lay them open to the invasion of tubercle.

Measles is ofen complicated by broncho-pneumonia, and broncho-pneumonia followed by empyema, while the latter is frequently the forerunner of a fibroid phthisis. Measles too may be directly followed or complicated by an acute tubercular broncho-pneumonia, or by causing catarrh of the lungs or bronchitis, may infect the bronchial glands also, leading to their enlargement and subsequent infection by the tubercle bacillus. Hilus Tuberculosis follows.

Of our 50 cases, all had had measles, four fifths whooping cough, none influenza. Measles would appear to be extraordinarily common, more than 95% of cases admitted to the hospital are said to have contracted it, so that unless pulmonary tuberculosis be a direct sequela or complication, the disease can hardly be said to be predisposing. In one only of our cases was this infection the direct preceding cause.

Pleurisy was at one time supposed to be a factor favouring the onset of phthisis. In such cases, the original pleurisy was doubtless tubercular itself, and secondary to the disease in the lung substance. There is no reason to suppose that pleurisy in itself leads to phthisis; on the other hand by compression it may, by keeping the lung at rest, aid in its recovery. After Bauer's operation of injecting nitrogen into the pleural cavity, an effusion often forms. This is rarely withdrawn, but is used to aid the process of compression. Many physicians leave tubercular pleural effusions to absorb, unless they become embarrassing to the heart's action, thereby affording the affected lung complete rest for a time.

It is rare for croupous pneumonia to end in phthisis.

Phthisis is stated to be very prevalent among members of the Jewish race. Out of our 50 cases, two were of Hebrew extraction, but out of the 200 cases in hospital, only four, which does not appear to be an excessive proportion, considering the number of children admitted from the East End of London.

Sources and Channels of Infection.

It is generally admitted that Tuberculosis in children tends to spread by lymphatic channels, and is therefore nearly always primarily in lymphatic glands. It is to the glands cervical, thoracic and abdominal, that we should turn to find out the source of infection.

The Paths of Infections are said to be

1. Inhalation Tuberculosis.

(a) The tubercle bacillus passes either into the bronchi or alveoli to produce a primary tuberculosis there, or, having traversed the walls of the respiratory surface, they lodge in the tracheo-bronchial glands. From there they gain access to lymph and bloodvessels, along which they are borne to the lung, where they settle and further develop.

(b) The bacillus settles on the mucous membrane of nose, pharynx, or tonsil either in inspired air or with food. From there, the bacillus passes to the lymphatic glands in the neck, and ultimately enters the blood stream, to be carried to the lungs.

2. Ingestion - Tuberculosis.

Bacilli are swallowed with food, mucus, saliva,

etc. and after escaping from the stomach, they produce either a primary intestinal tuberculosis, or more frequently, travense the intestinal epithelium and become lodged in the mesenteric glands, where they are carried by the thoracic duct and blood channels to the lungs.

3. Inoculation Tuberculosis.

Bacilli pass through skin or uro-genital mucous membrane, gain lymphatic glands and are blood or lymph borne to the lungs.

4. Congenital Tuberculosis.

Bacilli penetrate the ovum from tuberculous sperm or - more probably - infection of the fetus takes place from bacilli which have traversed the placental circulation from a tuberculous mother.

The two last methods of infection are so rare as to be of but little account. Discussion, therefore, has been confined to the two first channels of invasion, and has resulted in the forming of two separate schools of opinion:-

Firstly, those who consider Inhalation the primary cause of infection.

Secondly, the other school who give precedence to the ingestion theory.

The former hold that the bacilli are inhaled and carried to the lung, or being entangled in the tonsil, the cervical or supraclavicular glands are secondarily infected and thence the lungs, or the bronchial glands are infected resulting in hilus tuberculosis.

Koch held that pulmonary tuberculosis was due to inhalation infection. First it was thought that dried particles of sputum were the means of the dissemination of the tubercle bacillus (Cornel), it has been proved, however, that it is only under exceptional circumstances that phthisical sputum becomes dry enough to form dust and that it is in the moist state, as minute droplets, in coughing, sneezing or even speaking that the tubercle bacilli are inhaled (Flugge)[4]

Von Behring, however, doubted the accuracy of the inhalation theory, maintaining that it was by ingestion and principally by the taking of milk from tuberculous cows, that the disease was spread: that the abdominal glands became primarily the seat of enlargement and infection by the tubercle bacillus, and from those glands the bacilli gained access to the blood stream and were so carried to the lungs. He considered that the intestine acted like a large pore filter. He explained the fact that consumption preponderates when the period of childhood is passed, by presupposing a very lengthy latent period amounting to months, years or even decades. Weleminsky holds that the bacilli are carried direct from the intestine to the bronchial glands, but this has been denied by other observers (Hart, Kitamusa, etc.)[4].

The consensus of opinion on both sides is great and no definite opinion has been reached as yet.

Dr. Kidd sums up the situation in favour of aerogenic infection, for the following reasons.

1. The great dissemination of tuberculous sputum from multitudes of consumptives.

2. The early anatomical lesions in the lungs.
3. That very minute doses induce tuberculosis when inhaled, the lung being apparently the most easily affected of all organs.

There is too the vexed question as to whether the bacillus of infection is human or bovine in origin. Gaffky's clinic after exhaustive experiments estimated that out of 400 cases of tuberculosis in children investigated by inoculation and subinoculation of guinea pigs and rabbits, 98.68% were of human, and only 1.32% of bovine origin.[4].

The Royal Commission in investigating 133 cases of tuberculosis in children, found 33 of bovine and 100 of human origin.

It appears then that the bacillus responsible for tuberculosis in children is in the great majority of cases of human origin, the probable source of infection in young children being in the home itself (Schlossmann)[4]

According to Still the three channels of infection are[6].

1. Through the lung. 2. Through the intestine.
3. Through the ear.

In 269 post mortem examinations on tuberculous children under 12, the channel of infection could be determined in 216, and among these 216, 138, i.e. 63.8% infection appeared to have entered through the lungs, in 63, i.e. in 29.1%, through the intestine, and in 15 through the ear.

In 100 cases in children in the infancy period - when milk infection would be expected to be greatest - Dr Still found that the proportion of intestinal in-

fection was less. Among 100 infants (up to 2 years) in whom the primary channel of infection could be ascertained, 65 appeared to be due to pulmonary infection, only 22 to intestinal. Again in 39 cases out of 50 under one year of age, 27 were infected through the respiratory tract, and only 5 through the intestine, in the remaining 7 it was through the ear.

Dr. Carr at the Chelsea Children's Hospital in 120 autopsies on tuberculous children found 65.8% with primary thoracic infection, only 16.7% shewed evidence of primary abdominal infection.

Dr. Shennan at the Royal Hospital for Sick Children, Edinburgh, out of 355 cases of tuberculosis was able to determine the channel of infection in 331, in 67.07% this was respiratory, in 28.1% it was alimentary.

Dr. Guthrie at Paddington Green and Dr.Northrupp in America both confirm these observations.

The question then as to whether the infection travels by the pulmonary or abdominal routes, is at present unsettled; while the general opinion as to whether the bacillus of tubercle is of human or bovine strain, seems to favour the former.

None of our cases shew primary enlargement of cervical glands, and there are no evidences of scars of operation wounds or of discharging sinuses. Among the 200 cases of phthisis in the hospital, enlarged cervical glands are found in 2 patients only, and in one of these the adenitis developed after the lung condition. The existence of enlarged tonsils is very common, but we shall consider this more fully later.

Symptoms.

In the early stages of Pulmonary Tuberculosis the symptoms are often very few and very indefinite. The history of the symptoms depends on the complaints of the child - which are frequently slight - and on the observations of the parents.

The usual complaints are that the child is lackadaisical; lacks go, 'lolls about', is easily tired; the appetite is poor - though this by no means always - is rather breathless on running about and is growing thinner - though here too such is not always the case; for often the child does not lose weight, so much as fail to put it on, and very often too there are no signs of breathlessness. Generally speaking, however, those are the usual symptoms, of which the most marked are the 'want of joy' in living, and the 'always tired' feeling, and we find in adults also, in what is known as the pretuberculous stage that these feelings of depression and fatigue are very much in evidence.

The four great symptoms of pulmonary tuberculosis in adults are Haemoptysis, Night Sweats, and most important cough and loss of weight.

Haemoptysis is rare in children, and as a first sign of tubercle - as in adults - is very uncommon. In the later stages of phthisis, it is not so uncommon, though it is very rarely an immediate cause of death. More often it is due to whooping cough or to pulmonary congestion in advanced heart disease.

Night sweats of Phthisis are also uncommon in children but they are not of much account in diagnosis in the young. Still[6] says "Children sweat very easily,

especially when the health is depressed: rickets, debility after any acute illness, anaemia from any cause may be ample explanation of profuse sweating at night. The presence of night sweats is, I think, of no value whatever in the diagnosis of tubercle in children" - we find them very rarely present in the cases here, and feel that they are of little diagnostic value, except as a symptom of constitutional debility.

Cough may or may not be present. It seems to be less common than is generally held. Out of 50 cases, 14 on admission complained of cough, or were found to cough by the sister in charge, and in 6 of these the cough was slight. It is usually worse at night or first thing on waking in the morning. Taylor says[7] "It is mostly easy at first, sometimes not much more than a clearing of the throat, becoming harder and more painful in the later stages, and with extensive cavities it occurs in prolonged attacks."

Sometimes the cough is paroxysmal in type, and suggestive of whooping cough, said to be due to enlargement of the bronchial glands pressing on the trachea, or on the recurrent laryngeal nerve.

Many cases of whooping cough which are supposed to drag on for months are probably cases which may have begun as whooping cough, but which are now suffering from secondary enlargement of bronchial glands.

Dr. Murray Leslie in his article on Peribronchial or Hilus Tuburculosis writes "In a large proportion of cases asthma in children is really due to reflex or direct irritation from enlarged bronchial glands and peribronchial thickening and infiltration".

On X raying such patients "In some cases large glandular masses have been discovered, particularly when viewed from the right and left lateral aspects. Occasionally the thoracic nerves may become involved in the hilus thickening, particularly the recurrent laryngeal branch of the vagus, and such nerve compression may have an important influence in the production of the asthmatic attacks, although in children of a nervous diathesis the mere irritation and compression of the bronchial tube may of itself be sufficient to induce asthma."

Thus we see that prolonged attacks of whooping cough and asthma in children may be due to reflex irritation from enlarged thoracic glands. Much more rarely paroxysms of coughing of a similar nature are due to enlargement of the thymus gland. It may be in some of these cases that adenoid growths in the naso-pharynx keep up the irritation, either by permitting cold, unfiltered, mouth-inhaled air to irritate the sensitive tracheal and bronchial mucous membrane, or by reason of discharge from the naso-pharynx be the actual cause of the enlargement of the bronchial glands; but we shall refer to this point again later.

None of our cases shewed signs of paroxysmal coughing.

Expectoration in the early stages is rarely present but is probably not so rare as was once thought. Children very frequently swallow their sputum, instead of spitting it out into sputum jars. Hence it is that we find such frequent implication of the abdominal glands, which are often not primary, due to the ingestion of milk from

tubercular cows, but secondary to the swallowing of tuberculous sputum. Ulceration of the intestine too may be due to the same cause.

Dr.Emmott Holt[6] has shewn that it is possible to obtain sputum from even very young children, and to verify the diagnosis by a bacteriological examination even in infants. He excites a cough by irritating the pharynx by a small piece of muslin held on artery forceps. The secretion is coughed up into the pharynx and caught on the muslin; in this way Tubercle Bacilli were detected in the sputum in 80% of cases examined at the Babies Hospital, New York, where most of the cases were under two years of age. We may say that we personally have not had very much success with this method, which we have tried on several occasions.

Of our series 12 have had expectoration at different times. These sputa have been tested several times each for Tubercle bacilli, but they have been found in 3 cases only; in more advanced cases the bacillus is easily detected. Usually polymorphs and large numbers of cocci are seen. In no case was the Antiformin method used.

The presence of expectoration in large quantity is of serious import, as it usually denotes that the disease is no longer 'closed', but that secondary infection has occurred. Shreds of tissue in the sputum denote destruction of lung tissue.

Cough is a serious matter, as it leads to the auto-inoculation of the patient; either by the actual muscular exertion involved in the act, or by dissemination of the bacilli into the blood stream, or into other

parts of the lungs. If a patient develops one, his temperature almost immediately rises, or the already existing swing on the chart is increased, that is if it be of sufficient vehemence. Cough, like excessive exercise results in auto-inoculation and a consequent fever and depression for the time being of the patient's powers of resistence. Tuberculin certainly seems to aid in relieving the condition, and at present among these selected cases only one child has a cough - and that only occasionally - while two have some expectoration.

Loss of weight. These children are usually on admission anaemic, badly developed and thin, their general condition poor. If they are well nourished at first sight, the flesh is usually found to be flabby, and the child unhealthy looking. In a typical case of tuberculosis, wasting is one of the first and most valuable symptoms. The fact that the child is growing thinner soon becomes apparent and attracts the attention of the parents. On examination the child is found to be thin, especially about the chest, where the ribs are rather prominent and the expansion poor. Another feature to be noticed is the inability of the patients to put on weight when placed under favourable circumstances. A child, who persistently remains at the same weight level, when properly fed and living under the best conditions who is not definitely tubercular, is likely to be the subject of a hidden focus of the disease somewhere. For instance there is a girl aged 11, who has been at Queen Mary's Hospital for over two years, and during that time, despite the open air treatment, and general attention to diet, bowels, etc. she

has not gained an ounce in weight. This child runs an irregular temperature, the swing is not very marked, and is, I think, a case of Early Pulmonary Tuberculosis. There are many others in whom for long intervals there has been no gain in weight where tuberculin treatment has often effected a marked improvement.

Several "weight for age" charts appear to err very much on the side of excess for any given age. Several children examined and found to be well nourished, were yet according to the weights on the chart, over a stone under the normal. So much depends upon the height and build of the child. A small child, small in build, to reach the requisite weight would need to be grossly fat, while a tall, largely built one, is often only just properly or even under nourished at its so called proper weight.

On admission, out of 44 patients, 22 were said to have a 'very poor' or 'poor' general nutrition, 13 'very fair' or only 'fair' and 9 to be 'well nourished'

The Appetite is said to be capricious and a dislike for fatty things a prominent feature. We have not noticed such symptoms here, owing probably to the fact that the patients have not been accustomed before admission to good feeding, and secondly to the open air existence on balconies. Certainly the open air treatment owes a measure of its success to its marked improvement in the appetite.

In one case frequent vomiting after meals was a symptom at different times. An interval of freedom from these attacks would follow, only for them to recur again. This patient was very neurotic, a Jewess, and the condition improved in time. An aper-

ient regularly given will usually stop vomiting of this type.

The symptoms then of early disease are few and indefinite. In my cases, Haemoptysis occured in none, night sweats were recorded in 3 cases, cough occured in 14 and expectoration in 12:. the appetite in the great majority was quite good, vomiting in one case, general nutrition very poor in 22, fair in 13, and good in 9. Practically all had a feeling of tiredness, and lack of go, and some few, mental lassitude, a difficulty in fixing the attention for any length of time, or making any concentrated effort. Let us now consider the signs in the early stages of the malady.

Signs.

The Tubercular diathesis. Various minor characteristics have been attributed to subjects with a predisposition to phthises, viz. pretty, fair complexion, long eye-lashes, downy hair on the back between the scapulae, large joints, fine spun hair, blotches of erythema upon the skin on very slight pressure or irritation. Often the tuberculous child is possessed of considerable personal beauty, at other times it is of the scrofulous type of William Jenner's classification, where the skin is coarse and the complexion dull and muddy.

Chest Configuration. Two types of chest are said to shew proclivity to phthisis and are known as 'phthinoid'.[9]

1. The alar. 2. The flat.

The former indicates a type where the vertebral borders of the scapulae stand out boldly, and

the shoulders droop, and is associated with a long neck and prominent throat. The latter type is due to loss of the normal convexity of the sternum - the sternum is less distant from the vertebral column than normal. This type is often associated with the alar. The former is comparatively common, but by no means an unfailing accompaniment of phthisis.

The chest development in a great proportion of cases is deficient and the expansion extremely poor, associated with deficient air entry. This lack of good expansion, is accounted for in some by the presence of adenoid growths in the naso-pharynx with very much enlarged tonsils, but this does not account for a large proportion, where no enlargement can be found. It is well known that the Tubercle bacillus attacks the most airless parts of the lungs and consequently those least well aerated with blood, viz:. the apices, anterior borders, etc. and it is interesting to find in these patients a marked poorness of expansion, and so loss of blood aeration. In our 50 cases, the chest expansion on admission was recorded as 'Good' in 2 cases, 'Fair' in 10, and 'Poor' or 'Very Poor' in 38. A very striking improvement can be obtained by appropriate breathing exercises, and not only does the actual expansion increase, but coupled with the ordinary exercises and drilling, the chest at rest enlarges by an inch or more in a short time. This diminution in expansion, unless due to the presence of enlarged adenoid growth, probably indicates deficient muscular development. In a normal child from 10 - 14 years of age, the average chest expansion is about 2 inches or more - it varies to some extent. In

these children with poor muscular development, the expansion is often less than an inch, and the breath sounds are weak and difficult to hear on auscultation. The practice of breathing exercises every day cannot be too highly recommended; coupled with arm, leg and body drill, or gymnastics. Some of these children do not understand the art of breathing, and need educating, just as babies have to be taught how to walk. Their education in this matter leads to a better blood supply to the lungs and improves the circulation generally, the tissues of the body are better oxygenated and so perform their several functions more satisfactorily, the general nutrition benefits by a better blood supply and the child puts on flesh; by Swedish drill and gymnastics the muscles become better developed, and the circulation is further improved.

Temperature. With regard to Temperature children with Pulmonary Tuberculosis may be divided into 3 classes.

1. These with definite signs of disease in the lungs with Temperature.

2. Those with definite signs of pulmonary disease without Temperature.

3. Those with no signs of pulmonary disease with Temperature.

In 50 cases - 25 had definite though early signs of Pulmonary Tuberculosis, of these 23 had temperatures which were irregular, 2 had normal ones. In all cases, except one the signs were quite well marked but the disease was in an early stage. In the one case the disease was more advanced and had gone on to cavity formation.

25 had no definite signs in the lungs. All of these had irregular, swinging temperatures.

The temperature in these cases varied, but in all there was a swing of two degrees or more. The range in the swing did not differ in those with definite signs in the lungs, from that in the cases without. In some of both types the temperature would swing to 99, in others to 100, 101 or even 102. There were periods of intermission in some, when the temperature was fairly steady, to be followed after a time by the usual swing. One or two of these patients have been in Queen Mary's Hospital for over two years, with a slight swing - 97 . 99 - in the temperature the whole time, and the signs in the lungs have made very little progress. Furthermore such a condition is not always accompanied by progressive loss of weight, but by a gradual gain.

Long continued temperature of such a character without definite signs of lung involvement is said to be due to enlargement of the bronchial glands. Before assuming such a diagnosis we have tried firstly to exclude all the other likely causes of temperature of such long duration, and secondly to investigate the signs and symptoms of enlargement of the bronchial glands, with a view to proving their presence in these patients.

Possible causes of prolonged temperature with latent physical signs.

1. Pulmonary Tuberculosis with definite signs in the lungs - referred to later - Tuberculosis in general.
2. Pyorrhoea Alveolaris.
3. Enlarged tonsils and adenoid growths.
4. Intestinal Intoxication.

5. Bacilluria.

6 & 7. Possibly acute Rheumatism and Chronic Septicaemia.

The presence of fever presupposes some focus or foci in the body from which toxins are being absorbed and fever produced with more or less disturbance of the metabolic processes.

Horder in[10] 'Clinical Pathology in Practice' referring to fever says 'Localised Tuberculosis is probably the common cause of fever with latent physical signs, or with signs difficult to elicit - the possibility of the disease must always be borne in mind and it is useful to remember certain situations where this infection is apt to lead to more or less disturbance of general health with fever usually mild and remittent. These situations include the lung, pleura, peritoneum, lymphatic glands, kidneys, suprarenals, Fallopian tubes and spine'.

In none of my cases were signs of Tuberculosis in peritoneum, glands, kidneys or suprarenals, tubes and spine forthcoming. The abdomens of all were carefully examined, and their motions periodically inspected. The urine was examined once a week, and always on the day following a Tuberculin injection. There were no signs of tubal mischief - a negligible quantity at this period of life - or of spinal trouble.

2. Pyorrhoea Alveolaris is a not uncommon cause of long continued pyrexia. There is sometimes a subnormal temperature.[11] Many instances are recorded of continuous fever with wasting and general weakness, where the patients have recovered completely after the removal of carious teeth. Out of our 50 cases, 14

had many carious teeth, others only an occasional stump, in the majority they were sound. All have been treated by the dentist, the offending members removed, the mouth made aseptic, and kept so by the regular use of the tooth brush. Good teeth lead to good mastication, and the latter to good digestion - an essential for the tubercular.

3. Enlarged Adenoids and Tonsils. Up to the age of puberty enlarged tonsils and adenoids are extremely common, after this time they tend to disappear. Their enlargement leads to many troubles in childhood. The cause of the hypertrophy is obscure. Cold and foggy air has been said to irritate the mucous membrane of the nose and pharynx, for instance when children sleep in rooms with the windows open on foggy nights. The mucous membrane subsequently becomes inflamed and swollen and if the condition is severe, a chronic form may persist. This might account for some but certainly not for all. Once the adenoids have become enlarged, the tonsils, irritated by cold, unfiltered and mouth-inhaled air enlarge secondarily, thus the condition is aggravated. The child by breathing through the open mouth, acquires the typical, rather inane 'adenoid facies'. His speech is thickened, his ears discharge. He snores at night, is deaf, complains of headache and is a backward child, partly due to deafness, partly to mental lassitude. This is the description of a well marked case, there are all degrees of severity. Furthermore these tonsils are considered a favorite means of entrance for the Tubercle Bacillus, which becomes entangled in them and from them is carried by lymphatics to the cervical,

supraclavicular, or axillary groups of glands. These latter become the seats of tuberculous deposits, and from them infection spreads to the bronchial glands, and pulmonary tuberculosis follows. Dr. Carr[12] says 'There is no doubt that tubercular disease of the cervical glands is most likely to recur where the pharyngeal mucous membrane is in an unhealthy state, if the tonsils are enlarged and unhealthy, or if adenoids are present; in fact Tubercle bicilli have been found in the crypts of the tonsils' - Tubercular cervical glands are not commonly found in this hospital to be the primary source of Pulmonary disease, nor do we find old scars. We are inclined to think, if anything, that tubercular cervical glands act as a protection to further spread, and are distinctly chary about removing them. There are, in point of fact, out of 200 phthisical patients, only 2 cases with preceding tubercular glands of neck.

There are other ways in which enlarged tonsils and adenoid growths in the naso-pharynx can lead to tuberculosis, we believe.

Dr. Eustace Smith has described a condition in which there is a muco-purulent discharge from the naso-pharynx - due to adenoids - which is swallowed by the child, and which gives rise to gastritis and enteritis. The abdominal glands may be enlarged as a result of this purulent material finding its way into the intestine, and tubercular disease follow. From the tubercular abdominal glands the disease is finally taken by the blood stream to the bronchial glands, which enlarge and pulmonary tuberculosis is the result.

The hypertrophy of these glandular structures leads to deficient expansion of the lungs owing to the obstruction to the air entry. We have already seen that the tubercle bacillus tends to attack the most airless portions of the lungs, and also that these phthisical children nearly always have very poor development and expansion of the chest. Consequently any factor aggravating the already unsatisfactory condition is doubly harmful.

Adenoids may be an exciting cause of Asthma, which weakens the resisting powers of the lung. Furthermore, they may excite cough by their presence, and thus lead to harmful auto-inoculation.

It is doubtful if the condition of chronic glandular hypertrophy is, in itself, sufficient to cause an irregular temperature, without a follicular tonsillitis intervening. In some cases it may tend to keep up pyrexia from some other cause. There are vast numbers of children with this enlargement, who do not shew any irregularity in temperature. Out of 50 cases, 17 had enlarged tonsils and adenoids, 7 enlarged adenoids only. It is not at all uncommon to find a great reduction in the size of these tonsils, when the patient has lived for some time under good conditions, and operation is by no means always essential. Those that do not improve quickly under general hygienic treatment should, however, have the offending structures removed. A child with these growths is very liable to sore throats on slight provocation, which may go on to abscess formation in the peritonsillar tissues. Again if diphtheria is prevalent at any time - and it is extremely difficult to eliminate its presence from a

large community - these children are most likely to become infected.

The results of the removal by operation are extremely good. The child becomes brighter both in appearance and intelligent understanding, for prior to removal the brain is kept in a state of partial asphyxia from deficient oxygenation of the blood. Reflex disturbances, such as nocturnal terrors, asthmatic attacks, incontinence and ataxy disappear, otorrhoea if adenoids be the primary cause, clears up, and most marked of all the chest expansion by aid of breathing exercises improves wonderfully. Instruction in the art of breathing through the nose must be patiently persevered with for a long time, for the old habit of mouth breathing is difficult to check.

Intestinal Intoxication leads to temperature and is caused by: (a) Errors of diet - in young children. (b) Worms and intestinal parasites. (c) Severe constipation.

The toxins absorbed from the intestine are a potent cause of temperature, and in none more so than in children. An aperient very often leads to the reduction of an unexplained temperature, while unhealthy stools are probably the most common cause of fever in childhood.

Once a week in the case of the older children the motions are examined with regard to their consistence, colour, quantity, and the presence or absence of blood, pus, mucous or worms. Among the younger children the motions are examined daily. All have been found quite normal. An occasional unhealthy stool among the younger ones has yielded quickly to a dose of castor oil, with a few doses of rhubarb and

soda or grey powder. Constipation has been corrected by cascara, belladonna and nux vomica. It is interesting to note that constipation leads to a tired feeling, lack of go, and loss of flesh, all symptoms of early phthisis.

The fact that they all have creosote with cod liver oil may account for the universally healthy state of the bowels, for the latter taken over long periods, especially in hot weather leads to diarrhoea and unhealthy stools.

The faeces have never been tested for the Tubercle Bacillus. Intestinal stasis with regard to Tuberculosis is referred to later on.

The Urine. Baccilluria has been known to be a cause of irregular temperature for some time. In these cases, however, the temperature does not as a rule, have a regular, consistent swing. There are instead, definite attacks of fever, with rigors preceding, the temperature rising to 104 or more with a marked swing, and then a gradual fall to normal. An interval without any temperature follows and then another sudden attack of pyrexia with alarming symptoms.

In 12 cases I examined the urine - catheter specimens - for B.Coli. The urine, as a rule, clear, in some cases contained pale urates, or phosphates, was acid, and there was no turbidity. It was centrifuged and from the sediment films made and stained with Gram. The B.Coli was in no instance seen. No indican was obtained at any examination. All urines are tested frequently for albumen, sugar and pus. There has been an occasional transient trace of albumen after a Tuberculin injection, but that was at

the beginning of the treatment, and has not occurred for some months. It is of no importance.

Phosphaturia is said by Sir R. Douglas Powell[13] to be an early indication of phthisis, and we have certainly noticed their presence in many of the specimens of urine from early cases of the disease.

Acute Rheumatism without physical signs, and chronic septicaemia without ascertainable cause we need not consider very much. No case had valvular heart disease, rheumatism of joints, chorea, or shewed the presence of nodules; while chronic septicaemia is not likely to have a long history of pyrexia without some indication as to the focus of infection.

We now come to the question of the enlargement of bronchial glands.

Symptoms. A peculiar clanging cough, paroxysmal in type, due to pressure on the bronchus or interference with the recurrent laryngeal nerve.

Inspiratory stridor due to pressure on the trachea.

Neither of these symptoms appeared among the cases we have selected, though we have seen them elsewhere very definitely, especially in younger children.

The Physical Signs of Enlarged Mediastinal Glands are quoted thus by Still, and are seven in number.[6]

1. Impairment of note in the 1st or 2nd intercostal spaces - right side - close to sternum.
2. Enlargement of veins - usually 2nd space - from the coracoid process to the inner end of the space.

Only of value when on one side.

3. Bruit at the inner end of clavicle with the head extended and the child either sitting or standing whereby the left innominate vein is compressed (Eustace Smith)

4. Marked deficiency of air entry into some part of lung - may be a whole lobe - due to compression of bronchus.

5. Dulness about the root, in the interscapular region.

6. Increased resistance on pressure over the manubrium - due to loss of normal resilience..

7. Enlarged glands felt behind the manubrium (v.rare)

On examining 50 cases with a view to finding these signs:

Sign 1. Two were positive, 48 negative. Of these two, in one the sign was quite definite, in the other slight. No dulness over the sternum was detected.

Sign 2. Five were positive. All of them shewed the veins on the right side. Enlargement of veins all over the front of thorax is comparatively common, and occurred in several cases.

Sign 3. Nineteen were positive. This sign is somewhat difficult to obtain, for unless the patient holds the breath completely, very often a hum can be heard, which appears to be venous, but which, when breathing has ceased entirely, cannot be heard. Complete cessation of breath sounds is difficult often to obtain in children. Furthermore, this sign is said to occur in other conditions than that of enlarged bronchial glands, and its value to be more from the negative rather than the positive aspect. I examined 96 patients, in whom there was no reason to suspect

enlarged bronchial glands, and of these 74 gave a negative result, 22 a positive. Of these 22, 18 gave a positive Von Pirquet's cutaneous reaction, and 4 negative. Its value, therefore is rather indefinite, at any rate in young children.

Sign 4. In 3 cases, the air entry into one whole lung, or whole lobe was very much diminished or in parts completely absent. In 2 patients, it was one whole lobe, in 1 patient the whole lung. The percussion note in these cases is only comparatively dull, the breath sounds very far away and the vocal resonance and fremitus impaired. This condition is not stable, and comes and goes. In one of my cases, a child aged 2½, the physical signs would alternate between moist sounds all over the lung, so as to hide the breath sounds, and complete loss of air entry, when the lung was almost silent; the percussion note being comparatively dull, the vocal resonance dimished and the vocal fremitus also lessened.

Sign 5. In two cases I found dulness in the inter-scapular region, with fine crepitations, but a condition of enlarged glands was not verified on screening with the X rays.

Sign 6. I have very little experience of this sign.

Sign 7. No case shewed this rare condition.

X ray examination.

The use of the X rays in the diagnosis of Pulmonary Tuberculosis has come to the front of late years, and is a most useful adjunct. The fluorescent screen in examining the chest is better than the photographic plate. A paper by Dr. Jordan of Guy's

Hospital, on 'Peribronchial Phthisis'[8] was the first communication in England of importance on this subject. In this paper he laid down the dictum that Pulmonary tuberculosis began at the root of the lung, spreading from enlarged bronchial glands, and giving rise to a tubercular broncho-pneumonia. Dr. Jordan states that "the great majority of a series of "healthy" lungs obtained by him from the post mortem room, and examined by X rays showed calcareous deposits in the lymphatic glands, either at the roots or along the course of some of the larger air tubes; that the Tubercular process generally spreads in the form of broncho-pneumonic extension in the peribronchial pulmonary tissues which in favorable cases may cicatrise into fibrous bands, appearing as dark radiatory shadows on X ray examination of the affected lungs.

In examining the chest one must remember:

1. That on inspiration the lung lights up, and becomes less opaque. This fact is of great value, for if, on making the patient take a long breath, one apex lights up, while the other remains opaque as before, it is positive evidence that there is something abnormal in the darker one, either consolidation or lack of elasticity, etc. which may be confirmed by the physical signs.

2. That the movement of the diaphragm is restricted on the side affected with Tuberculosis. This restriction of movement occurs before any definite shadow is seen at the apex or at other portion of the lung tissue proper. This may be due to glandular or pulmonary tuberculosis restricting the air entry.

3. A vertically placed, tube like heart, is supposed to be characteristic of tuberculous soil and to be one of the Dystrophies that predispose to Tuberculosis.
4. It is often essential to screen the chest in the lateral oblique position, as enlarged glands can sometimes be seen by so doing, which are hidden when viewed from the front.

The appearance of other areas of mottling or of shadows in the lung tissue proper are very suggestive and confirmatory of shadows seen in the region of the roots of the lungs. In those cases where the tuberculous condition is quiescent and has gone on to the formation of calcareous deposits in the roots, the shadows cast on the screen are definite and easy of interpretation.

In these early cases of pulmonary tuberculosis, such a calcareous condition of the root is never seen, the disease is still active, as shewn by the presence of an irregular temperature, cough, wasting, etc. and has not reached the quiescent stage with its consequent fibrotic and calcareous changes. It is well to screen these children several times, and to make comparison with the chests of healthy lunged patients, before arriving at any diagnosis. The diagnosis of cavity formation, collapse, consolidation, displacement of the heart and so on is comparatively easy, though in early cases only the questions of consolidation, loss of elasticity, restricted movement of the diaphragm, or shadows of enlarged bronchial glands around the roots of the lung need be considered. In rapid cases cavity formation is seen. This latter picture on the screen is most striking, the cavity itself being a patch of

lighter colour, with a darkened band all around representing the fibrous walls. The X rays in these cases are very accurate. In cases of loss of elasticity or of consolidation of the apex, the changes from the normal are a matter of degree. The area affected is more opaque than its fellow of the opposite side, and on deep inspiration does not assume the accustomed lighter shade. Areas of shadowing, grape like masses in appearance, can in some cases be seen scattered over the lungs denoting consolidation, or possibly collapse.

The X rays then, shew well marked lesions very clearly. We know that the existence of a few crepitations may be diagnostic of great and extensive damage to the tissues further in the pulmonary substance: and that until the external or surface portions of the lungs are involved, these few crepitations may be the only indications of this widespread disease. It is in these cases that we have found the X rays so useful.

In many cases too, we have found restricted movement of the diaphragm on the side affected by diseases. In other cases where there are at present no definite signs of disease in the lungs, we have seen restricted diaphragmatic movement also. It is too early to say whether tuberculosis of the lung will develop on that side or not.

Where the signs of pulmonary disease are indefinite or wanting, but continued pyrexia is present, the X rays do not give us very much help. The detection of enlarged bronchial glands is very difficult and somewhat unsatisfactory and we have found it comparatively rare to find any really distinct shadow cast round

the roots of the lungs, unless the signs of hilus tuberculosis are well marked.

One notices on screening normal children that the picture presented is far more clearly cut and stands out more distinctly in outline than in those subjects suspected of enlarged bronchial glands, who give no definite shadows. This indistinct haziness, not an actual shadow - this lack of a clear cut picture, seems to be of some diagnostic importance.

The vertical position of a tube like heart has not been seen in any case screened so far, and apparently must be a rare condition, at any rate in children.

It is well to remember that after meals, if there be any flatulence the stomach is dilated, and the left half of the diaphragm moves less than normally.

Each of our 50 cases was examined several times. In over 20 the examination was negative, except that this haziness of outline was frequently observed. Among the rest in about a dozen fairly definite shadows round the roots of the lungs were observed which were ascribed to the presence of enlarged bronchial glands. In the rest the signs were of consolidation and loss of elasticity, and in two cases there was cavity formation. In about half, restriction of the movement of the diaphragm was well marked.

Von Pirquet's Cutaneous Reaction.

It is convenient to make mention here of this cutaneous reaction to Tuberculin, and discuss its value in brief. There have been several methods devised for the detection of Tuberculosis in the living subject. Among these are:

1. The Wolff Eisner or Calmette's Conjunctival Reaction.
2. Von Pirquet's Cutaneous Reaction.
3. Moro's Percutaneous Reaction by Inunction.
4. Subcutaneous Injection of Tuberculin.

Calmette's Reaction unfortunately - for it is accurate - is attended with some danger, for unless local tubercular disease in the eye and in the structures around be rigidly excluded, serious results may follow. The subcutaneous injection of large doses of old Tuberculin is by many considered a dangerous proceeding. This danger is strenuously denied by Camac Wilkinson[14], who maintains that in his experience no evil has resulted from this method of diagnosis. However, there seems to be a real danger, in some cases at any rate, of lighting up areas of disease once active, but now quiescent, and for this reason it is doubtful if it is justifiable to use it promiscuously or in the routine way in which Von Pirquet's method is employed. Moro's Ointment is not very generally used, and it is usually regarded as not so reliable as other means.

There are several advantages to be claimed therefore for the Von Pirquet method. It is harmless, it is simple, it is universally adaptable, it is reliable within limits. It labours under the disadvantage shared by all other methods, in that as soon as the body tissues have been exposed to and reacted to tubercular toxins, the reaction is from henceforth positive. Therefore, patients with healed tuberculosis will give a positive reaction. The tissues have been sensitised by the toxins of the tubercle bacillus and so they react to local contact with

Tuberculin. Thus, Tuberculin, in enormous doses can be given to a person who has never had tuberculosis, without causing any reaction or leading to any harm, but if once the tissues have been sensitised by an attack of the tubercle bacillus, be it tubercular cervical glands, or hip or spine, etc. tuberculin even in small doses leads to reactions, and in large doses to serious results and even death.

The Reaction is therefore of more value in diagnosis when negative, as an indication of freedom from Tuberculosis. Under the age of two in particular, when Tuberculosis is rare, the test has an added weight, and decreases in value as the age of the patient increases.

In acute generalised tuberculosis, in acute miliay pulmonary tuberculosis, in advanced phthisis, in Tubercular meningitis, the reaction is negative. In chorea it is often positive. In lupus the reactions are violent and usually the most marked of all.

The technique is simple. The skin of the patient's forearm is rubbed with ether, and he is then vaccinated with old Tuberculin, Human and Bovine, and a control is added of sterile water. An ordinary darning needle, boiled, or flamed with a spirit lamp does very well. It is not necessary to draw blood, but only to abrade the epidermis. The reaction is shewn by a red, raised papule, with erythema around and sometimes a ring of vesicles. This may occur 12 hours after or 24 - 48 hours, and sometimes only appears after the 3rd or even 4th day. Originally any reaction after 48 hours was not considered positive.

All our cases gave a positive reaction, usual-

ly after 20 - 24 hours. Where Bovine Tuberculin was used as well the reaction was positive to both. Recently Von Pirquet has devised a modification of his original method, known as the Von Pirquet Quantitative Cutaneous Tuberculin Test, or more shortly Quanti-Pirquet (Q.P) In this method he utilises the fact that the grade of the reaction of the body to Tuberculin - its tuberculin sensitiveness - is in general an index of the amount, i.e. the activity - of the disease present. By a scarifier four circular holes are drilled in the skin about an inch apart, a drop of tuberculin is now applied to each scarification in turn, the weakest dilution distally. The diameter of the resulting papules is measured in millimetres after 24 and 48 hours - only the papule is measured, not the surrounding zone of hyperaemia. The tuberculin of course is in four different strengths, usually 64%, 16%, 4% and 1%. The measurements are charted after 24 and 48 hours, the mean taken, and deductions drawn from the result, the greater the measurement with a weak dilution the more active the disease. This method is somewhat in its infancy, and it is too early to form an opinion of its value.

With regard to the old method, if we remember the conditions in which the reaction is negative, although disease is present, and if we use it appreciating its negative value, we shall find a large sphere of usefulness for it.

The Pulse.

According to Hutchison the pulse rates are:[15]

Ages 5 - 9, 95 - 100
9 -10, 90.
16 -17, 80.

That is between 10 and 16 years of age, the pulse rate lies between 90 and 80. We know that phthisical adults shew a definitely increased pulse rate, that this occurs in the early stages and is therefore of diagnostic importance. In children, the rate is increased also. We found with a twice daily record of pulse in phthisical children from 12 - 15 that in the great majority the rate lay between 96 and 110, and of these children none were cases with high swinging temperatures at the time. If the temperature chart shews variations from normal or below, up to 101° or 102°, the pulse rate rises accordingly, but in the usual early case with little or no temperature or with an occasional rise, or with characteristic swing from 97 - 99 at night, the pulse rate is also distinctly quickened. The pulse rate must be taken over a considerable period and compared constantly with the temperature, as any marked rise of the latter will of course be accompanied by an increase in rate of the former. The pulse frequency like the temperature, in children, varies very much and responds to slight stimuli, it is therefore not of such high diagnostic value as in the adult.

The Blood Pressure.

Fever, acute or continued, reduces blood pressure. In phthisis therefore we should expect a reduction, and such is the case.

As judged by the standard of Cook and Briggs, the blood pressure in children up to 2 years is 75 - 90 mms of Hg, of older children 90 - 110 mms, of young adults 130 mms.

Professor Sahli quotes the following

figures[16]:

Age	12	Blood pressure	112	mms -	systolic
"	10	"	110	"	"
"	4	"	110	"	"

Potain estimated the figures at different ages as follows[17]:

Age 6 - 10	89	systolic
10 - 15	135	"
15 - 20	150	"
The adult about	170	"

With his more complete apparatus Erlanger reports that the average pressure in the brachial artery is 110 mms (systolic), when the psychical factor is excluded. Von Recklinghausen gave the figure for adults also as 116 mms of Hg.[19]

There is, therefore, a fair margin of difference in these figures. The personal equation in the use of an instrument such as the Riva Rocci Sphygmomanometer is considerable and the psychical element to which Erlanger drew attention is also a disturbing factor. A truer reading of the pressure is to be obtained after it has been taken once or twice, and has thus allowed the strangeness of the proceeding to wear off, especially is this so in children. We have used French's modification of the Riva Rocci instrument, and have taken the systolic pressure only, by obliterating the pulse and taking the reading at the moment of its return. We find that the better developed a child is the higher is the reading of the instrument, unless the temperature has

a pronounced swing. In other words the child who reacts but little to the Tubercle toxins has a higher blood pressure than the one who is greatly affected. We find too that the phenomenon known as Tache Cerebrale - that is if the finger nail be drawn across the skin of any region a red line comes out quickly - can be obtained with much ease in these phthisical patients, shewing a vasomoton insufficiency. However in Tubercular Meningitis and Typhoid Fever, conditions in which this sign as a rule well marked, Osler says it is[18] of no diagnostic importance. We do not quote it, therefore, as of any value in diagnosis, but rather as shewing additional evidence of the low pressure of the blood due to lack of or diminution in the vasomotor control as a result of the action of the Tubercular toxins.

We have seen that these early cases of pulmonary tuberculosis are anaemic, and that poor chest expansion is a dominating feature of their condition. It is interesting to find that the Blood pressure is lowered in chlorotic anaemia, and that it is raised in the act of inspiration: good expansion of the lungs therefore, raises the pressure.

It is not common to find true chlorosis, however, in these patients. Lloyd Jones says 'Examination of the blood generally shews no definite change or at the most there is slight secondary anaemia, still more rarely true chlorosis'.[4]

In active pyrexial disease leucocytosis is present, which often does not correspond to the degree of fever but to the amount of disease in the lungs. We have already noted the blueness and coldness of

extremities commonly found in these children due to the deficient and feeble circulation.

Figures. The age is given, and the blood pressure - systolic - calculated in millimetres of mercury. The blood pressure figures are the mean of three readings taken on three different occasions. It will be seen that on the whole the figures are lower than those obtained in the normal child at corresponding ages.

Age	Blood Pressure - systolic. (the mean of 3 readings - taken on 3 separate occasions)
5	80
7	96
7	92
8	105
9	118
9	104
10	98
10	96
11	114
11	85
12	108
12	118
12	106
12	105
12	96
12	95
13	108
13	88
13	96
13	109
14	95
14	102
14	105
15	105
15	104

Early Physical signs of Pumonary Tuberculosis.

It is difficult in the extreme to say that the presence of any one sign or group of signs, constitutes a diagnosis of phthisis. In the more advanced stages of the disease these are as a rule definite and easy of detection, and so the same difficulty does not arise. But this is a by no means constant condition, for even in advanced cases the signs are sometimes curiously wanting, where on post mortem examination extensive disease of the lungs is found. In cases of very acute phthisis, the toxins destroy the patient so rapidly that there is no time for definite signs in the lungs to develope. In other cases, yet again, the disease begins so deeply in the lung substance that it is a very long time before it arrives at the surface and so betrays its presence. Dr.Gee's aphorism that "therapeutics must begin before physical signs are developed, and if you wait for physical signs you wait too long" is only too true.

In 1880 Sir James Kingston Fowler stated that a spot situated from one to one and a half inches below the actual apex of the lung was the seat of disease in the large majority of cases, and that the disease spread firstly backwards and downwards along the anterior portion of the lobe. The apex of the lower lobe became affected at a comparatively early period, the disease tending to spread downwards along the posterior surface of the lung and laterally along the line of the interlobar septum. The apex of

the upper lobe of the opposite lung soon followed the implication of the lower lobe apex. Fowler attributed the frequency of apical disease to the lesser functional activity of the lung, which is imperfectly expanded in ordinary quiet breathing, such impairment of function being associated with lessened resisting powers. There is, in fact, a certain degree of local anaemia, or isehaemia. The result is, we have the very conditions most favorable to the deposit and development of the Tubercle bacillus, such deposit not being surrounded by well filled capillaries, which are uniformly present in the actively functioning areas of the lungs at the bases and elsewhere. There is, in short, 'defective lymph lavage'. Dr Jordan states that in 40% of cases of phthisis 'the disease commences as a definite peribronchial infiltration and spreads from the hilus in all directions, but most rapidly along the ascending and descending branches of the main bronchus, and that sooner or later the disease reaches the apex by way of the ascending bronchiole'[8].

We have considered elsewhere various views on the source and channels of spread of infection of the Tubercle bacillus, whether by inhalation or by ingestion; how in the opinion of some the abdominal glands are primarily infected, principally by ingestion of milk from Tuberculous cows, or in other ways, with subsequent spread to the lungs by the blood stream; how others consider the infection is by direct inhalation into the alveoli, thence the bacillus is taken to the tracheo-bronchial glands by lymphatics,

and by the blood stream to the lungs, or alternatively the cervical glands are infected through the tonsils, the supraclavicular glands next, then the bronchial again and finally the lungs.

We must not forget that Pulmonary Phthisis occurs in the lower lobes of the lungs in children, whereas in adults basal phthisis is a rare occurrence.[21] I have one marked case with basal signs at present. According to Clifford Allbutt's System of Medicine. The Early Signs of Phthisis are:

1. Slight diminution of Respiratory movement - often absent.
2. Cog wheel breathing - found in other conditions.
3. Harshness of breath sounds - expiration equals inspiration in intensity - due to want of elasticity in the lung substance.
4. Expiration higher pitched and prolonged - an early symptom of consolidation.
5. Vocal Resonance and Vocal Fremitus slightly increased.

Dr. Latham writes "The Physical Sign to which I attach the most importance, is the persistent presence of crepitations at the apices of the upper lobes. It is of the utmost importance that the posterior surface of the apices should be examined as thoroughly as the anterior, for it is the former position that the earliest physical signs are usually to be detected. We should always make the patient cough, and then examine, to see if this act brings out a shower of crepitations. These - known as post-tussic crepitations - are the most valuable sign we possess

of the presence of an early Tubercular focus, but they tell us nothing by themselves of its activity or latency."

Later on in the disease, diminished movement, flattening of the chest, particularly supra and subclavicularly, dulness, tubular or amphoric breathing, vocal resonance and fremitus markedly increased etc. make the diagnosis practically certain, although it cannot be said to be complete unless Tubercle Bacilli have been found in the sputum. This is the one absolute proof of the disease which there is no gainsaying, and no pains therefore should be spared to demonstrate the presence or absence of the bacillus.

We have come to look upon crepitations as the most reliable early sign in pulmonary tuberculosis, the more so because the X rays have frequently clearly demonstrated disease in the lungs when crepitations were the most notable sign obtained on clinical examination. We have found also a great lack of chest expansion in these early phthisical patients.

The most frequent site of disease has been the apices of the lungs, the upper as a rule, but the lower ones not uncommonly, the right and left sides have been fairly equally affected. In about half the 50 cases the only signs and symptoms present have pointed to an affection of the bronchial glands, only occasionally demonstrated by the X rays. At present we have one case of basal phthisis only, but it is by no means uncommon.

The signs of enlarged bronchial glands have been described elsewhere.

To diagnose these early cases of Pulmonary Tuberculosis, we inquire about the family history, the surroundings and circumstances, and occurrence of frequent colds, attacks of fever, or bronchitis, possibly measles and whooping cough. We are told the child lacks go, lolls about, is easily tired, is getting thinner, may be breathless, coughs or sweats at night. We find a child, usually over 5 years of age, with, maybe the Tubercular diathesis, long lashes, fair complexion, etc. with the flat or alar type of chest, cold extremities, anaemic looking, expansion of chest very poor and general nutrition bad. After further observation we discover a swinging temperature - possibly it may be normal - perhaps one with the characteristic swing 97^{o} - 99^{o} at night. We look for enlarged glands and scars, nodules, etc. inspect the mouth for oral sepsis, the throat for enlarged Tonsils and Adenoids, examine the urine and the motions. On measuring the chest we find it badly developed and lacking in girth. Our Von Pirquet's Reaction is positive, examination of the chest may or may not reveal definite signs of disease, if definite, they are found most often in the upper apices, maybe in the lower, and sometimes at the bases, the most dependable thing being the presence of crepitations which persist, and are either always present, or can be elicited after the patient has coughed. X rays may shew definite signs of a lesion or lesions in the lung substance, or enlargement of glands round the roots. Some of the signs of enlarged bronchial glands may be present, with paroxysmal cough or inspiratory stridor. The pulse is often quickened and the blood pressure lowered.

Treatment.

The Modern Treatment of Pulmonary Tuberculosis as carried out in a Sanatorium or in the home aims at:

1. The Increase of the patient's resistance.
2. The Regulation of Auto-inoculation.

The first is attained by attention to general hygienic and dietic principles, with due regard to the surroundings and environment, and the mode and manner of living: the second by graduated exercise and labour, inhalations, and tuberculin injections.

The treatment at Queen Mary's Hospital is conducted on the lines laid down, and will accordingly be shortly described.

The Hospital stands high, is isolated, covering 136 acres, is built on gravel soil and on the pavilion system. Off a central main street there are six side streets, three on each side. Each street contains four blocks of buildings, and each block two wards, the wards standing out at each end and separated by two dayrooms. The blocks are placed one behind the other in a line running slightly upwards, so that each one stands four square to the world. The Tuberculosis blocks are the end ones, and so the least hedged in - open-air verandahs are built all around, with shutters, and glass roofs, the former can be let down in bad weather. These verandahs face the south, and in them phthisical and tuberculosis patients are nursed, remaining out of doors day and night unless

some complication - which rarely happens - contra indicates. Hot bottles and warm but light clothing protect them from chills.

The children fit to 'get up' go to school for an hour or two, are taken for prescribed walks by an attendant, have daily drill, breathing exercises, and graduated light labour, such as rolling the grass, digging trenches, etc. The drill lasts for an hour and consists of various exercises for improving the general musculature of the body; arm, leg and body drill, after the Swedish fashion. Some have only half an hour, according to age and condition, others the full drill. All exercise is increased very gradually.

Graduated exercises are arranged as follows, the children being divided into classes.

Class A+ Treated in bed on their backs as enterics.

" A In bed, but allowed to sit up.

" B Up 2 hours a day.

" C Up ½ day with ½ hour's walk.

" D Up whole day, with 1 hour's walk.

" E Up whole day, with 1 hour's walk. ½ drill.

" F Up whole day with 1 hour's walk and full drill.

On admission each patient is kept in bed under observation for a few days. Any swing in the temperature is noted, or if normal, whether it rises on the patient's getting up. The pulse rate similarly is observed. The urine is tested, the motions in-

spected the bowels well opened. Physical signs and the condition of teeth and tonsils noted. Von Pirquet's cutaneous test is performed, the chest X rayed and blood pressure ascertained. The child is then assigned to the class considered suitable, and moved up and down as her condition improves or otherwise. Any temperature leads to rest in bed for some time, to obtain its reduction by lessening the autu-inoculation. If however, rest fails to do this, the patient is allowed to get up for a short time each day.

Diet and Drugs. The food is liberal but plain, and contains a fair proportion of proteid and fat. Fat bacon, eggs, bread and butter, plenty of milk, joints of meat, cocoa, milk pudding and jam etc are the principal items. No child is encouraged to stuff or to overeat, but to eat well and properly masticate the food. In some cases, where the general nutrition is very poor and remains so despite treatment, we have tried Plasmon, Glidine and Sanatogen. All cases receive cod liver oil in some form, either combined with extract of malt, or as an emulsion. In anaemic looking children we combine the Tincture of the Perchloride of Iron with Liquor Strych Hydrochlor and Liquor Arsenici Hydrochlor, which we believe improves the colour, encourages the appetite and leads to increase of weight. We have found this mixture of much greater value than the well known Parrishs' food but the Syrupus

Ferri Iodidi we have also found useful. We find, only too often, that those children who start to put on weight, increase steadily on almost any diet, while others remain unaffected, their weight stationary, no matter what the diet, and what drugs be prescribed.

In cases with much cough and profuse expectoration we have added creosote to the cod liver oil emulsion. The really beneficial results of this drug, according to Dr. Pritchard,[1] do not become apparent until very large doses have been given. We usually begin with two minim doses three times a day, and week by week, or at greater intervals increase the dose by two minims until the patient is getting 16 - 20 drops. It is most useful in advanced cases with cough and much expectoration. Although the digestion is said to be affected by such large doses we have not had any complaint as yet.

The invariable healthiness of the motions may be due to the exhibition of creosote.

Mr. Arbuthnot Lane has recently drawn attention to the association between intestinal stasis and the tuberculous process in children. "Intestinal stasis, or any condition that tends to intestinal toxaemia, is quite one of the most unfavourable elements in tuberculous disease" says Dr. Pritchard.[1] Attention to the bowel therefore should be one of our first considerations in treatment. He recommends the following prescription, which we have used for a long time.

Liq. Pancreatis	x m
Calcii Hypophosphitis	gr 1
Emulsions Petrolei	ad. 3 1 3 11

after each meal.

Petroleum, as an emulsion, promotes the healthy function of the bowel, liquor pancreatis is a digestive and calcium hypophosphite as a tonic.

We have seen that creosote with cod liver oil emulsion checks cough and expectoration but cod liver oil alone effects this, and seems to be more efficacious in the pure state than when combined with malt extract. Cough that persists we treat with a linctus containing opium, but with some reserve. It is mainly given at night time. For cough with profuse expectoration we make use of Burney Yeo Inhalers. These masks are placed over the mouth and the sponge sprinkled with a few drops of the following inhalant.

Carbolic Acid	3 i.
Creosoti	3 11
Tr. Iodi	3 11
Sp.Etheris	3 11
Sp. Chloroformi	3 i

They begin by wearing the inhaler for 4 hours every day, and this is gradually increased so that they eventually wear it the whole day, and only take it off to sleep and have their meals. The inhalant is sprinkled on every two hours or so. There is practically no difficulty in getting even small children to wear them, but occasionally a sore spot occurs from chafing, unless good watch be kept. These inhalations stop the cough, dry up the secretion, and sweeten the

breath, and the general health improves in consequence. The checking of cough is important owing both to the wearing out of the patient's strength by the continual strain involved, and by the temperature produced by auto-inoculation.

The above inhalant mixture in two cases caused carboluria. The carbolic acid is now omitted from it and the Oleum Pini substituted.

Temperature.

The presence of a temperature swinging above normal proves that auto-inoculation is going on, and that the patient's body is reacting to the influence of certain toxins in the blood. It is essential that this auto-inoculation should be reduced to a minimum. Now such conditions as cough, restlessness at night, mental anxiety and fatigue lead to auto-inoculation, and must be relieved wherever possible, but the principal cause is bodily movement, exercise and labour, muscular exertion in fact, and this can be restricted as required or reduced to a minimum by confining the patient to bed, and in the most extreme cases by keeping them on their backs and treating them as enterics.

Twelve cases were taken, the signs in whose lungs were indefinite, but whose temperatures were irregular. They were placed in Class A 1 and treated as enterics. Being mainly young children they were lightly strapped over the chest to prevent them sitting up. However they soon understood that they must keep lying flat, and aided much by their obedience. They were fed lying

down, used the bed pan lying down, and in all respects were treated as typhoids. For six weeks they remained in this state, and in not a single instance was the temperature reduced in the least. Six of the twelve had injections of Tuberculin during this treatment, and six remained without it. At the end of this time they were in a very weak state, and could not even sit up so that it was deemed inadvisable to keep them longer on their backs. Accordingly they were allowed to sit up in bed, and gradually advanced to more energetic classes, strangely enough in a few instances the temperature was lowered on their getting up.

Complete rest has not much effect on these temperatures typical of enlarged bronchial glands, i.e. 97° with a nightly rise to 99° or 100° but in cases of high temperature with definite signs the swing is greatly reduced, or brought down to normal, and the general condition improved.

The exercises, walks and drill assigned to each child are regulated by the temperature. Any rise is regarded as a sign to reduce the amount of physical exertion and the patient is placed in a lower class. Conversely, absence of temperature is the signal to promote the child to a class with more exercise.

It is practically never necessary in the early stages to reduce temperature by antipyretic drugs. Night sweats, too, are rare, and never very severe, they are best treated by Niemeyer's pill.

Tuberculin Treatment.

The Treatment of Tuberculosis by Tuberculin is still more or less in its infancy. Nevertheless considerable differences of opinion exist with regard to dosage the kind of Tuberculin, and the method of administration to adopt. One school regards, the other disregards, reactions. One aims at a maximum standard dose, the other at a maximum optimum dose for each patient. Some use Bovine, others Human tuberculin. Some employ Human for Pulmonary, and Bovine for abdominal Tuberculosis and glands in the neck, while others again prefer Bovine for Pulmonary and human for abdominal disease - the crossed method. Some put their faith in the Old Tuberculin, others in the New, or Bacillary Emulsion, while others start with Bovine or New, go on to the Bacillary Emulsion and end with Old. Though there are, then, many ways of using the different kinds of Tuberculin, the results claimed are much the same whatever plan of campaign be adopted.

The effect of an injection of Tuberculin, if given to a susceptible subject in large enough quantity, is to produce a reaction. A reaction may consist of three phases :

1. A General Reaction, with rise of temperature, a feeling of malaise, headache, vomiting, abdominal pain, or pain in the side.
2. A Focal Reaction, i.e. at the seat of the lesion. If in a case of lupus there is swelling and redness of the part, if in a joint, the parts around are swollen

and inflamed and the discharge increases for a time, if in a lung, the focal reaction may be indicated by greater cough, and more expectoration, with the appearance of rales or crepitations not present before, or an increase in signs already existent.

3. A Local Reaction at the seat of injection, shewn by swelling, heat, redness and tenderness of the arm, and in some cases pain in the axillary glands. There may be inflammation along the needle track.

Whatever the reaction, in a few days, usually in 12 hours, the evil symptons have subsided, a feeling of well being replaces the malaise, the appetite and general health improve, the parts settle down again having been flushed with blood and the local reaction disappears.

The primary object of Tuberculin is to create a hyperaemia of the parts. Tubercular lesions are typically avascular, blood vessels are debarred by the tendency which always exists to the formation of fibrous tissue. Tuberculin appears to be the only means in our power to create a hyperaemia, and so a flushing out with blood - a lymph lavage - of tuberculous masses situated in the lungs, mediastinum and abdomen, as well as of old lesions of joints, and cervical glands.

Secondly by Tuberculin we hope to increase the patient's powers of resistance, to increase the amount of antibody or lysins in the blood and so afford him a protection against further infection. Unfortunately these antibodies or lysins in destroying the tubercle

bacilli set free harmful toxins into the blood stream, and in the case of a tuberculin injection or auto-inoculation give rise to the well known tubercular reaction. In auto-inoculation the patient by exertion, muscular or even mental, as by exercise or coughing etc. infects himself. Either by creating a hyperaemia from his exertions, or by direct breathing down of a tuberculous focus bacilli are poured into the blood, and being destroyed by the resulting antibodies or lysins, toxins are liberated. By graduated injections of tuberculin we aim at raising the patient's powers of resistance to a pitch at which he can deal successfully with the toxins of his own making, and secondly by bringing increased blood to the parts hastening their recovery.

To obtain a reaction the patient must be sensitive to Tuberculin, and this sensitiveness can only be acquired as the result of infection with the tubercle bacillus. Large doses of Tuberculin can be injected into a non tuberculous person with no ill effect, but let that person have had at any time an infection with the bacillus and sensitiveness is established and a reaction inevitably follows. Wolff Eisner[2] explains this phenomenon of sensitiveness as we have already seen by supposing the blood of a tubercular subject to possess a substance capable of breaking down the tuberculin molecule with the formation of toxic products. This substance or antibody he calls tuberculo-lysin. The non tubercular do not possess this lysin and therefore

the injected tuberculin is not broken down, hence there is no reaction.

Tuberculin to be successful as a therapeutic agent, must attain certain ends, and to push it in the face of certain danger signals is as dangerous as to continue the use of digitalis with decrease of urine, a quickened pulse and digestive disturbance. What, then, are the indications that Tuberculin is effecting its purpose, and doing good?

1. Increase in well being. This has been alluded to before. After a reaction a period sets in when the immunising response has been increased, and is marked by a feeling of greater comfort and well being, of more energy and improved health.

2. Increase in weight - eventually - and this must be added, for it is usual to find that with the administration of the drug - regarding reactions - there is a slight loss of weight at first, and then after a week or two of slight decrease, or of stationary weight, the patient begins to put on flesh and the weight line rises. In some cases the weight line remains stationary or only very slightly rises during the whole time that Tuberculin is given, and on stopping the course, the line rises very quickly and weight is put on rapidly. This interval of slight loss or stationary weight is important. In an article in the British Medical Journal of Feb. 1, 1913[23] there is an account of a comparison made at the Brompton Chest Hospital, between 10 patients treated with Tuberculin and 10 untreated. At the end of three months it

was shewn that those who received no Tuberculin had put on weight more rapidly than those who had had injections and in other respects, that is in their ability to walk certain distances and perform certain work, and in the progress of the physical signs in the lungs, those who had had no injections had shewn the greater improvement. The conclusions drawn as to the value of Tuberculin do not seem to us quite logical, believing as we do, that the weight increases - at any rate in some subjects - more after the course of Tuberculin has been discontinued for a time. A peculiarly 'sensitive' patient will often have big reactions, no matter how careful the dosage, and until the required amount of tolerance has been gained, must lose weight at first. There are cases, of course, where the patient starts to put on weight from the beginning and the line rises steadily week by week, but we repeat that the majority of cases treated in this Hospital, shew a slight loss of weight when Tuberculin is begun, or a stationary condition, followed eventually by a rising weight line, which rises still more rapidly - for a time - when the course of injections have been discontinued. This rapid rise ceases after a time, and sometimes is an indication to restart the injections. If Tuberculin be given without regard to reactions, this further retards the patient during the time of the injections, owing to the frequent attacks of malaise, high temperature, restricted diet and confinement to bed.

Any continued marked fall in weight must, in most cases, be taken as an indication that Tuberculin is doing harm and ought therefore to be stopped. In some cases, however, even under these circumstances, one may be justified in going on if the patient is strong and can - so to speak - afford to lose some weight, and if other aspects of the case are improving, such as the physical signs diminishing, the temperature lower, cough and expectoration less and general improvement in well being.

3. Diminution in the character and extent of the physical signs in the lungs, with consequent lessening of cough and reduction in the amount or entire disappearance of the sputum, or of the numbers of Tubercle bacilli in it. A focal reaction will sometimes lead to an extension of the area of the physical signs, or to an increase in the intensity of the existing signs in an affected part of the lung. This results in an increase in the cough and expectoration and occasionally to an haemoptysis. These changes in the patient's condition, are, as a rule, transient, and indicate that the dose has been too large. Any continued increase in the intensity, or the area of the physical signs, in the cough, and expectoration or in the numbers of Tubercle bacillus in the latter, must be followed by witholding the injections for some time at any rate, if not altogether.

4. Reduction of Temperature. In the febrile, sometimes Tuberculin reduces the temperature in a remarkable

manner. I append a chart shewing this point. In this case the Tuberculin, in small doses, very carefully and slowly increased in dose, brought down the temperature from 102 - 103, to normal, and kept it at this level. This chart shews too the loss of weight on first giving the drug, and the steady rise afterwards.

However varied the opinions with regard to the administration of Tuberculin may be, everyone is unanimous on the point that to give to afebrile cases is a comparatively simple proceeding, while the real difficulty lies in giving it to those who are running a temperature. With the afebrile cases a reaction can be seen by a glance at the chart. The normal line is overshot at some point, by a rise of one or two degrees, followed by a fall to normal, and the temperature remains level for an indefinite period, until a small overdose causes it to rise again. But in the febrile cases, with a temperature down in the morning and up in the evening, or one which is up and never reaches normal, the difficulties are greatly increased, and much judgement is required. Certainly in these cases it is never safe to disregard reactions, for very serious consequences may result. The existence of a swinging temperature is proof that the system is absorbing a quantity of toxins day by day - the higher the temperature, the greater the proof - and to add to the already over-loaded system large doses of Tuberculin is only to court disaster whereas by small doses, judiciously given with regard to general focal and local reactions, a steady immunising

response is obtained, and the tissues aided gradually to overcome the auto-inoculation produced by their own toxins.

The most commonly used Tuberculins are the Bovine Old Tuberculin (P.T) The Old (T) The Bovine New (P.T.R) The New (T.R) and the Bacillary Emulsion (B.E) There are, of course, a great many others.

The New Tuberculin is made by grinding dried cultures of virulent bacilli, till no intact organisms remain. It is treated with distilled water and centrifuged, when a white deposit sinks to the bottom. This deposit is further ground up, and glycerine is added up to a strength of 20%. This fluid contains the insoluble parts of the bacillary bodies, and being insoluble is much less quickly absorbed, and so is safer to use than the Old Tuberculin, which contains the toxins, or exogenous products. The New Tuberculin, containing the bacilli themselves is known as Endogenous.

The Old Tuberculin is made by taking a pure culture of human tubercle bacilli on nutrient broth containing 5% of glycerine, sterilising by steam for half an hour, evaporating to one tenth of its volume, and then filtering. Finally .5% of carbolic acid is added, the preparation stands for some weeks and is then refiltered. It is, therefore, exogenous, since it contains the toxins, and not the bacilli themselves.

The Bacillary Emulsion is made by suspending $\frac{1}{2}$ a gramme of finely powdered Tubercle bacilli in 100 c.cs.

of a mixture of equal parts of glycerine and water. By prolonged shaking a fine emulsion is produced. The Bacillary Emulsion is therefore but T.R or the New Tuberculin with the supernatant fluid containing the more soluble products of the Tubercle bacillus unremoved. It is therefore both endogenous and exogenous.

The Tuberculins used in the treatment of Pulmonary Tuberculosis in this hospital have been the New Tuberlulin T.R and the Bacillary Emulsion B.E. At first T.R. only was used, but as the doses rose, owing to the cost, it was thought expedient to try B.E. We may begin therefore, with T.R. and then go on to B.E. or use B.E. from the beginning.

The dilutions are made according to the following scheme:

1 unit = .00001 c.c. of original Tuberculin T.R.

$$= \frac{1}{10{,}000\text{th}} \text{ m.gr. of T.R.}$$

$$= \frac{1}{20{,}000\text{th}} \text{ m.gr. of B.E.}$$

i.e. 100,000 units = 1 c.c. of Original T.R.
= 10 m.gr of T.R.
= 5 m.gr. of B.E.

There are seven dilutions :

1 c.c. of dilution A contains .00001 c.c. of original T.R.
Dilution B is 5 times as strong as A.
" C " 4 " " " " B
" D " 5 " " " " C
" E " 5 " " " " D
" F " 4 " " " " E
" G " 5 " " " " F

There is a scale of dosage in units, shewing the

percentage increase of each dose, and this scale is arranged so that no dose is more than .33% increase on the one preceding. The scale ascends by units from 1 to 100.000, and opposite the units are indicated the dilution of the Tuberculin to be used, and the number of the divisions of the syringe. Thus to give 2 units one takes 4 divisions of the A dilution, to give 10 units 4 divisions of the B dilution (because B is 5 times as strong as A), to give 40, 4 divisions of C, (because C dilution is 4 times as strong as B) and so on.

We used Eyre's all glass syringe, with a platino-iridium needle. The barrel is divided into twenty divisions, each division is one twentieth of a C.C.

In giving Tuberculin to cases of Pulmonary Tuberculosis we have used the method of "rising doses at short intervals", Surgical Tuberculosis we have treated by Wright's method of small doses at long intervals. At first no regard was paid to reactions, except ones that were very severe - marked by rise of Temperature to 102 or more, headache, vomiting and pain in the side or abdomen. The injections were twice a week, and unless a severe reaction was obtained, the dose was increased each time. It must be admitted that no marked ill effects followed this method of procedure. If the reaction was severe the injections were omitted until the condition had settled down to

normal more or less, and a smaller dose than before was injected. In two months the dose in many cases had reached 400 units = .004 c.c. of original T.R. = .04 of a milligram of T.R.

We have abandoned this method of giving Tuberculin and now regard reactions, however slight, and try as far as possible to avoid them. In disregarding reactions the weight almost invariably falls during the exhibition of the drug, though it rises after the injections have been discontinued. By avoiding reactions the weight in the majority of cases rises slowly at the same time that the injections are being given, after the first week or so, or at any rate remains stationary; there are no set backs as the result of violent reactions, and if the patients are up, no need to be constantly referring them to bed again, until the temperature is down. Violent local reactions are also avoided, which cause a good deal of discomfort to the patients, and there is much less danger of lighting up quiescent foci in the lungs, which might lead to serious extension of the area of disease. Tuberculin in gradually increasing doses may lower the temperature, and keep it low, with large doses quickly attained the temperature invariably rises. The initial dose depends to some extent on the age of the child, its general condition and weight, but usually 2 units are given. If there be no reaction 4 units are injected, then 6, 10, 12½, 15, etc. The injections

are given twice a week. A reaction is followed either by increasing the interval before the next injection, lowering the dose, or repeating it, according to the severity. In no case is another injection given until the reaction has quite passed off and no increase of dose, till the former dose fails to produce a reaction. A local reaction, if large, should be regarded with suspicion and the dose remain the same, or be lowered: A focal reaction means leaving off the injections till the signs have reached their previous condition, and then continuing more slowly. As the doses are raised, and tolerance established, they can be increased more quickly. At the start it is better not to raise the dose by more than a half of the preceding dose, though one can usually double two, and go to four units, and often four to eight, but rarely eight to sixteen, but each case must be judged on its own sensitiveness to the drug. We allow our patients to be up - that is if they are up as a rule - on the day injections are given, but no drill or violent exercise is permitted. If there are any signs of malaise, they go at once to bed. We find that if there is any temperature reaction, the rise as a rule occurs at 6 a.m. the following morning, the injections being given at 10.30 a.m. the previous day, sometimes it occurs at 10 p.m. or twelve hours after. An aperient is always given the night of an injection. Many of our patients have had a swinging temperature before Tuberculin was started, and this class of patient is the most difficult

to treat. However, one is helped by the fact that Tuberculin sometimes lowers the temperature. In patients where the swing is from 97° - 99°, unless over 99, the temperature is disregarded. If the swing is to 101 - 102 or more and Tuberculin does not reduce it, then the advisability of discontinuing must be considered, paying due regard to the general condition and comfort of the patient, lessening of cough and expectoration, the physical signs and weight: A rapidly falling weight and a high temperature is an indication that Tuberculin is doing harm.

The injections are given twice a week to start with, but when the dose has reached 1,000 units, every week or ten days is often enough. There is never any need to hurry. Fairly frequent intervals of freedom from the injections should be allowed, when the weight rises quickly and all the good effects of the drug become more marked. Then after an interval they can be restarted, until the patient is having large doses, when the interval can be much increased. Probably the whole course should extend over a year.

The maximum dose to be aimed at, is the one that is the optimum for each individual patient. As long as he does well with the increase of dosage, the injections should be continued, but as soon as he begins to feel 'not so well' after the Tuberculin, or to run a temperature where previously there was none, or to lose weight though formerly he had gained, the optimum dose for that particular subject has been passed, and

accordingly the dose should be lowered. In changing from T.R. to B.E. the dose should be diminished as B.E. is the stronger, containing as it does the toxins as well as the bacilli.

To recapitulate briefly : We believe T.R. is the best preparation of Tuberculin in these cases, owing to its slowness of absorption, and its mild action, but its cost makes it prohibitive for large doses. We therefore change to B.E. lowering the dose appropriately. It is better in giving Tuberculin to pay attention to the reactions, local, focal and general, and to advance the dosage accordingly. In case of a reaction it is well to keep the patient in bed, on a light diet, clear out the bowels, and either reduce or repeat the dose, or lengthen the interval between the injections. The local reaction should be regarded and a violent one met by a reduction or repetition of dose. There should be intervals of freedom from the injections, increasing as the dosage increases. An optimum maximum dose should be the goal. The whole course should last at least a year. Rapid loss in weight, increase in the physical signs, of cough, expectoration, or continued rise of temperature, should make us consider the advisability of leaving off the injections. Tuberculin is on its trial. There are many cases in which it does undoubted good, others on whom it seems to have no effect at all, others again to whom it does harm. It is difficult to say whether any particular case will benefit or not.

Perhaps the most likely to receive permanent good are those in an early stage, where the disease though apparently not advancing has existed for some time. It seems in these cases as though the natural forces of resistance in the patient are just not sufficient to overcome the condition, though enough to keep it in check, and that Tuberculin provides just that amount of impetus required to effect a cure. Where the disease is in a very early stage, either without physical signs, or with very few or indefinite, it is hard to say whether their disappearance and the improvement generally is due to the Tuberculin, or to other means of treatment. Those cases in which no improvement occurs until Tuberculin is begun, can fairly be claimed as having derived good from the injections. As to whether the benefit received is permanent, or the cure is indeed a cure, time alone can furnish the proof.

In conclusion we would urge the vital necessity for early diagnosis in this insidious disease, and the need of utilising every possible means of establishing the same. When the signs are definite, the symptons well marked, it is often too late for hope of any permanent cure, the case is now no longer early, but advanced. It is in the early stages that we must look to combat the disease with any degree of success.

Tuberculin as a means of diagnosis may be of use, but it is a two edged sword, and the danger of lighting up quiescent foci is too real to be lightly disregarded. In the words of Sir Clifford Allbutt[24] There is too much at stake; and even for the expert the number of 'normals' (or persons practically normal) who react is embarrassing" In the stage preceding the presence of physical signs, we must look for slight irregularity of temperature - a sub-normal temperature in the morning is suspicious, 98.5 at that hour is too high and even more suspicious (Clifford Allbutt),[24] and most characteristic, one with a swing from 97^{o} - 99^{o} at night. A 'feeling of tiredness, general malnutrition, poor chest development and expansion, with deficient air entry, all these go towards establishing an affirmative diagnosis. A positive Von Pirquet's reaction - with certain reservations - and, on X raying, a restricted movement of the diaphragm on one or other side, or shadows round the roots of the lungs are confirmatory; while a family history of phthisis or exposure to infection must give additional weight to the evidence. The opsonic Index in skilled hands may afford valuable information.

The earliest physical sign of value is the continued presence of crepitations, brought out often only by coughing. When expectoration is present the presence of Tubercle bacilli makes the diagnosis certain.

With regard to treatment, we shall increase the patient's resistance by an open air life, by a diet

with a due proportion of proteid and fat, the latter added to by cod liver oil, and by the exhibition of creosote. We shall regulate his auto-inoculation by rest, coupled with graduated exercises and labour, and in suitable cases by the use of Tuberculin. The exercises should aid in improving the general musculature, the chest expansion and development and the air entry.

Preventitive treatment consists in removing the child from debilitating conditions, and surroundings, in his avoidance of chills and colds, and in attending to his teeth, throat, bowels and other organs and functions, so that a high standard of health may be reached and maintained.

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11. Pyorrhoea Alveolaris, Joseph Turner, Practitioner, Jan. 1910.

12. Ways of Entrance and Spread of Tubercle bacilli in the body, W.Carr, Practitioner, Jan. 1913.

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20. Localisation of Lesions in Phthisis, 1880, Kingston Fowler.

21. Clinical Examination and Treatment of Sick Children, John Thomson.

22. Diagnosis and Treatment of Pulmonary Tuberculosis in the Early Stages, by Arthur Latham, Prac. Jan. 1913.

23. On the 'controlled' therapeutic use of New Tuberculin, by E.A.T.Rigg.

24. Introduction to Tuberculosis Number of Practitioner, Jan. 1913.

Selected Cases. 15.

Case 1. R. Lambert. 13. Adm: Dec. 11. 1912. as Incipient Phthisis.

General Condition.

Good. Big girl for age. Carious teeth. Breath foul.
No glands or scars. Throat. Tonsils ++.

Symptoms. Indefinite.
No cough or Expectoration. No night sweats.

General Signs.

Temperature. occasional rise to 99°. nightly.

		stone	lbs.	ozs.
Weight.	Dec 12. 1912.	6	2	8
	Mar 6. 1913.	7	1	0.

Blood Pressure. 105 mms of Hg.

Von Pirquet's Reaction. +.

X Rays. No definite abnormal shadowing.

Cardiac System. Heart. MA. 1st sound impure. Otherwise nil.

Respiratory System. Expansion of Chest fair — improving. No flattening.
No adventitiae. Nil to note.

Chest Measurement.

Dec 12. 1912.	29" - 31"
Jan 13. 1913.	29½" - 31"
Mar 3. 1913.	29½ - 32.

Distance Smith's Sign +. No sternal or costal dulness. No interscapular signs.
No prominent veins on thorax. right.

Treatment. Creosote mv - mx. Class. A. on admission.
Mist. of Month. 3os. " D. Dec 16 1912.
" F. Jan 13. 1913.

Case II. S. Jeffes. 5½. Adm. May 16. 1912. as Early Phthisis.

General Condition. on adm:

Pale. anaemic. General nutrition. poor.
Teeth good. No glands or scars. Throat nil.

Symptoms. TB. diathesis.

Easily tired. Cachadaxical. No cough or Expectoration. No night sweats.

General Signs.

Temperature. swings from 97° or 98° – 100° nightly.

Weight. May 21st 1912. 2 - 9 - 2.
Jan 31st 1913. 2 - 12 - 2. Tuberculin given.
Mar 5th 1913. 2 - 13. 0.

Blood Pressure (systolic). 88 mms of Hg.

Von Pirquet's Reaction. +.

X. Rays. No definite abnormal shadowing.

Respiratory System. Lungs. No dulness in note. VR. VF. unaltered.

On adm: Few fine crepts at R. upper apex. post.
Breath sounds as a whole indistinct.

Aug 21. 1912. A few fine crepts at R. upper apex post.

Oct 8 1912. Lungs. nil definite.

Chest measurement.

May 23. 1912. 21" - 23½"
Dec 9. 1912 22" - 23½" ? difficult to obtain.

Sputum Smith's Sign –. No charnel or costal dulness. No tuberculous signs.

Present Condition. General nutrition. fair.
No cough or Expectoration.

Lungs. L. apex. post. Creaking breath sounds. Otherwise nil.

Treatment. Creosote mj - mx.
Mist. Ol Morrh. 3os.

Tuberculin. Jan 31. 1913. 2½ mmls BE.
Mar 7 1913. 5·0 mmls BE.
Two Reactions. Slight

Class A. +. Oct 20 1912. No reduction of Temp.
" A. Dec 5. 1912. Temp. Lower.
" B. Dec 16 1912. Temp definitely lower.
" C. Jan 7 1913
" E. Jan 31. 1913. Temp almost level.

Case III. M. Schroder - 14. Adm: Aug 20. 1912. as Pulmonary Tuberculosis.

General Condition. on adm:

Development fair. Carious teeth. Breath foul. anaemic.

No glands or bones. Throat. inf.

Symptoms. Always feeling tired. always ailing.

No Cough. Occl. Expectoration.

Sputum. Many polymorphs. No T B.

General Signs.

Temperature. Occasional rise to 99° nightly.

Weight. Aug 29. 1912. 5 - 6 - 0

Oct 17. 1912. 5 - 10 - 5 Tuberculin given.

Mar 5. 1913. 6 - 1 - 0

Blood Pressure (systolic). 102 mms of Hg.

Von Pirquet's Reaction. +.

X Rays. Vague shadows seen to Rt & L. of middle line in root area of lungs.

Diaphragm moves well.

Resp: System. Lungs. Expiration prolonged all over.

On adm: Few fine Crepts Rt apex Ant^r & both lower apices.

Nov 11. Lungs. Int abnormal.

Chest Measurement. Nov 16 1912. 28½" - 29½"

Dec 16 1912 28" - 30"

Jan 6 1913. 28½ - 30¼.

Mar 5 1913 29 - 30½.

Eustace Smith's Sign +. No sternal or costal dulness. No interscapular signs.

Pres: Cond: Temp. fairly steady. Weight improving.

Lungs. Int to note. No Cough or Expectoration.

Treatment. Class. C. Oct 24 1912.

Ord & Malt. — D. Dec 14. 1912.

Mist Ferri et Strych. — F. Jan 1. 1913.

Tuberculin. Oct 17. Minis 2. TR.

Mar 7. - 4000 BE.

Discontinued from Dec 17 1912 - Jan 5. 1913.

At first big reactions - general & local. not focal. Headache & vomiting.

No Temp. reactions now. Local. occasionally great. Vide Chart III

Case 4. A. Flood. 6. Adm: Nov 23. 1912 as Early Phthisis.

General Condition. Fair. Thin. Colour good.

Teeth good. No glands or scars. Throat. nil.

Symptoms. Indefinite. No cough or Expectoration.

General Signs.

Temperature. 98° - 100° usually. as a rule.

Weight. Nov 28. 1912. 2. 11. 8.

Dec 6. 1912. 2. 11. 14. Tuberculin begun.

Mar 6. 1913. 3. 1. 4.

Blood Pressure. 84. mms of Hg.

Von Pirquet. +.

X Rays. Shadows to Rt & Lt. of Spinal Column.

Emphysema moves well.

Resp: System. Lungs. Rt apex part dulness. VR & VF +. Breath sounds

On adm: markedly prolonged - Expiration - & almost amphoric. Inspiration reversed.

Dec 2. Signs improved - Expiration at R. apex part still prolonged.

- 16. Signs in lungs indefinite.

Chest measurement. Dec 6. 1912. 21½" - 22¾"

Mar 5 1913 22" - 23½"

Eustace Smith's Sign +. No sternal or costal dulness. No interscapular signs.

Pres: Cond: General Nutrition fair.

Mar 6. No signs in lungs. No cough or Expectoration. Temp. nil.

Treatment - Ord & Hall.

Class A. Nov 28 1912. No Reduction in Temp.

Dec 14 to Class B. Temp: gradually falling

Jan 7 1913. Class F.

Tuberculin. Nov 29. 1912. 5 Units TR. Temp: 100° at night.

Mar 7. 1913. 400 Units BE. Temp: steady.

Case V Boy Fellows. 12. Adm: Jan 20. 1911. as Pul: Tuberculosis.

General Cond: On adm: Pigeon chested. Pale. thin. nutrition poor.
No glands or scars. Throat. nil. Teeth good.

Symptoms. Cough. No Expectoration. No night sweats.
General tiredness & want of go.

General Signs.

Temperature. Irregular. 97° - 99° or 100° at night.

Weight. Jan. 26. 1911. 3 - 8 - 0
Jan 1 1912. 3 - 6 - 0
Jan 2. 1913. 3 - 7 - 0 Tuberculin begun.
Mar 6. 1913. 3 - 7 - 6

Blood Pressure. 94 mms of Hg.

Von Pirquet. +.

X Rays. Shadows to Rt & L of Spinal Column. Diaphragm moves better on the Rt side. No mottling in Lung substance.

Resp: System. Lungs: On adm. Rt. apex dull. Expiration prolonged unduly. but else.
May 11. 1911. L upper apex post. Few râles.
Jan 4 1912. L apex upper. post. Impaired note. Rhonchi over L. Lung & some râles
Rt Lung. Numerous coarse râles. No cough.
Sept 25 1912. L. upper apex. Crepitations.
Feb 24 1913. R. Lung. Ant. Occ Rhonchus. Otherwise nil.

Chest measurement.
May 16. 1912. 23" - 24½"
Nov 20 1912. 24" - 25¼"
Dec 16 1912. 25" - 26"
Mar 6 1913. 25" - 26½"

Eustace Smith's Sign +.

No clavicular or costal dulness.

No intercostal signs.

No prominent veins. right side.

Pres: Cond: No cough or Expectoration. Very anaemic.
Temp. very little. Very little improvement.
Weight. Loss of 10 ounces in over 2 years.

I. Fellows. Case V. continued.

Treatment.

Oil + Malt.

Mist. Ferri et Strych.

Liq Arsenicalis ♏ VIII t.d.s.

Plasmon.

Liq Pancreaticus 3ss t.d.s.

Thyroid Extract. gr ½-I t.d.s.

Tuberculin. Dec 1. 5 Units TR.

Mar 7. 600 Units BE.

Discontinued. Dec 17. 1912 — Jan 5. 1913.

Ho grent. resetorium. Temp: Level.

Class. A. for some time.

Class F. at present.

Case VI G. Kent. 13. Adm: Apl. 13. 1912. as Early Phthisis.

General Condition. poor. on adm:

Anaemic. Chest-development. fair. Teeth good.

No glands or ears. Throat nil.

Symptoms. Indefinite. Energetic.

No cough or Expectoration.

General Signs.

Temperature. none.

Weight. April 15. 1912. 3-6-0

Nov 2. 1912. 4-2-6 Tuberculin begun.

Mar 5. 1913. 4-4-0

Blood Pressure. 108 mms of Hg.

Von Pirquet +.

X Rays. Vague shadows to the R. & L. of Spinal Column.

Diaphragm moves well.

Resp: System. Lungs. No dulness anywhere. VR. VF. normal.

on adm. L lower lobe. 'crepe'

Nov 2. 1912. R. apex post. Expiration much prolonged. B.S. Semi-bronchial.

VR +. slightly. No dulness.

Cough & Expectoration.

Sputum. Many polymorphs. No TB.

Chest measurement. Nov 16. 1912. 25"-27"

Feb 12 1913. 26"-28½"

Eustace Smith's Sign. +. No sternal or costal dulness. No intercostal

Pres: Cond: General nutrition. V fair. Throat. Tonsils +. Vegus.

No Cough or Expectoration. Temp nil.

Treatment. Bed & Rest. Class. C. May 15. 1912.

— F Oct 17 1912.

Tuberculin. Nov 2. 2 units. TR.

Mar 5. 6000 units BE.

No reactions at any time except at beginning.

Case VII M. Ray. Ho. Adm: Oct 19. 1912. as Pul: Tuberculosis.

General Condition.

Colour good. Nutrition poor. Teeth good.

Throat nil. No glands or scars. Chest. Ala in type.

Symptoms. Much Cough. Profuse Expectoration.

Sputum. Many Polymorphs. No TB.

No night sweats. Lachadaïreal. Easily tired. Ailing.

General Signs.

Temperature. Irregular. up to 100° nightly. on adm.

Weight. Oct 24 1912. 3 - 10 - 8.
Nov 18 1912. 3 - 13 - 7 Tuberculin begun.
Mar 6. 1913. 4 - 4 - 11.

Blood Pressure. 98 mms of Hg.

Von Pirquet. ++.

X Rays. Shadowing of Rt upper & probably Rt middle Lobe.
On expansion of chest differences in the two apical shadows marked. Diaphragm moves less on Right side.

Resp: System. Lungs. Rt. apices. upper. back & front. dulness. bronchial breathing. VR ++. Fine creps. flattening. dim: movement. on adm:

L. Lung. clear.

Jan 22. 1913. Rt. apex. antr. supraclavicular. dulness not so marked.
BS. prolonged expiration marked. VR +-
Rt apex. postr. ditto.

Feb 19. 1913. Rt supraclav: region. slight impairment of note.
BS. normal. VR + slightly.
Postr. nil.

Chest Measurement. Nov 18 1912. 24½" - 25½"
Jan 12. 1913. 25¼" - 27"
Mar 6 1913. 25½ - 27½".

Eustace Smith's Sign +.

No sternal or costal dulness.

No intercostal signs.

No prominence of veins.

M Ray. (Con).

Pres: Cond: No cough or Expectoration.
General Nutrition. Very good.
Weight increasing.
Temp. still slightly irregular.

Treatment.

Creosote ℞ 11-x. Tuberculin.
Mist ol Morrh. ʒss. Nov 19. 1912. 2. units TR.
Mar 7. 1913. 800 units BE.
Discontinued Dec 17 - Jan 5. 1913.
No great reactions.

Class A. Oct 19. 1912.
" B. " 24. 1912. Creosote Inhalations.
" D. Dec. 14. 1912.
" F. Feb 3. 1913.

Case 8. h. Ward. 5. Adm: Mar 12. 1912. as Pul: Tuberculosis.

General Condition.

Pale. anaemic. Nutrition poor. Teeth good.

Throat. nil. No glands or scars.

Symptoms. Cough. No expectoration. No sweating.

General Signs.

Temperature. Irregular. 97°-100° nightly.

Weight. Mar 25. 1912. 2-4-3.

Oct 17 1912. 2-7-4. Tuberculin begun Nov 13 1912

Mar 5 1913. 2-9-8

Blood Pressure. 80 mms of Hg.

Von Porquet. +.

X Rays. Distinct shadow at root of Right lung spreading into lung. Right diaphragm moves less than left.

Resp: System. Lungs. Expansion poor. L. upper apex post^r crepts.

L. Lung. Expiration prolonged all over.

July 25. 1912. Nil definite in Lungs.

Mar 6 1913. Lungs. nil to note. Air entry not good.

Chest measurement. May 16 1912. 20½" - 22"

Dec 12 1912. 21" - 22". Difficult to obtain.

Eustace Smith's Sign +. Pulmo over manubrium. No costal pulmo.

No intercostal signs. No prominence of veins.

Pres: Cond: No cough or Expectoration.

Lungs nil to note. Throat. Tonsils ++. Improved.

Treatment. Oil & Malt. Tuberculin. Nov 13 1912. 2 milli TR

Mar 7 1913. 1000 milli BE.

Omitted Dec 17. 1912 - Jan 5. 1913.

No great reactions. Temp Level.

Class A +. Oct 20 1912. No reduction of Temp.

" A. Dec 5 1912. " " " "

" B. Dec 11. 1912. " " " "

" E. Jan 19. 1913. Temperature less.

Case IX. W. Senior. T. Adm. June 6. 1912. as Early Phthisis.

General Condition. Colour good. Teeth carious. Breath foul.
Nutrition poor. No glands or scars.
On adm: Throat. Tonsils +.

Symptoms. "Lazy child" Lolls about. Always ailing.
No cough or Expectoration. No night sweats.

General Signs. Temp irregular 97°-99°. Nightly.
Weight. June 7. 1912. 3-3-8.
Oct 17. 1912. 3-6-14. Tuberculin begins Nov 13. 1912.
Mar 6. 1913. 3-11-0
Blood Pressure. 96 mms of Hg.
Von Pirquet. +.
X Rays. Shadow to left of middle line. Diaphragm moves less on left side.

Resp: System. On adm.
Lungs. Rt. apex ant. B.S. weaker than left — tubular — no adventitiae.
Aug 16. 1912. L. Ant apex. Dulness. Mil on auscultation.
Oct 2 1912. Lungs. No signs. Early clubbing of fingers.
Mar 6 1913. Lungs. Not to note.

Chest Measurement. 23" — 24½"? Child so stupid, could get no measurements.

Eustace Smith's Sign —. No sternal or costal dulness.
No interscapular signs. No prominence of veins.

Pres: Cond: Cough at night. No expectoration. Wt. increasing.
Temp: more level

Treatment. Creosote mj ii - mj x
Mist. Ol Morrh. ℥ss.

Tuberculin.
Nov 13 1912. 2½ mils TR.
Mar 7. 1913. 1250 " BE.
Big reactions at first. None now.
Temp. fairly level.

Class A +. Oct 22. 1912. No Reduction in Temp.
" A. Dec 6 1912. Tuberculin begun. Nov 13. 1912.
" B. Dec 11. 1912.
" E. Jan 12 1913. Temp. occ. rise to 99°.

Case 10. B. Flatman. 10. Adm: May 17. 1912. as Pulmonary Tuberculosis.

General Condition. on adm:

Nutrition. very poor. pale. thin. Teeth good.
No glands or scars. Throat: nil. Chest: ala. T.B diathesis.

Symptoms. Want of energy. delicate child.
Cough at night. No expectoration. No night sweats.

General Signs.

Temperature. Swing from 97°-99° with rises to 100° nightly.

Weight. May 21. 1912. 3-2-2.
Oct 17 1912. 3-6-0 Tuberculin begun.
Nov 6 1913. 3-9-0.

Blood Pressure. 96 mms of Hg.

Von Pirquet. +.

X. Rays. Nil abnormal seen in chest.

Resp: System. on adm.

Lungs. Rt. dim: movement. Expiration prolonged all over. No creps.
TR & VF normal.

Aug 1. 1912. Chest ala-marked. Expansion poor.

Nov 11. 1912. Rt apex upper ant. flattened. note slightly dull. No creps.
Post: Rt. apex. Crackling rales. BS. weaker on Rt side.
Inspiration poor.

Chest Measurement. May 23. 1912. 23"-23¾"
Sep. 10 1912. 22½"-24"
Jan 6 1913. 22½-24½"

Eustace Smith's Sign +. No sternal or costal dulness. No intercostal recession.

Pres: Cond: Nil definite in lungs. except poor development of chest & poor air entry. General Nutrition fair.
No cough or Expectoration. Temp lowered.

Treatment: Oil & Malt. Tuberculin.
Oct 17. 1912. 1½ mills TR.
Nov 7 1913. 4000 " BE.

Class A for some time
Class F.

Omitted from. Dec 17. 1912. — Jan 5. 1913.
Oil retention. Temp much lowered.

Case XI.

1. Bugge. U. Adm. Feb 7. 1912. as Early Phthisis.

General Condition. On adm:

Pale. Hem. poorly developed. Fingers clubbed.
No enlarged glands or scars. Teeth good. Throat-inf
Chest flat & ales in type.

Symptoms. Some cough at nights. No expectoration. No sweating.

General Signs.

Temperature. Irregular. 97°-99° with occasional rises to 100°-102° at nights.

Weight. Feb 12 1912. 3-9-0.
Oct 17 1912. 4-0-14. Tuberculin begun.
Mar 6 1913. 4-4-0.

Blood Pressure. 88 mms of Hg.

Von Pirquet. +.

X. Rays. Inf definite. Picture not clear cut. Hazy. Emphysema more well both sides.

Resp: System. On adm.

Chest flattened. Expansion very poor
Lungs. Rt apex ant. note impaired. B.S. feeble. Expiration prolonged. No adventitiae.
April 11. 1912. L. Lower apex. Fine creps. Expansion much improved.
Rt apices. clear.
Aug 16. 1912. Rt apex ant. dulness & few fine hair like creps.
Rt post apices. Occ. hair like creps.
Oct 2. 1912. Slight Expectoration. No TB in sputum.
Jan 1. 1913. Lungs. Inf definite.
Mar 3 1913. Lungs. Clear.

Chest Measurement.

May 30 1912. 24"-27". "greatly improved". No previous measurements.
Nov 18 1912. 25½"-27½"
Jan 13 1913. 26½"-28"

Eustace Smith Sign —. No sternal or costal dulness.
No interscapular signs.

Case XI (Con).

Pres: Cond:

Temp. very much steadier.

Lungs. cl. Weight- increasing.

No cough or Expectoration. Reconstruction. Mar. 1913.

Treatment-

Ord Hall-. Tuberculin.

Inst- Teni et Strych. Oct-17. 1912. 2 mils. TR.

Plasmon. Mar 7. 1913. 4000 " BΣ.

Omitted. Dec 17. 1912 — Jan 6. 1913.

Big Reactions. general aloes at first.

Class A. for some time.

- B. Dec 1. 1912.
- D. Dec 14. 1912.
- F Jan 1. 1913.

Case XII.

Alice Dean. U. Adm. July 27. 1912. as Early Phthisis.

General Condition Bn adm.

Pale. Ill nourished. No glands or scars.
Teeth carious. Breath foul. Throat. clear.
Nose. Septum deflected to Right.

Symptoms - Indefinite.
No Cough. No expectoration. No sweating.

General Signs.
Temperature. Occ rise to 99° - 100° nightly.
Weight.
July 20 1912. 3 - 5 - 8.
Oct 17. 1912. 3 - 8 - 8 Tuberculin begun.
Mar 6. 1913. 3 - 12 - 0.
Blood Pressure 95 mms of Hg.
Von Pirquet - ++.
X. Rays. Outlines of a cavity Rt upper Lobe. Light area with thick band all around. Diaphragm movement - re-stricted on Right side.

Resp: System. Bn adm.
Lungs. Rt. apex Ant. BS. higher pitched. Note not impaired.
VR. VF normal
Rt. lower lobe. Crepts.
Expansion poor but equal.

Nov 11. 1912. Lungs. Rt apex ant. deficient expansion. Note dull. BS bronchial, expiration markedly prolonged.
Rt apex post. Note dull. expansion deficient. BS. faint all over Rt lung post. tubular in type. VR ++. VF ++.
Pectoriloquy.

Mar 6. 1913. Lungs. Rt apex ant. Note duller than left. somewhat boxy, flattening & dim: movement. BS. supra & sub: cavernous bronchial, almost amphoric. VR ++. whispering pectoriloquy. VF ++. Expiration greatly prolonged. Cavity Rt apex.
Rt apex post. ditto. Left Lung. clear.

Case 12. (Con).

Chest Measurement.

Aug 6 1912. 24" - 25."
Nov 16 1912. 24½" - 26"
Dec 16 1912. 24½" - 26"
Mar 6 1913. 25" - 26½"

Eustace Smith's Sign +.
No General dulness or Cogwheel.
No subscapular signs.
Veins prominent. R. side of thorax.

Lungs: Cond. No Cough or Expectoration.
Temp: Steady.
Weight: Slow increase.
General Nutrition very fair.

Treatment.

Oil + Malt.
Syr. Ferri Iodidi.
Plasmon.

Tuberculin.

Oct 17. 1912. 2 mill. TR.
Mar 6 1913. 4000 " BE.

Creosote Inhalations. Omitted Dec 17 1912 - Jan 5 1913
No general reactions.
Two by local reactions.

Class F all the time.

Case XIII

Mary Sinclair. 11. Adm. Mar 19. 1912. as Early Phthisis.

General Condition.

Plump. not anaemic. Teeth good.
No glands or ears. Throat clear.

Symptoms.

Tired feeling. No sputa.
No Cough or Expectoration. No night sweats.

General Signs.

Temperature. Morning. 97°–99° & 100° mostly.

Weight. Mar 25 1912. L – 7 – 2.
Nov 13 1912. L – 12 – 0 Tuberculin begun.
Mar 6 1913. 5'– 1 – 0.

Blood Pressure. 85 mms of Hg.

Von Pirquet. +.

X Rays. Mottling in the region of Rt apex. not very dense. Emphysema more less on the Right.

Resp: System. On adm:

Chest fairly well developed. Expansion good. but to note on Lungs.

June 27. 1912. Lungs. Rt axillary line – lower lobe. note impaired.
Rt lower lobe. Crepts.

Aug 3 1912 Sputum. negative. TB.

– 8 1912. Signs have cleared up except crepts in Rt upper lobe in front.

Dec 2. Lungs. Rt. in front. 'crepts' especially in Rt axillary line be-
1912. low transverse Nipple line. Coughing a lot. No expect. No TB.
found in sputum.

Jan 27 1913. Lungs. Rt ant. Med Viles & rhonchi. over by 6. 8.
Post. ditto.
Left Lung. clear.

Mar 5. Lungs. Rt. ant. Crepts. occ rhonchus. No dulness. Expiration
prolonged.
Post. Crepts at apex.
Left. Clear by 6. 8. all over.

Case XIII

Chest Measurement -

May 11. 1912. 26" - 27½"
Nov 16. 1912. 27" - 28½"
Dec 16 1912. 27" - 28½"
Jan 15 1913. 26½" - 28½"

Eustace Smith's Sign —.
No sternal or costal dulness.
No interscapular signs.
No prominent veins.

Pres: Cond: Temp. fairly steady.
Wt. slow progress.
Slight cough.
No expectoration.
General nutrition good.

Treatment. Tuberculin.

Creosote ℳ ii - ℳ xii Nov 13. 1912. 2 units. TR.

Ext. O. M. ʒss. Mar 6 1913. 800 units BE.

Linctus Camph Co. Omitted Dec 17 - Jan 5 1913.

(nocte). One big reaction.

Creosote inhalations.

Class A for some time.
Class C. Dec 1. 1912.
Class D Dec 14 1912.
Class F. Jan 1. 1913.

Case XIV.

E. Brown. 13. Adm Oct 8 1912. as Phthisis.

General Condition. Very bad. Pale. emaciated.
Chest very poorly developed. TB. dullness.
Teeth good. Throat clear.

Symptoms.
Cough. Expectoration profuse. Night-sweats.
Wasting marked. Dyspnoea. No appetite. General weakness.

General Signs.
Temp: varied 98.4° — 101° & 102° & 103° at night.
Weight.
Oct 8 1912. 3. 11. 4.
Dec 12 1912 4. 3. 12. Tuberculin begun.
Mar 6 1913. 3. 13. 0 Considerable loss. then steady gain.
Blood Pressure. 96 mms of Hg.
Von Pirquet +.
X Rays. Too ill to go to X Ray department.

Resp: System. On adm.
R. Lung. Much flattened. Expansion restricted. dull. BS. bronchial to pari amphoric. Rales all over R + all over more at base than apex. Whispering Pectoriloquy at base.
L. apex post. Note impaired. 'Crepi' L Base. crepo.
Heart pulled over to Rt. nipple line.
Oct 11. 1912. No TB found in Sputum. Cells polynuclear. Small cocci in abundance.
Dec 9. Rt. Lung. Moist sounds not so many. Much the same otherwise. TB in Sputum in abundance.
Feb 5. 1913. Rt. Lung. Ant^r much as above.
Post^r as before. moist sounds fewer & signs altogether not so marked
Left Lung. BS harsh respiration prolonged.

Case XIV (Con).

Chest Measurement. Not taken.

Pres: Cond: Improved.

Lungs. Not greatly improved as regards physical signs.

Temp: much lower & steadier.

Weight. after a good drop with Tuberculin has steadily risen again.

Cough better. No expectoration. No night sweats.

Treatment.
Creosote m III - m XIV.
Mist. Ol Morrh. ℥ss t.d.s.

Inhalations. 16 hours a day.
Carboluria followed until
Carbolic acid was left out.

Tuberculin.
Nov 14. 1912. 2½ units TR.
Mar 6 1913. 350 " BE.
Omitted from Dec 17 1912 — Jan 5 1913.
No reaction - general - focal or local.
Temp: much lower.

Muslin Camph Co. mask.

Since admission. Class A.
Oct 20 - Dec 6. Class A+.
Since Dec 6. Class A.

Case XV

N. Sampson 2. Adm. Nov 5 1912. as Pulmonary Tuberculosis.

General Condition.

Very fair. Colour good. No glands or scars.

No signs of Rickets or Syphilis. Throat nil.

Symptoms. Cough. Expectoration at times. Not fretful. Motions normal. Cold extremities. No dyspnoea.

General Signs.

Temperature Irregular. 97° - 99.6° or 100° nightly.

Weight. Nov 7. 1912. 1 - 7 - 4.

Jan 31 1913. 1 - 8 - 12 Tuberculin begun

Mar 6 1913 1 - 9 - 10.

Von Pirquet + . not marked.

Resp. System. On adm.

Lungs. Left. Very dull all over. B.S. faint. VR –

left apex post^r note resonant & b.s. tubular.

Rt lung clear.

Heart. No displacement.

Chest explored twice before adm: nil found.

Nov 13. 1912. L. lung. not so dull. Coarse moist sounds all over both lungs.

Expectoration. No TB in sputum. Some staphylococci.

Dec 16. 1912. Both lungs examined with moist sounds. Dullness fairly equal. Heart not displaced

Jan 27 1913. Left lung. ant^r. B.S. hardly heard. No great dullness. VR – . movement diminished. Not a moist sound heard.

Rt lung. fairly normal.

Feb 3rd 1913. Left lung Ant^r Moist sounds all over.

Post^r. R & L. Numerous coarse moist sounds.

Feb 24. Rt lung Ant^r. Clear

Post^r. Moist râles everywhere.

Case XV (Con).

Resp System (Con).

Feb 24. 1913. Left Lung. back & front. packed with moist sounds. B.S. not heard. No dulness. Heart not displaced.

Pres: Cond:

weight improving.

Temp: level. Cough better. No expectoration.

Treatment.

Creosote $\overline{\text{m}}\,\text{III} - \overline{\text{m}}\,\text{XIV}$

Mist. Alb. Mixt. $\overline{\text{3}}\,\text{III}$

Creosote Inhalations.

16 hours a day.

Lactic Camph Co.

Mixt. Pot. Iod. & Belladonna.

Tuberculin.

Jan 31. 1913 2 Mmls. BE.

Mch 7. 1913. 40 mmls BE.

One slight reaction - temperature.

No local reaction.

Class A. On admission.

" B. Dec 16 1912.

" D. Jan 15 1913.

Tuberculin Charts.

Tuberculin Charts.

Chart 1. Typical of many. Reactions - temperature - not regarded up till January 1913. Shows increase in weight when Tuberculin was discontinued, and a levelling of the temperature on the whole, with a steady increase in weight.

Chart 2. Shows a continuous well marked increase in weight from the time of admission due to change of environment & good conditions of living. Physical signs slight. Occ rise of Temp. Von Pirquet +. No Tuberculin given. The rise of Temp: to 102.6 is accompanied by a drop in weight.

Chart 3. Reactions disregarded up till January. Increase in weight notwithstanding. On restarting B.E. shows the initial drop in weight followed by a steadily rising weight line. Temp: on the whole more level. As a rule now we omit the Tuberculin for a week or ten days when the dose has reached 1000 units. Shows too the general reactions. headache etc at first.

Chart 4. Shows a lowering of the Temp: since January with steadily increasing weight. Pulse is seen to be more rapid than normal on occasions. A fairly typical chart.

Chart 5. Shows a level weight line. No increase in weight in this child for one two years. Tuberculin has not benefitted her at all. The rise of temp: to 101° was due to a sore throat. Shows the marked drop in weight as the result of it.

Chart VI. Shews practically no reaction to BE. since January 16. 1913. Local reactions very slight. The figures denote varying degrees of local reaction ½ & 1. mean slight, 2-3-4 more severe. 4 would indicate a swollen, hot & reddened arm with pain in the axilla. The dosage was raised more rapidly than usual, as there were no reactions. The weight has increased but not markedly.

Chart 7. Shews an occasional reaction since January with steadily rising weight. Signs in lung in this child fairly advanced. She has much improved. The doses have been much more slowly advanced than in the last case. Local reactions slight.

Chart 8. Shews the very irregular temp: at first; also that the treatment in bed in Class A + as enteric failed to reduce the temp: After 3 weeks Tuberculin was begun with no success with regard to reduction of pyrexia - reactions were disregarded. Shews subsequent fall in temp:, which has remained more level under the influence of BE. Slight local reactions. Weight shews slow increase, also the stationary weight line on first restarting Tuberculin, followed by a steady rise.

Chart 9. Shews the very irregular temp: - the failure of Class A + to reduce it, or Tuberculin combined with the baths - reactions disregarded till January. Shews the subsequent reduction maintained by BE - the stationary weight for some time after restarting BE, followed by a steady rise. Local reactions slight.

Chart X. Shows the irregular temp:, with two marked drops in weight without rise in temp: & the following rise in the weight line. Shows too the big local reactions obtained, but no general or focal ones. After every injection this child has a most swollen & painful arm. Injections are now given every week or ten days. Very sensitive to Tuberculin.

Chart XI. Shows 3 months of stationary weight with irregular temp: followed by a gradual increase, and lowering of the latter by means of Tuberculin. This child has improved in many ways since its administration. No general reaction one big focal reaction.

Chart XII. Shows no reduction of temp: with Tuberculin with one big rise & with 3 big local reactions. – no general or focal – hyperæsia in this child well marked. Nevertheless the weight is increasing well, otherwise the Tuberculin would have been discontinued or given at much longer intervals. Shows then hyperæsia, & local reactions to tuberculin with simultaneous increase of weight, which is unusual.

Chart XIII Shows an irregular temp: reduced by Tuberculin. hyperæsia in this child quite definite. weight has varied but on the whole has increased. One general reaction – temp: 103° with headache & vomiting. This has been a rare occurrence. Local reactions slight. Tuberculin should have been omitted for a week or more after the general reaction. After temp of 100.4° of Feb 19, omitted for a week. Now being given every 10 days.

Chart XIV. Shews a very irregular temp: unaffected by treatment in Class A+. The drop in temp: after Tuberculin was given very carefully in January. The weight dropped at first then slowly rose, but has dropped slightly again. This child has advanced phthisis. Though Tuberculin has reduced the temp:, & the weight continues to fall, it seems to indicate discontinuing the Tuberculin.

Chart XV. Merely shews how the treatment in Class A+ has reduced the temperature. Signs in this child quite definite. Temp: has remained down since its first fall.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

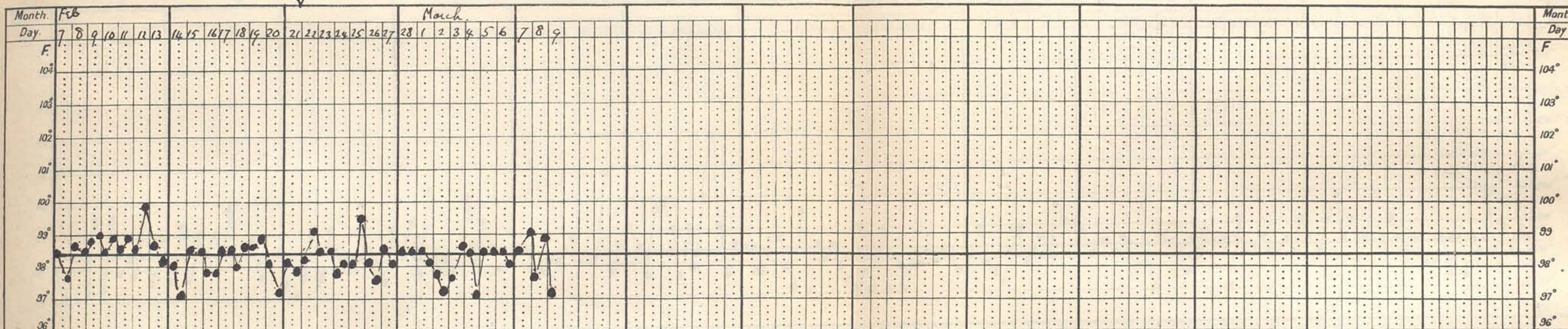
Coward

TUBERCULIN CHART. 1.

Ward ____ No ____

Name Annie Chauley . Age 9. Born ____ (Normal Weight ____) Admitted ____ Disease ____

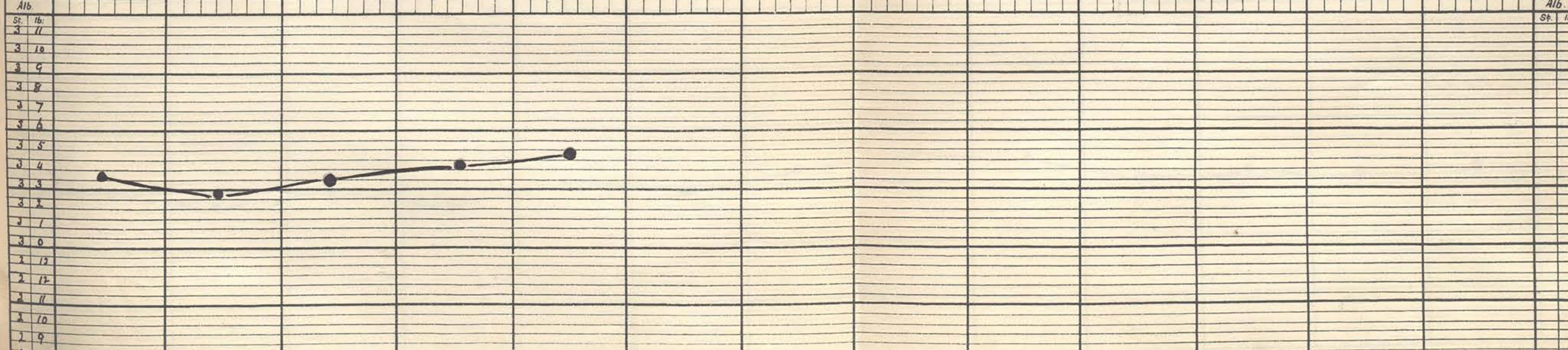
Month: Feb / March
Day: 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 1 2 3 4 5 6 7 8 9



Pulse. / Respirⁿ / Motions

Exercise Class.	F								
Tuberculin	250	300	300	350	400	400	450	500	600
Local Reaction	½	½	½	½	1	1	1	1	1
Headache									

Cough / Expectⁿ / Haem. / Alb.



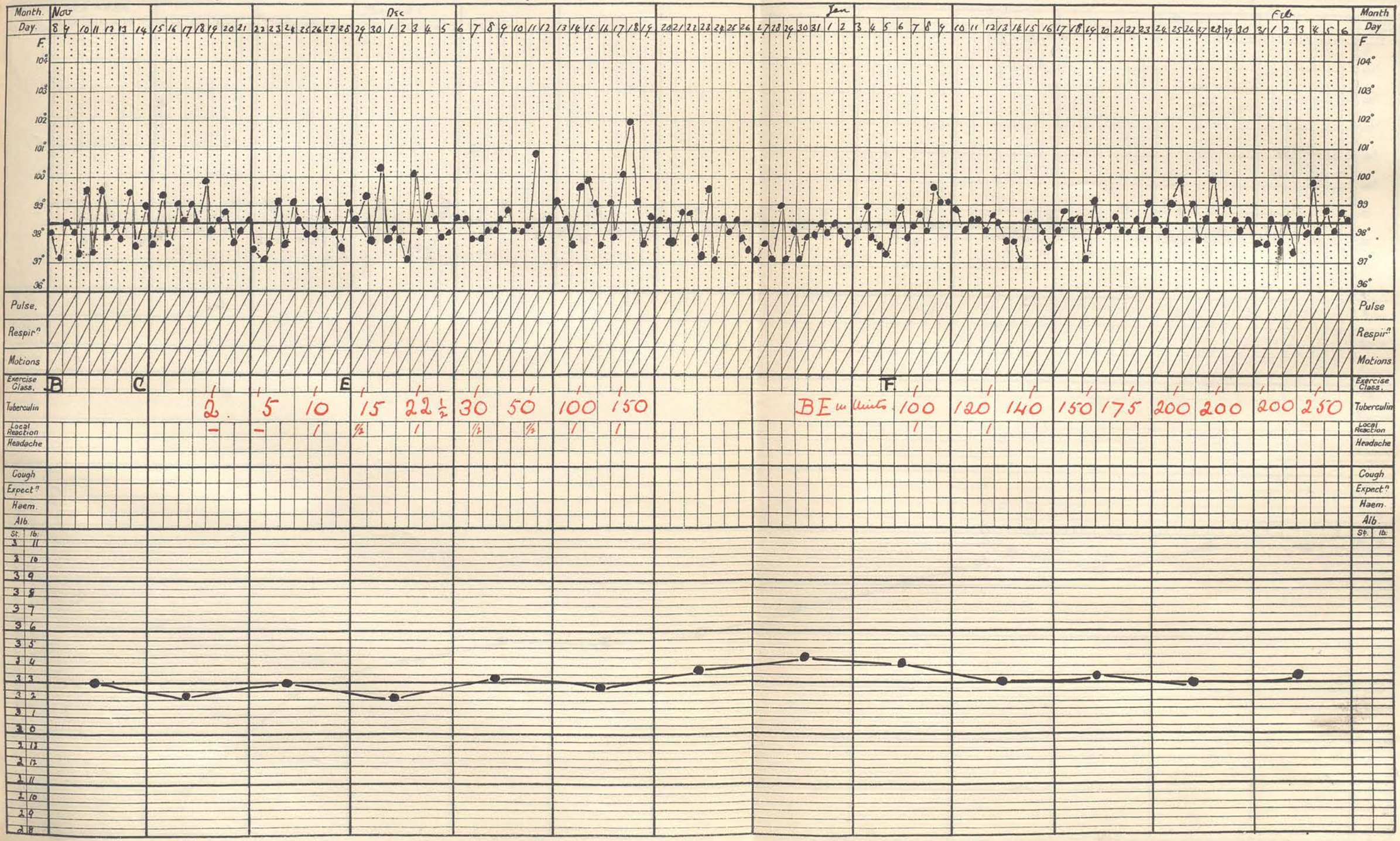
METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART.

Ward E 6.

No.

Name Annie Chantley . Age 9. Born Jan 6 1903. (Normal Weight 4- 0- 0) Admitted June 25. 1912 Disease Pulmonary Tuberculosis



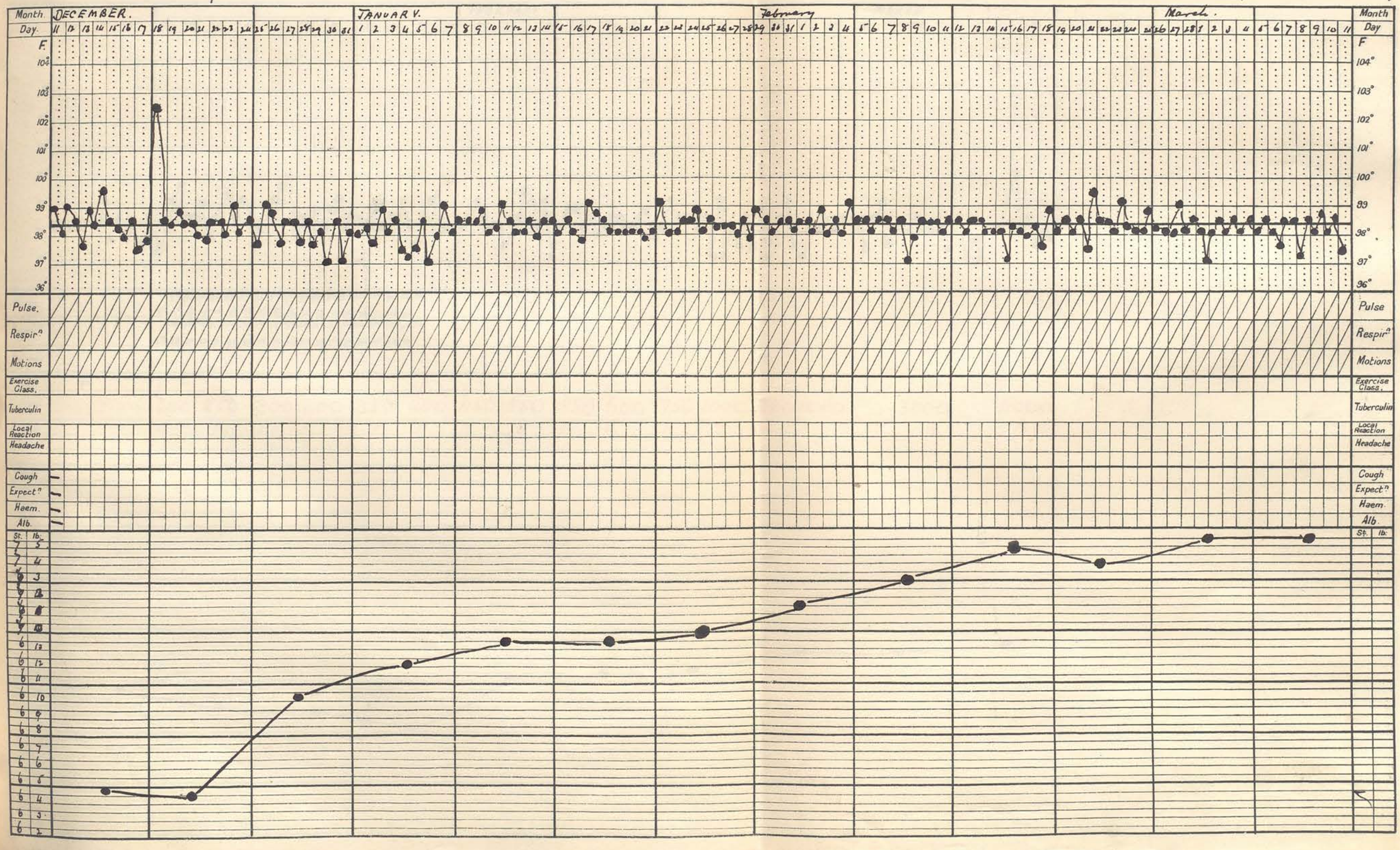
TUBERCULIN CHART. 2.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward E 5

No.

Name Beatrice Morgan. Age 12. Born (Normal Weight) Admitted Dec 11. 1912. Disease Early Pulmonary T B.

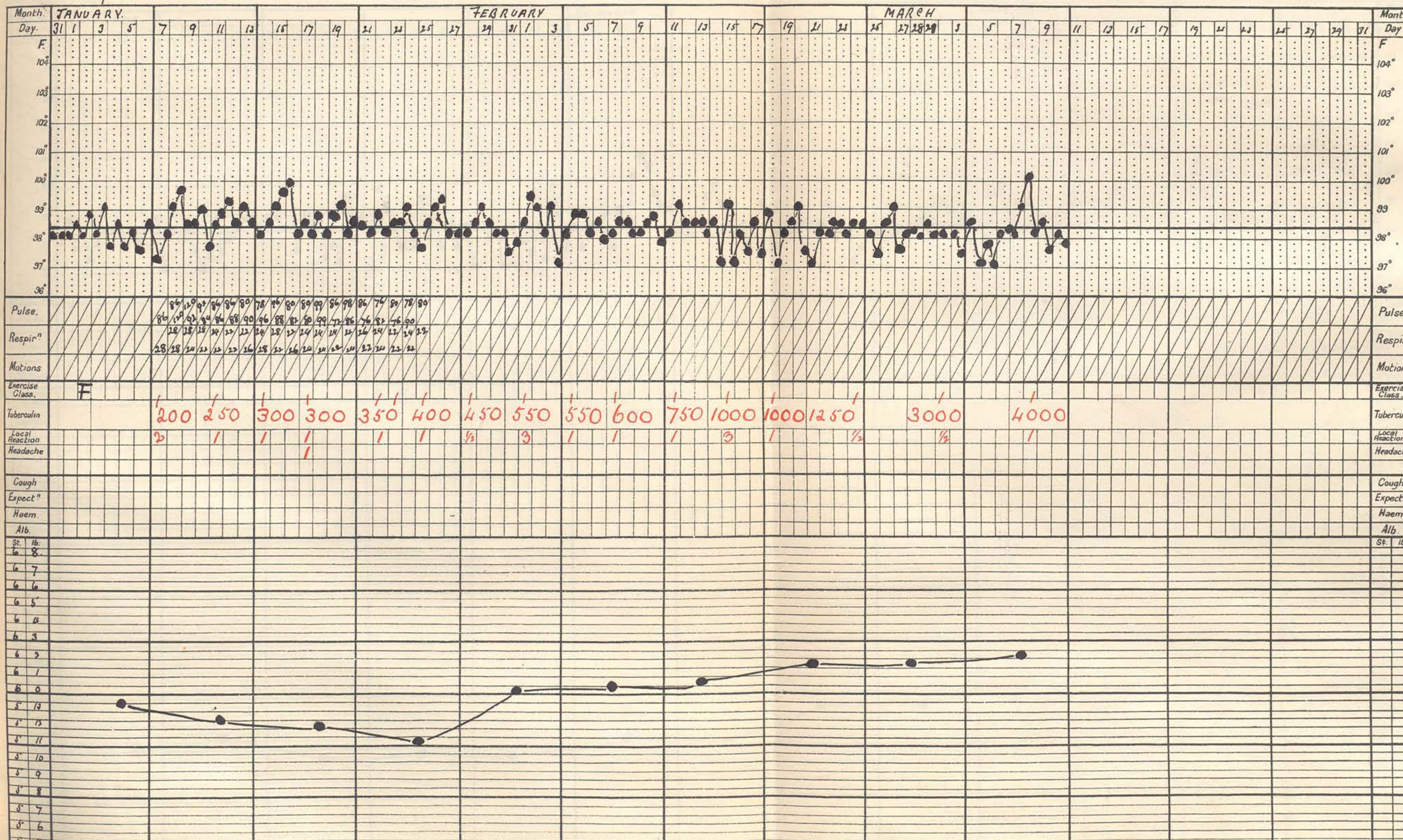


TUBERCULIN CHART. 3.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward ____ No. ____

Name Margaret Schroder . Age 13 . Born ____ (Normal Weight ____) Admitted ____ . Disease ____

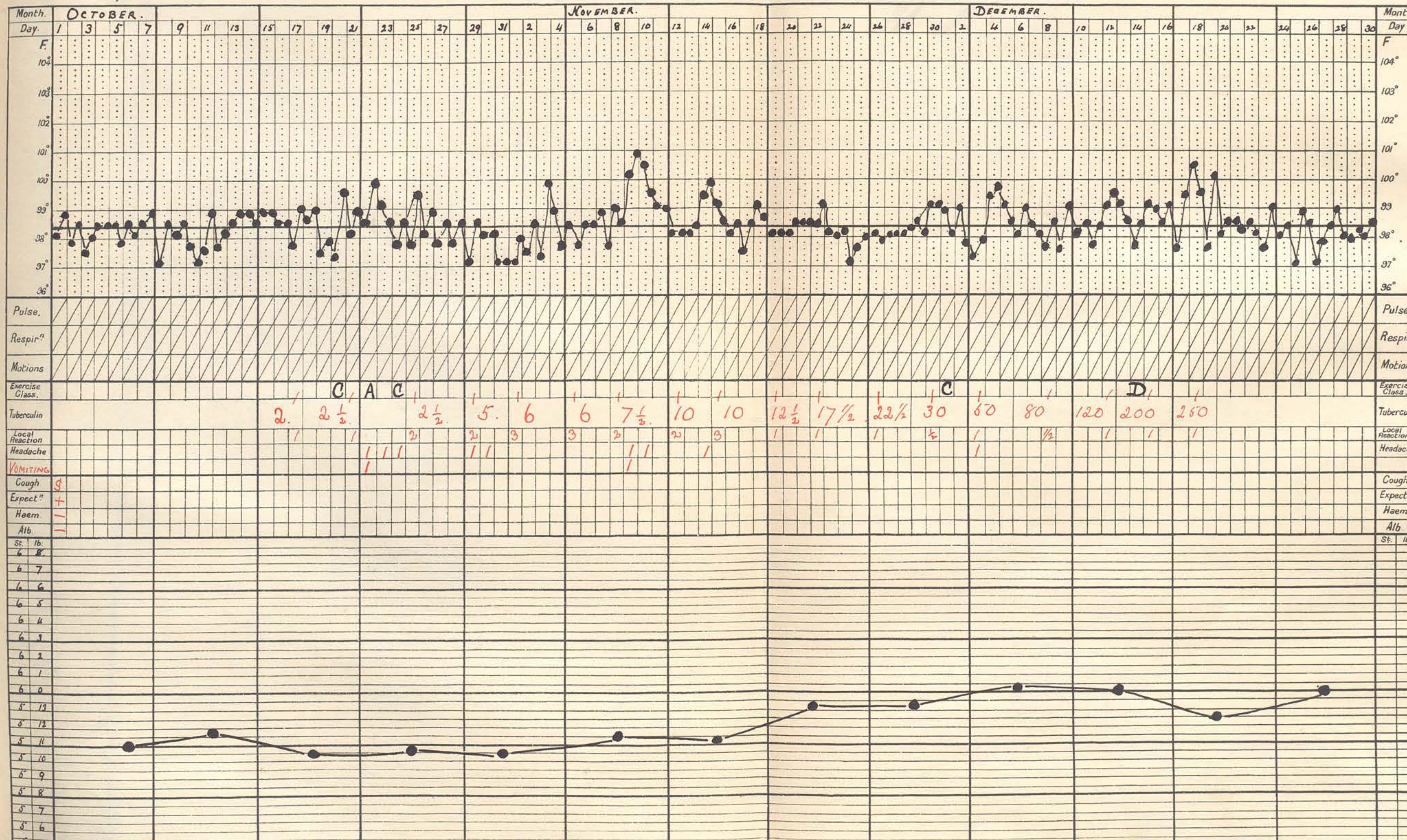


TUBERCULIN CHART.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward E6.
No.

Name *Margaret Schrode* . Age *13.* Born *July 14. 1898.* (Normal Weight *7 - 0 - 0*) Admitted *Aug 20 1912.* Disease *Early Phthisis*



METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART. 4.

Ward E 6.

No 6579

Name Annie Flood. Age 6. Born June 11th 1906. (Normal Weight 3. 0. 12.) Admitted Nov 23rd 1912. Disease Pulmonary T.B.

Month.	Feb					March																										Month.
Day.	21	23	23	24	25	26	27	28	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	Day.
F.																																							F.
104°																																							104°
103°																																							103°
102°																																							102°
101°																																							101°
100°																																							100°
99°																																							99
98°																																							98°
97°																																							97°
96°																																							96°
Pulse.																																							Pulse
Respir^{n}																																							Respir^{n}
Motions																																							Motions
Exercise Class.	E																																						Exercise Class.
Tuberculin										350			350			400																							Tuberculin
Local Reaction										/			/			/																							Local Reaction
Headache																																							Headache
Cough																																							Cough
Expect^{n}																																							Expect^{n}
Haem.																																							Haem.
Alb.																																							Alb.
St. lb.																																							St. lb.
4 0																																							
3 13																																							
3 12																																							
3 11																																							
3 10																																							
3 9																																							
3 8																																							
3 7																																							
3 6																																							
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3 0																																							
2 13																																							
2 12																																							
2 11																																							

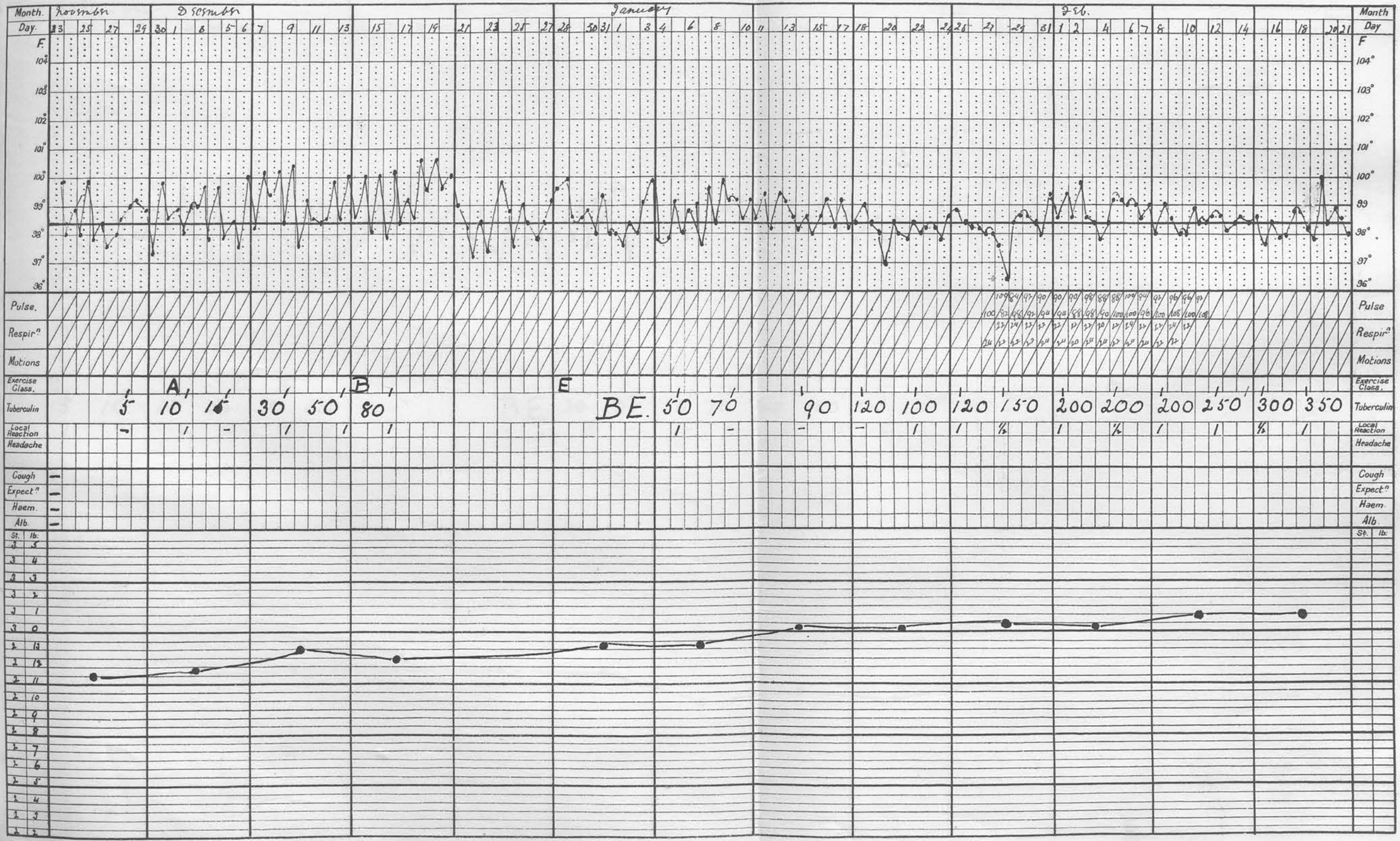
3 C in hosp.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART.

Ward Ethel
No. 6579

Name Annie Hood . Age 6 . Born Jan 11th 1906 . (Normal Weight 3 - 0 - 12) Admitted Nov 23rd 1912 . Disease Pulmonary T B

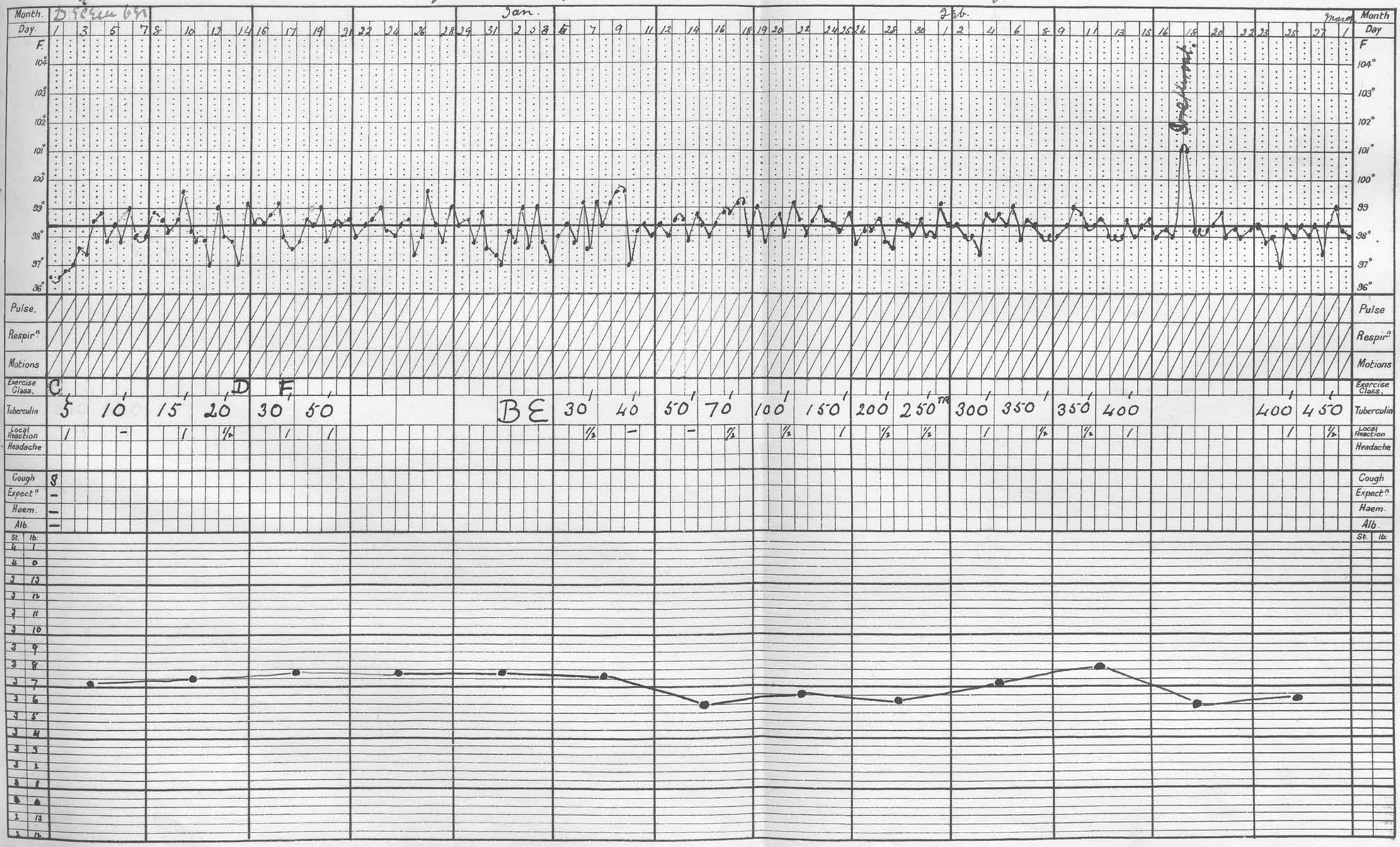


TUBERCULIN CHART. 5.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward S W
No. 4281

Name Ivy Fellows . Age 9 ? . Born May 15th 1900. (Normal Weight) Admitted Jan 20th 1911. Disease

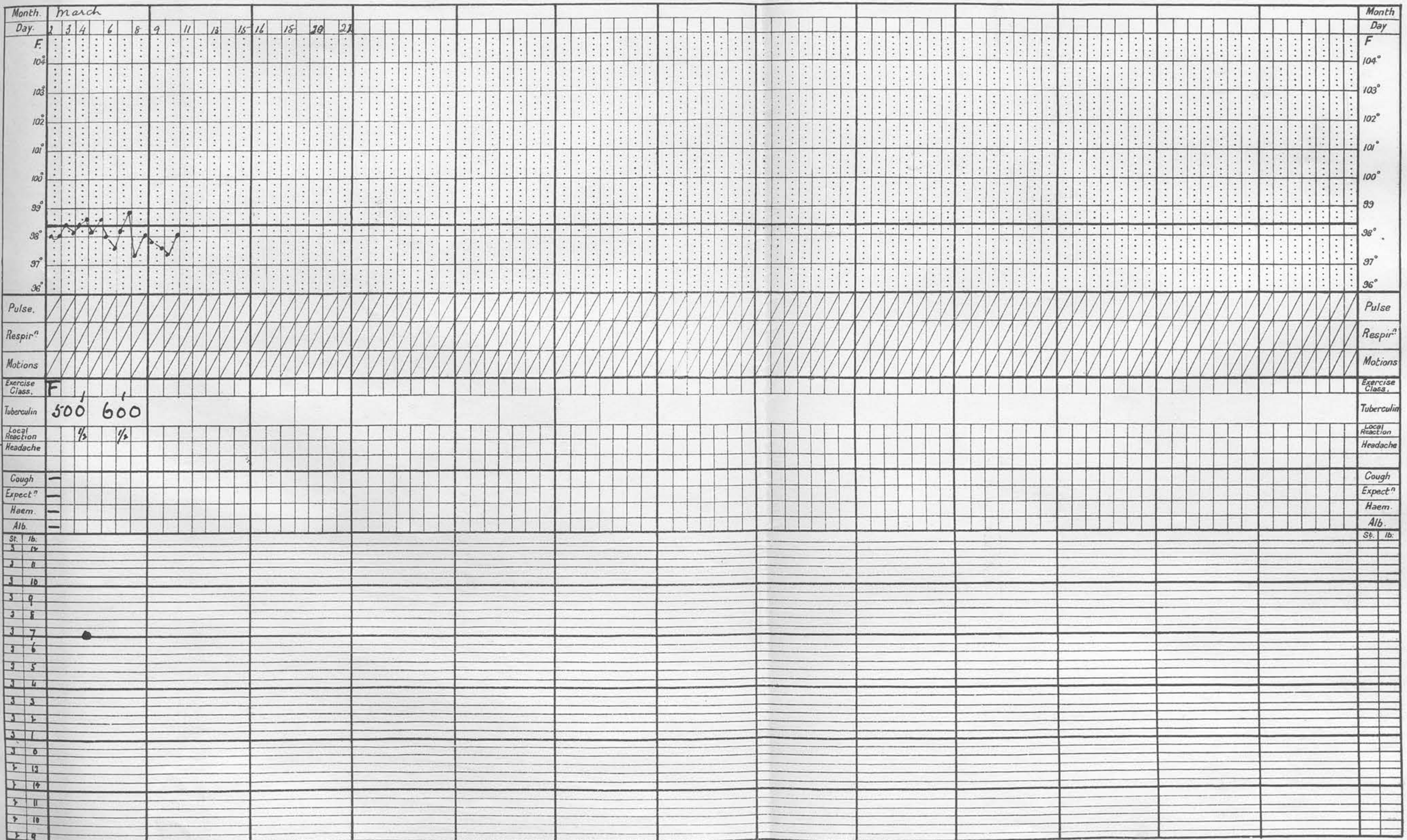


TUBERCULIN CHART.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward S VI
Nº 4281

Name Ivy Fellows . Age 9? . Born May 15th 1900 (Normal Weight) Admitted Jan 20th 1911. Disease



TUBERCULIN CHART. 6.

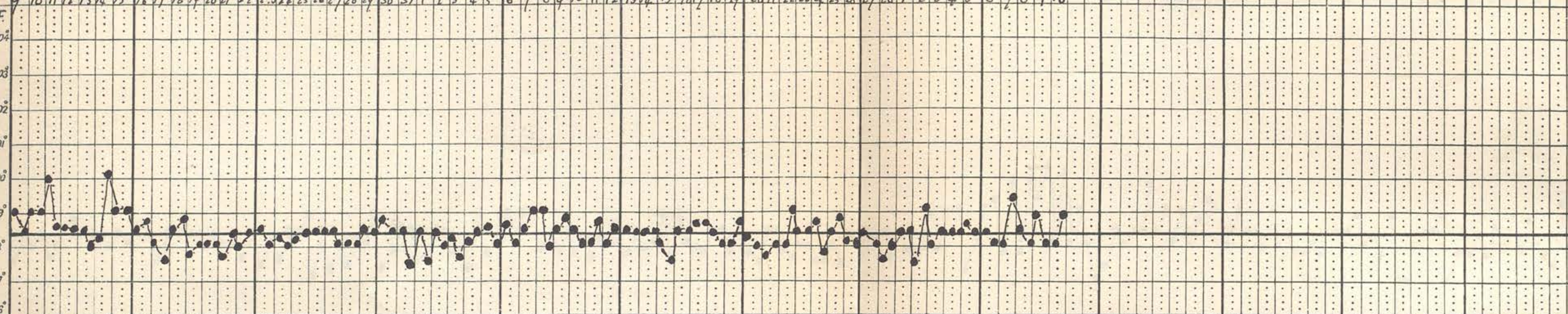
METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward ______
Nº ______

Name Gladys Kent. Age 12. Born ______ (Normal Weight ______) Admitted ______ Disease ______

Month: Jan / Feb / March

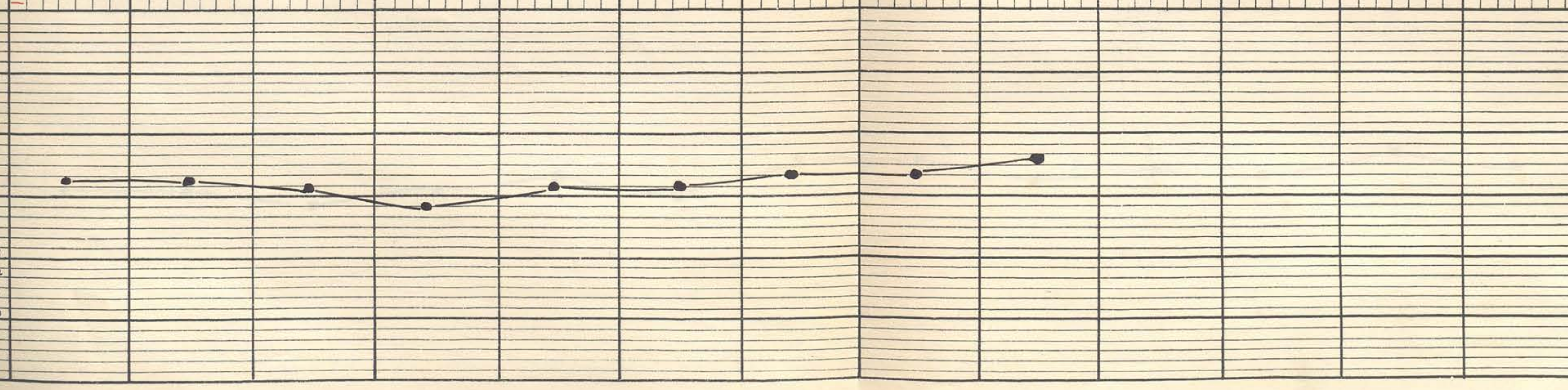
Day: 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 1 2 3 4 5 6 7 8 9 10



Pulse.
Respirⁿ
Motions
Exercise Class. F.

Tuberculin	350	400	400	500	600	750	1000	1250	1750	2250	3000	4000	5000	6000.
Local Reaction	/	½	/	/	½	½	/	/	½	/	/	/	½	/

Headache
Vomiting
Cough —
Expectⁿ —
Haem. —
Alb. —



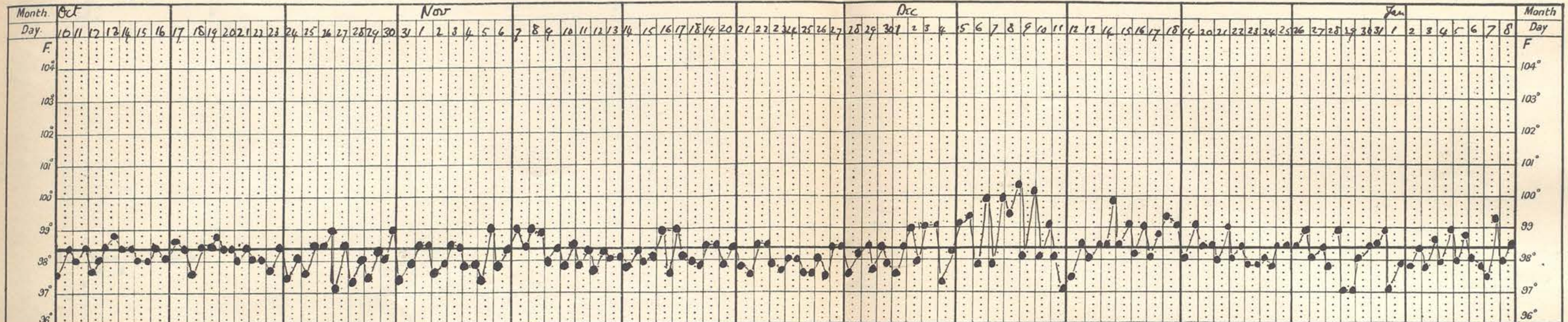
E. ... Units.

TUBERCULIN CHART.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward E 6.
No

Name Gladys. Kent. . Age 12. . Born Nov 17. 1900. (Normal Weight 5 - 7 - 4.) Admitted April 13. 1912. Disease Pulmonary Tuberculosis

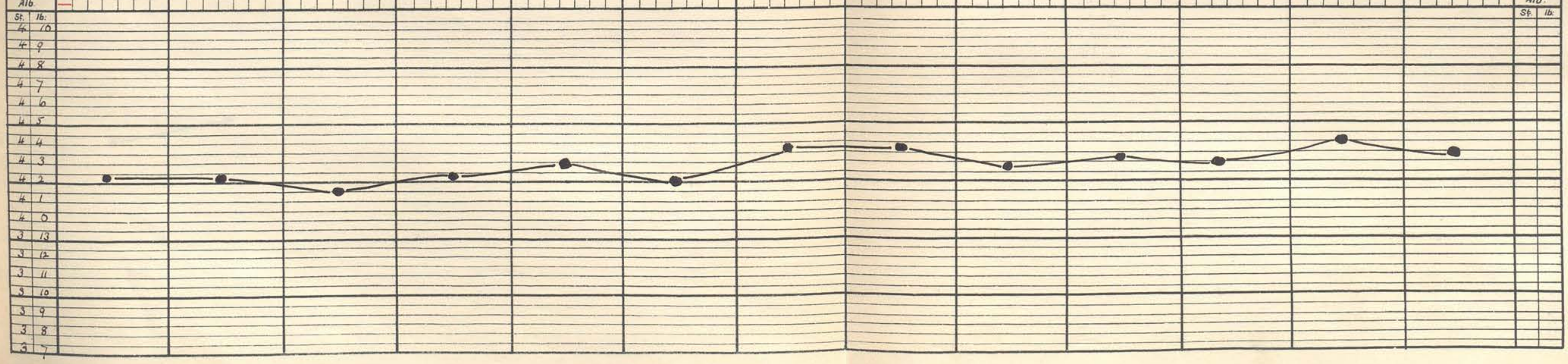


Pulse. Respirⁿ Motions

	Exercise Class.	Tuberculin	Local Reaction
	F		
		2.	1
		2½	1
		4½	3
		5	2
		6	1
		7½	2
		12½	2
		17½	2
		22½	2
		30	½
		40	½
		50	½
		80	½
		120	1
		150	½
		250	½
		300	1
		400	1
		300	½

Headache

Cough S
Expectⁿ —
Haem. —
Alb. —



TUBERCULIN CHART. 7.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward E 5 & 6.
No. 6467.

Name Margaret Ray. Age 10 Born Sept 29-1902 (Normal Weight 4-6-0) Admitted Oct 19-1912. Disease

Month: Mar.
Day: 2 3 4 5 6 7 8 9 10 11 12 13 14 15

F.
104°
103°
102°
101°
100°
99°
98°
97°
96°

Pulse.
Respir.
Motions
Exercise Class. F.
Tuberculin 750 800.
Local Reaction ½ 1
Headache
Cough
Expect.
Haem.
Alb.

St.	lb.
4	4
4	3
4	2
4	1
4	0
3	13
3	12
3	11
3	10
3	9
3	8
3	7
3	6
3	5
3	4
3	3
3	2
3	1
3	0
2	13
2	12
2	11
2	10
2	9
2	8
2	7
2	6
2	5
2	4
2	3
2	2
2	1
2	0

METROPOLITAN ASYLUMS BOARD.

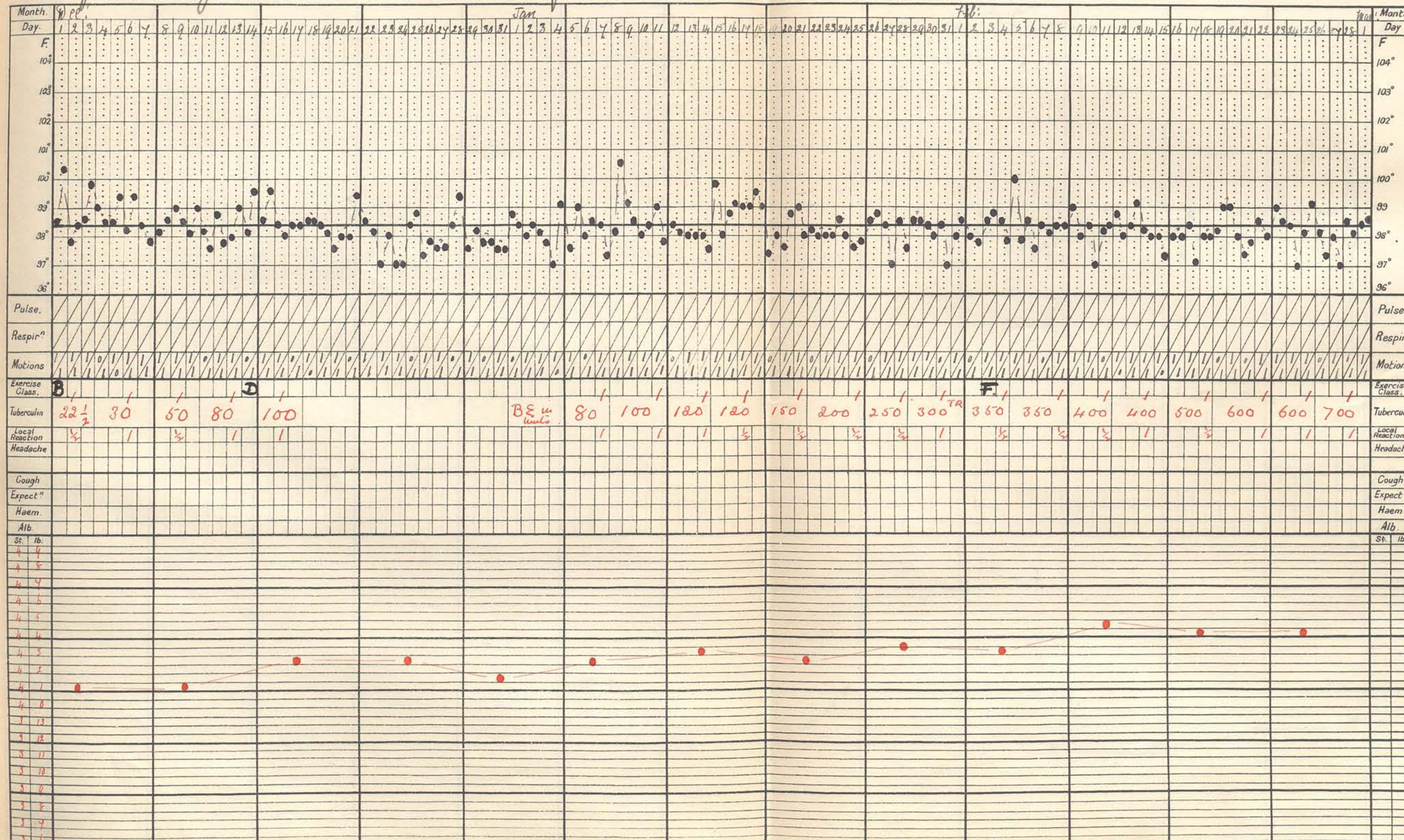
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART.

Ward E 5 & 6

Nº 6467.

Name Margaret Ray . Age 10 Born Sep: 29 - 1902 (Normal Weight 4 - 6 - 0) Admitted Oct: 19 - 1912. Disease



METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART.

Ward E 5.

No.

Name Margaret Ray . Age 10 . Born Sept 29. 1902 (Normal Weight 4 - 6 - 0) Admitted Oct 19. 1912 Disease Pulmonary TB

Month.																														Month
Day.																														Day
F.																														F.
104°																														104°
103°																														103°
102°																														102°
101°																														101°
100°																														100°
99°																														99
98°																														98°
97°																														97°
96°																														96°
Pulse.																														Pulse
Respirn																														Respirn
Motions																														Motions
Exercise Class.																														Exercise Class.
Tuberculin																														Tuberculin
Local Reaction																														Local Reaction
Headache																														Headache
Vomiting																														
Cough																														Cough
Expectn																														Expectn
Haem.																														Haem.
Alb.																														Alb.
St.	lb.																													St. lb.
4	11																													
4	10																													
4	9																													
4	8																													
4	7																													
4	6																													
4	5																													
4	4																													
4	3																													
4	2																													
4	1																													
4	0																													
3	13																													
3	12																													
3	11																													
3	10																													
3	9																													
3	8																													

November

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30

JB.

TR in units.

2	5	10	15	Tuberculin
1	1	1	1	Exercise Class.
—	1	½	—	Local Reaction
		1 —		Headache
		— —		

TUBERCULIN CHART. 8.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

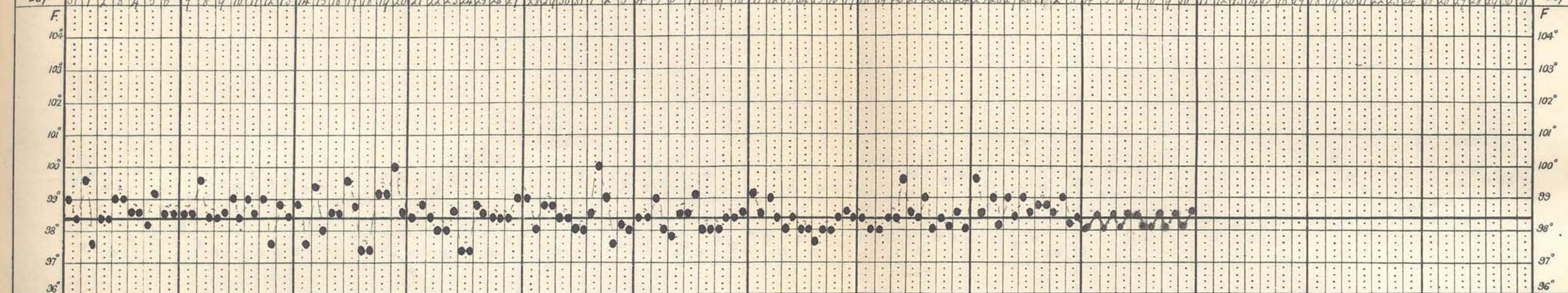
Ward 7 5 9 6

No. 5672.

Name Nellie Ward. Age 5. Born Aug 7: 1907. (Normal Weight 2 · 11 · 8) Admitted Mar: 19 - 1912. Disease

Month: Jan: / Feb: / March

Day: 31, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27, 28, 29, 30, 31



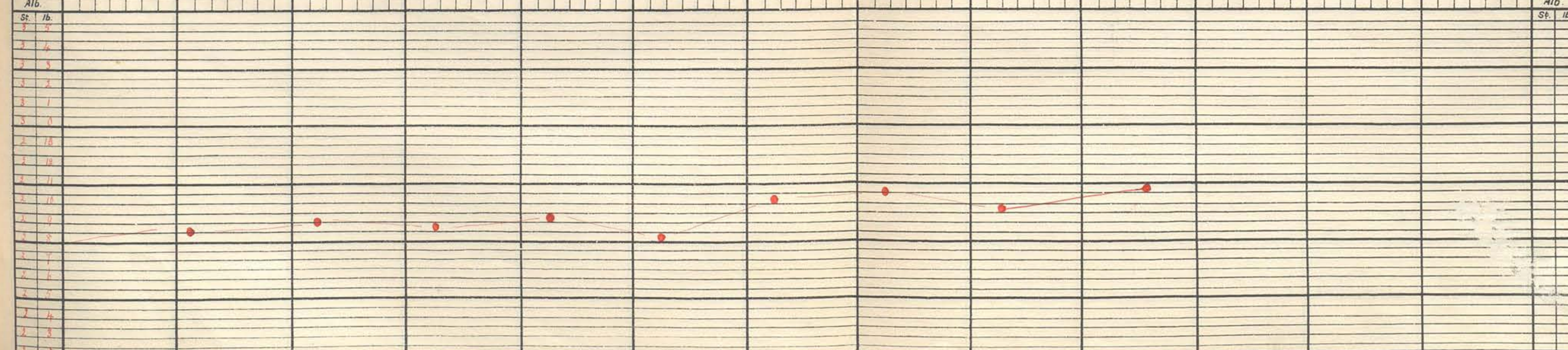
Pulse. / Respir^n / Motions

Exercise Class. E

Tuberculin	50	70	90	120	150	200	250	300	300	400	400	500	600	600	700	750	1000
Local Reaction	2	1	1/4	1/4	1	1/4	1/2	1/2	1/2	1	1/2	1/2	1	1	1	1	1

Headache

Cough / Expect^n / Haem. / Alb.



TUBERCULIN CHART.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward E.5 & b.

No. 56.72

Name Nellie Ward. Age 5 Born Aug. 7. 1907. (Normal Weight 2 - 11 - 8) Admitted March 19. 1912. Disease

Month. Oct. Nov. Dec. Month

Day. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 Day

F 104° 103° 102° 101° 100° 99° 98° 97° 96° F

Pulse

Respir.n

Motions

Exercise Class. D. A+ A B

Tuberculin TR in limbs 2½ 5 7½ 10 15 22½ 30 50 80

Local Reaction ½ ½

Headache

Cough

Expect.n

Haem.

Alb.

St. lb.

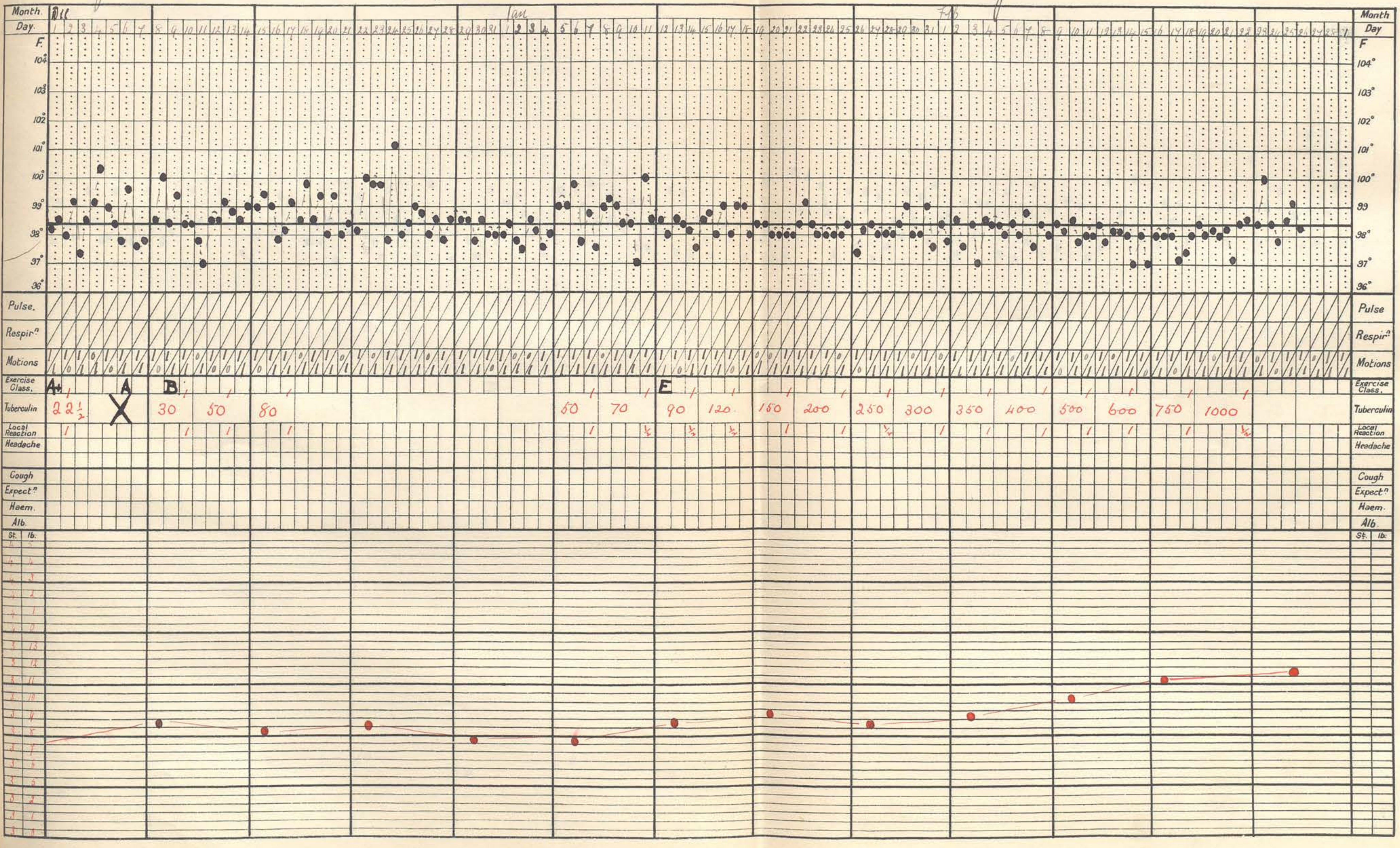
5 2
5 4
5 3
5 2
5 1
5 0
4 13
4 12
4 11
4 10
4 9
4 8
4 7
4 6
4 5
4 4
4 3
4 2

TUBERCULIN CHART. 9.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward E 5 & 6.
Nº 5984.

Name Winifred Senior. Age 7 Born March 23 1905 (Normal Weight) Admitted June 6 1912. Disease

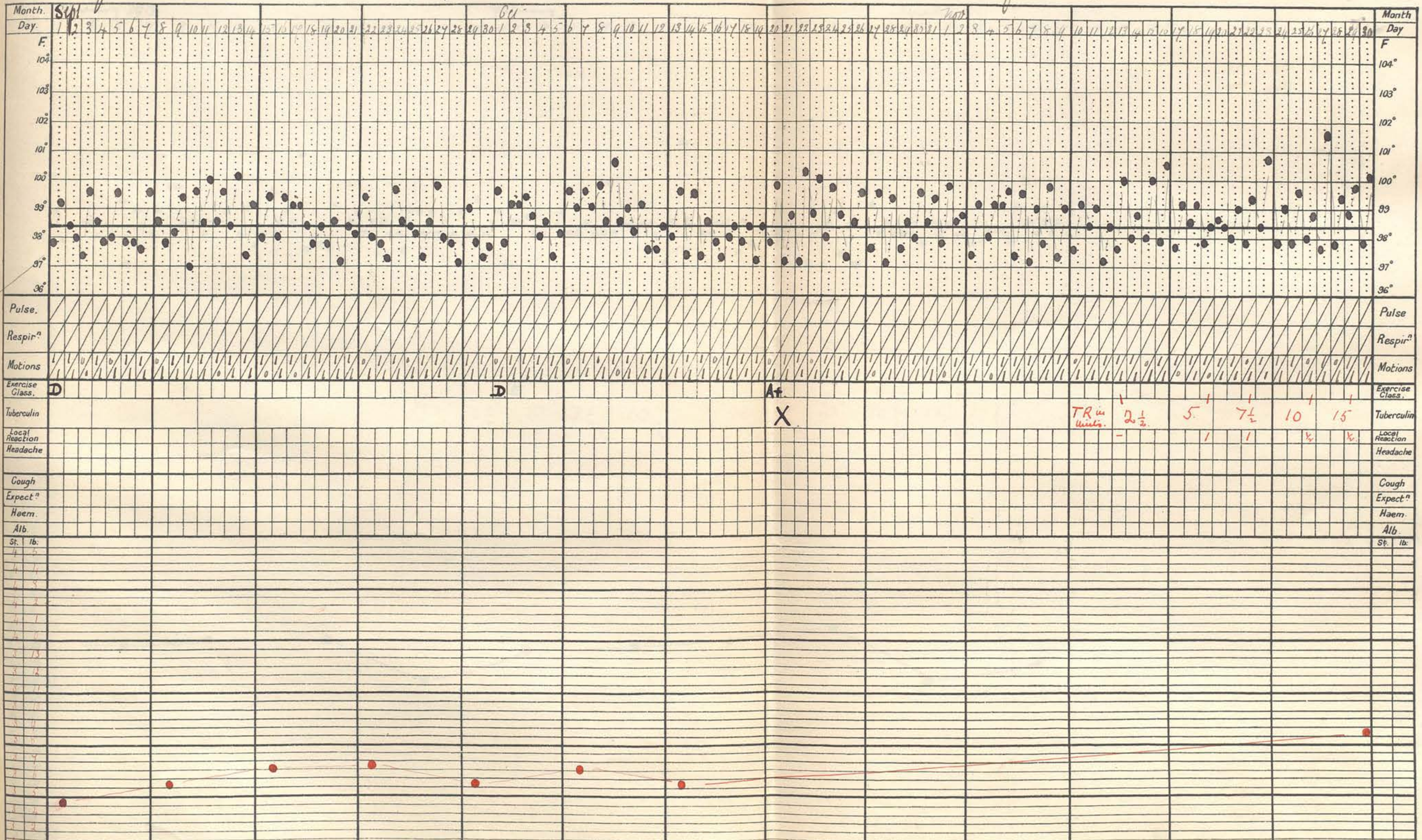


TUBERCULIN CHART.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward 6.5+6
No. 5986.

Name Winifred Senior. Age 7 Born March 23 - 1905. (Normal Weight) Admitted June 6 - 1912. Disease



METROPOLITAN ASYLUMS BOARD.

QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART. 10.

Ward E 6.

No

Name Minnie Bryan . Age 6 . Born May 16: 04 . (Normal Weight) Admitted March 12 - 12. Disease Phthisis (Early)

Month: January 1913 | February | March

Day: 1 3 5 7 9 11 13 15 17 19 21 23 25 27 29 31 1 3 5 7 9 11 13 15 17 19 21 23 25 27 28 1 3 5 7 9 11 13 15 17 19

F: 104° 103° 102° 101° 100° 99° 98° 97° 96°

Pulse.

Respir.n

Motions

Exercise Class. D E

Tuberculin: 2½/1 5./1 7½/1 10/1 10/1 10/1 10/1 12½/1 15./1 17½/1

Local Reaction: 1/½ ½/½ ½/½ 1/1 2/2 3/3 2/2 2/2 3/3

Headache

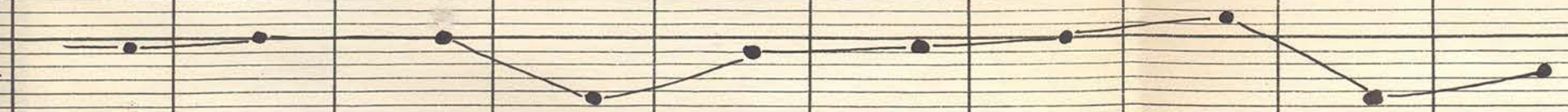
Cough

Expect.n

Haem.

Alb.

St. lb: 4 4 | 4 3 | 4 2 | 4 1 | 4 0 | 3 13 | 3 12 | 3 11 | 3 10 | 3 9 | 3 8 | 3 7 | 3 6 | 3 5 | 3 4 | 3 3 | 3 2



METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

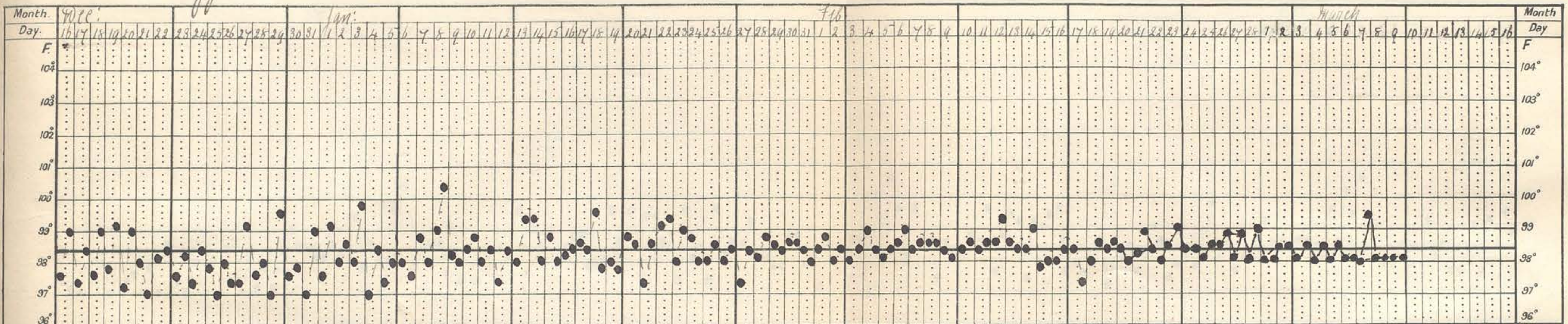
TUBERCULIN CHART. 11.

Ward E.5.96

No. 5496

Name Irene Bugge . Age 13 . Born Feb 2 1899 (Normal Weight) Admitted Feb 7 1912. Disease

Month: Dec. / Jan. / Feb. / March



Pulse.

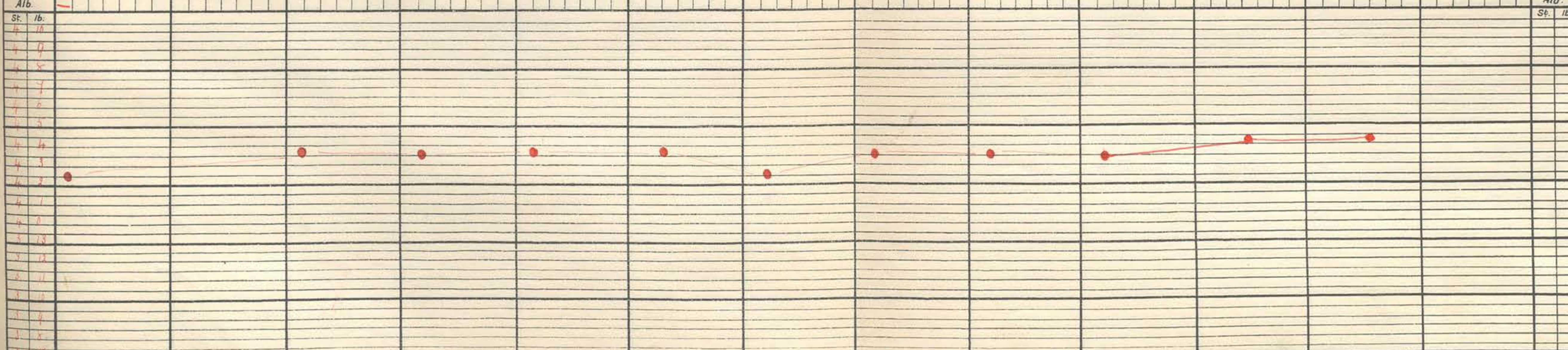
Respirn

Motions

Exercise Class.	D	F	F														
Tuberculin	200	150	200	250	300	350	400	450	550	750	1000	1250	1750	2250	2250	3000	4000
Local Reaction	½	½	½	½	½	1	1	1	½	1	½	1	3	1	1	1	
Headache																	

Cough	—
Expectn	g
Haem.	—
Alb.	—

St. lb.

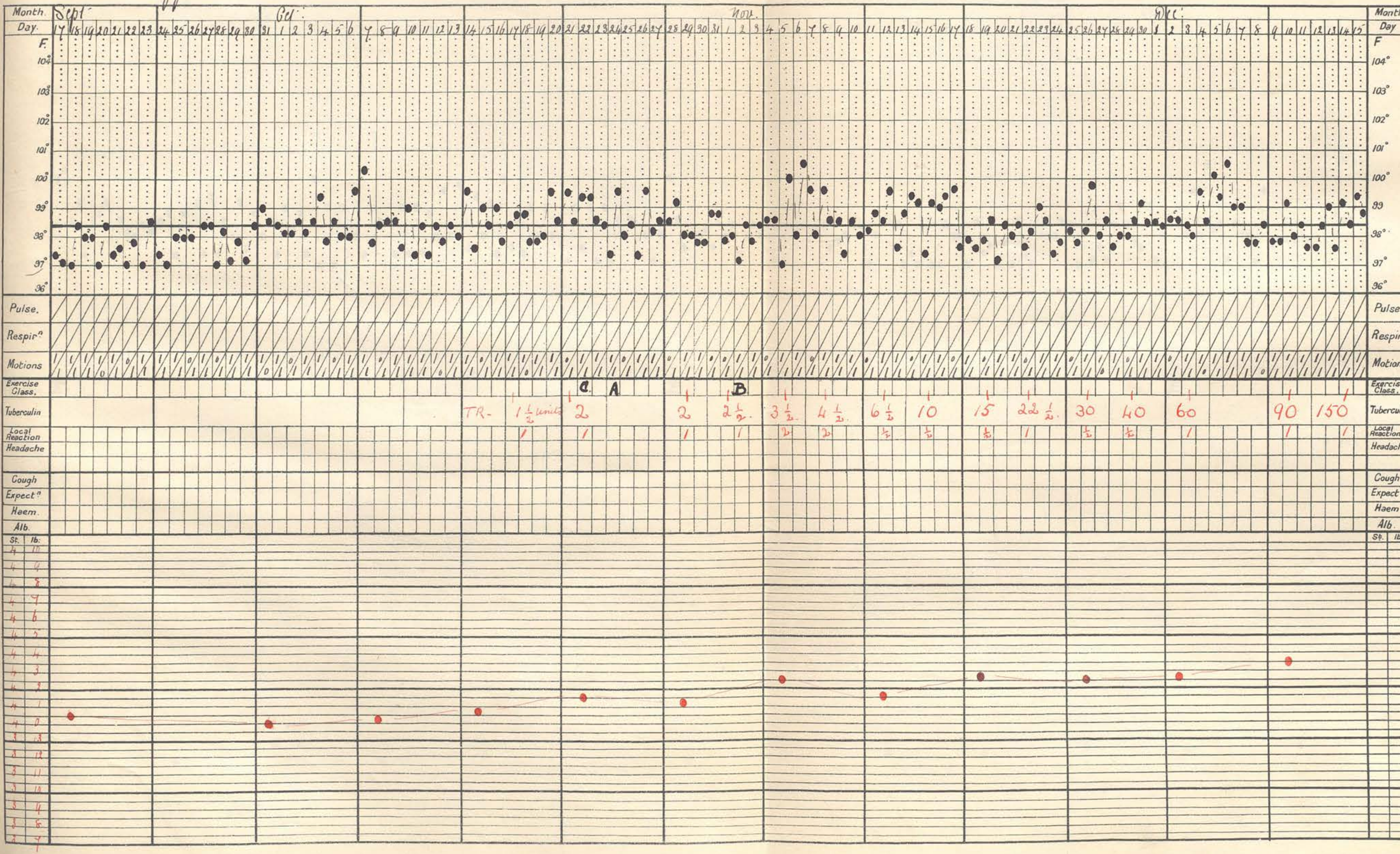


TUBERCULIN CHART.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward 6.5 ? 6
No. 5496

Name Irene Bugge. Age 13 Born Feb. 2. 1899. (Normal Weight) Admitted Feb. 7. 1912. Disease



METROPOLITAN ASYLUMS BOARD.

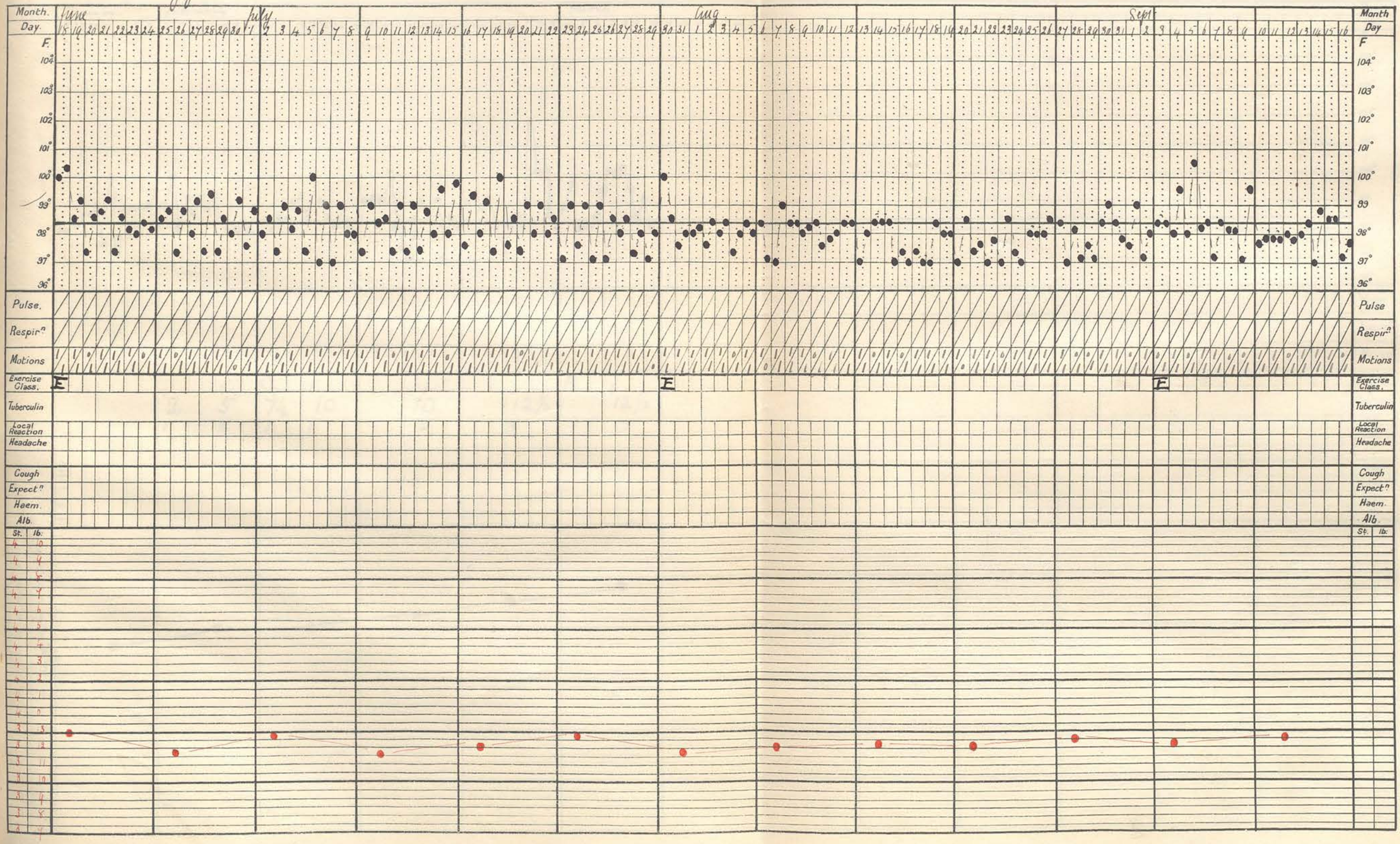
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART.

Ward 6.5.6.

No. 5497

Name Irene Buggs . Age 13 . Born Feb. 2. 1899 . (Normal Weight) Admitted Feb. 7. 1912 . Disease



METROPOLITAN ASYLUMS BOARD.

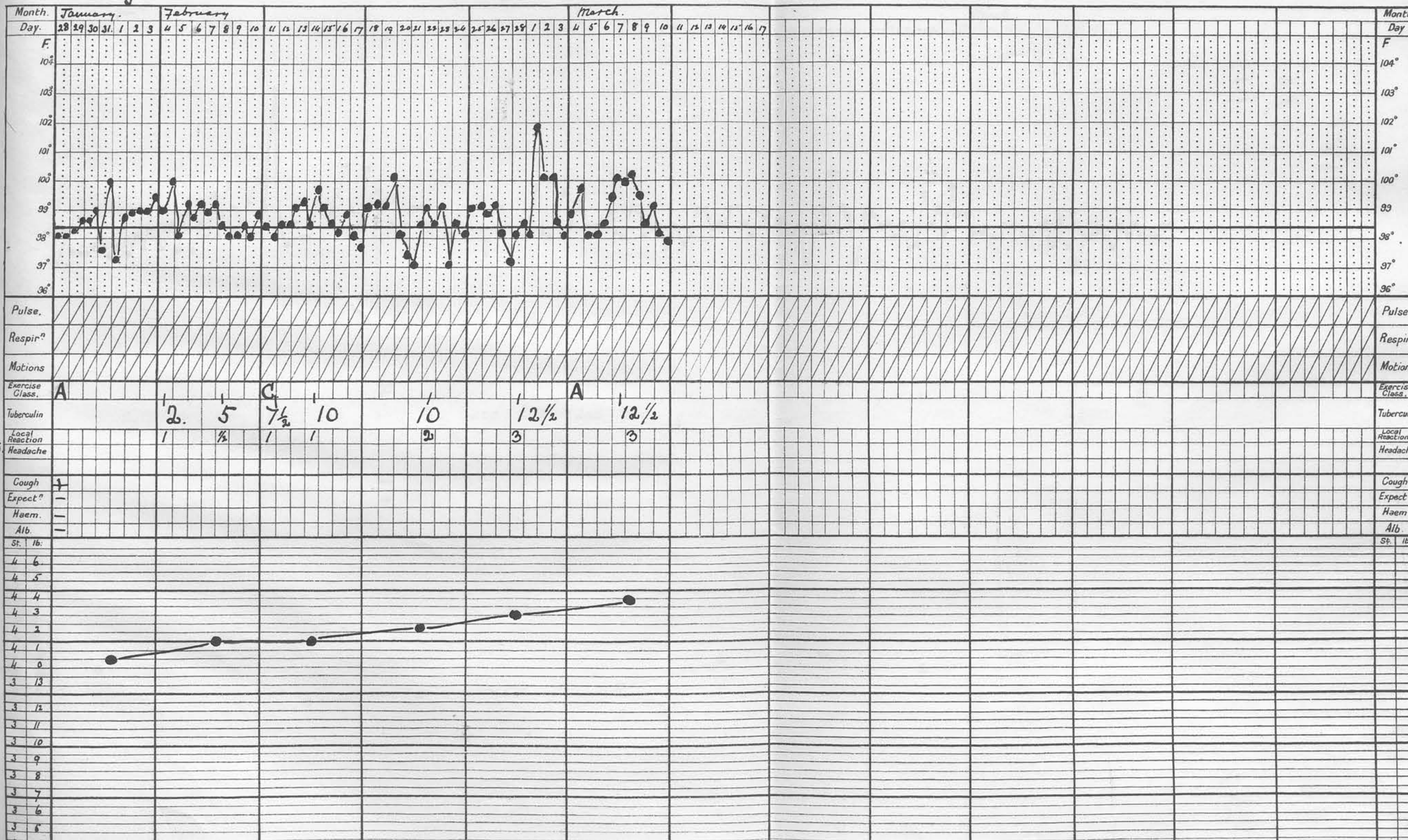
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART. 12.

Ward E 6.

No. ____

Name Betsy Simons. Age 10 Born June 5. 1902. (Normal Weight ____) Admitted Jan. 28 Disease Phthisis.



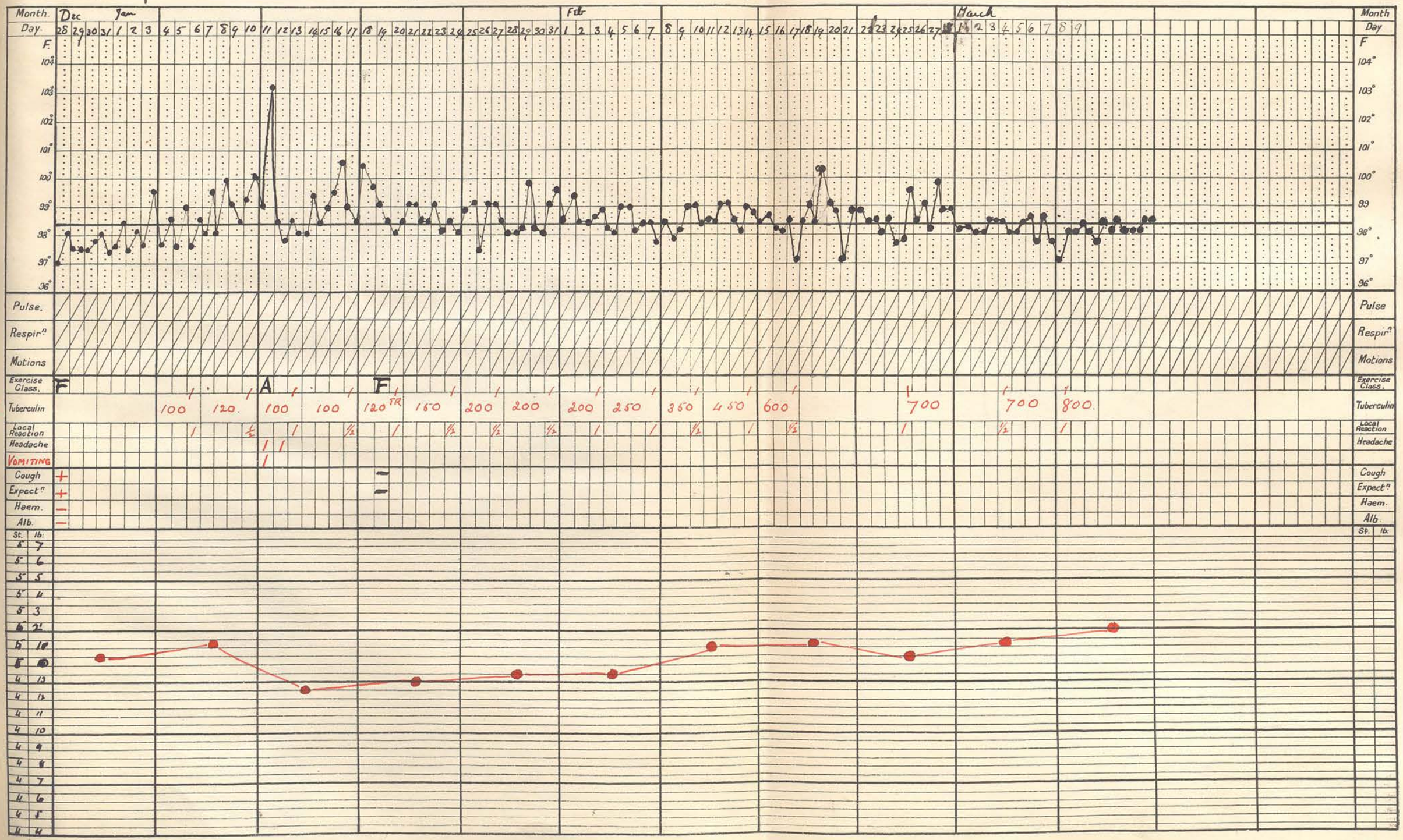
METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART. 13.

Ward _____

Nº _____

Name Mary Studdart. Age _____ Born _____ (Normal Weight _____) Admitted _____ Disease _____

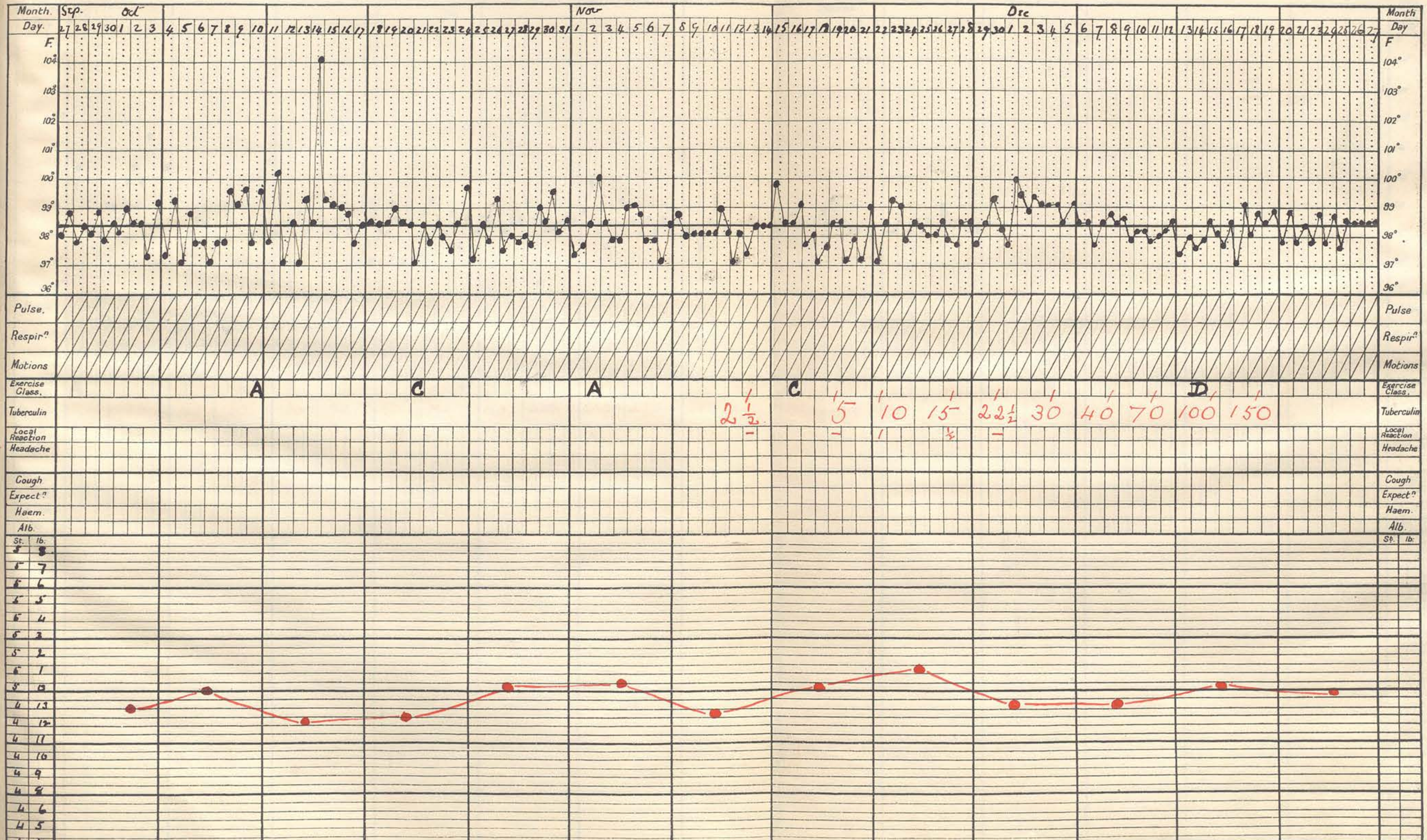


TUBERCULIN CHART.

METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

Ward E5.
No.

Name Mary Studdart. Age 12. Born 29-4-01. (Normal Weight 5-7-4.) Admitted May 19. 1912. Disease Pul: Tuberculosis.



METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

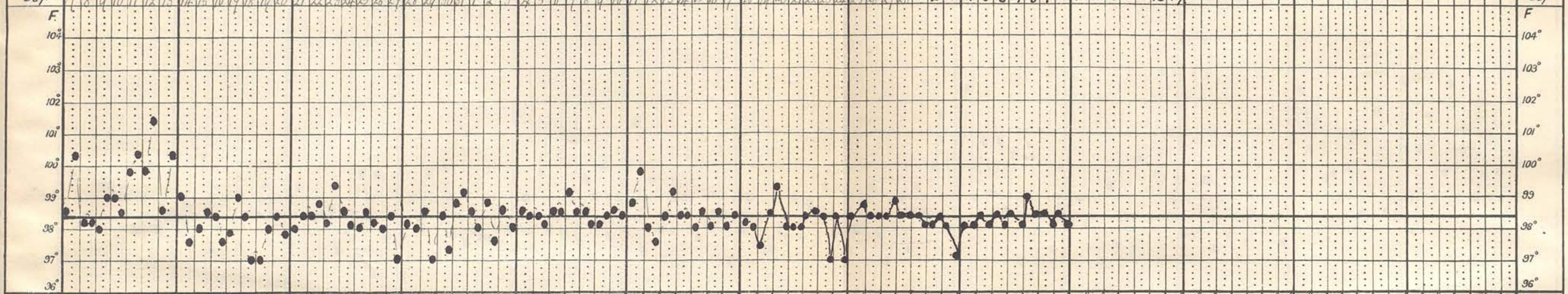
TUBERCULIN CHART. 14.

Ward E 5 + 6
No. 6414

Name Edith Brown . Age 13. Born Aug: 29. 1899. (Normal Weight 6 - 3 - 12) Admitted Oct: 8. 1912. Disease

Month: Jan — Feb — March.

Day: 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17



Pulse: 102/86, 106/92, 94/112, 116/138, 130/108, 100/86, 100/108, 104/98, 94/99, 94/97, 98/93, 82/84, 90/80, 84/88, 84/84, 84/82, 84/88, 90/94, 84

Respir.ⁿ: 24/32, 34/26, 22/28, 30/20, 28/26, 26/28, 28/26, 24/22, 22/26, 22/22, 22/24, 22/26, 24/22, 24/22, 22/24, 24/24, 24/26, 24/24, 24

Motions

Exercise Class	Tuberculin	Local Reaction
A		
	40	1
	40	1
	50	1
	60	1
	70	1
	90	½
	120	¼
	150	1
	200 TR.	1
	200	½
	250	1
	250	1
	250	1
A	300	1
	300	1
	300	1
	350	1

Headache

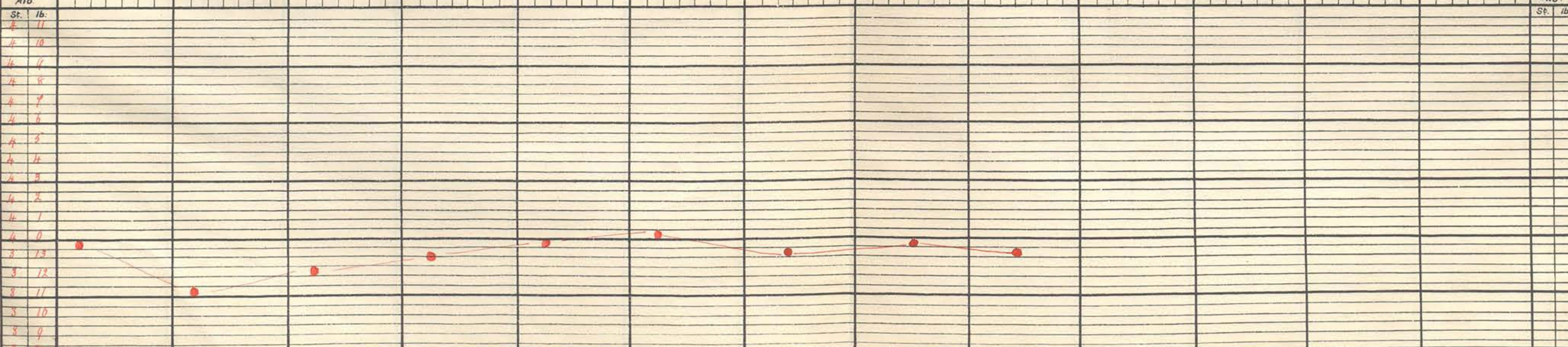
Cough

Expectⁿ

Haem.

Alb.

St. lb.



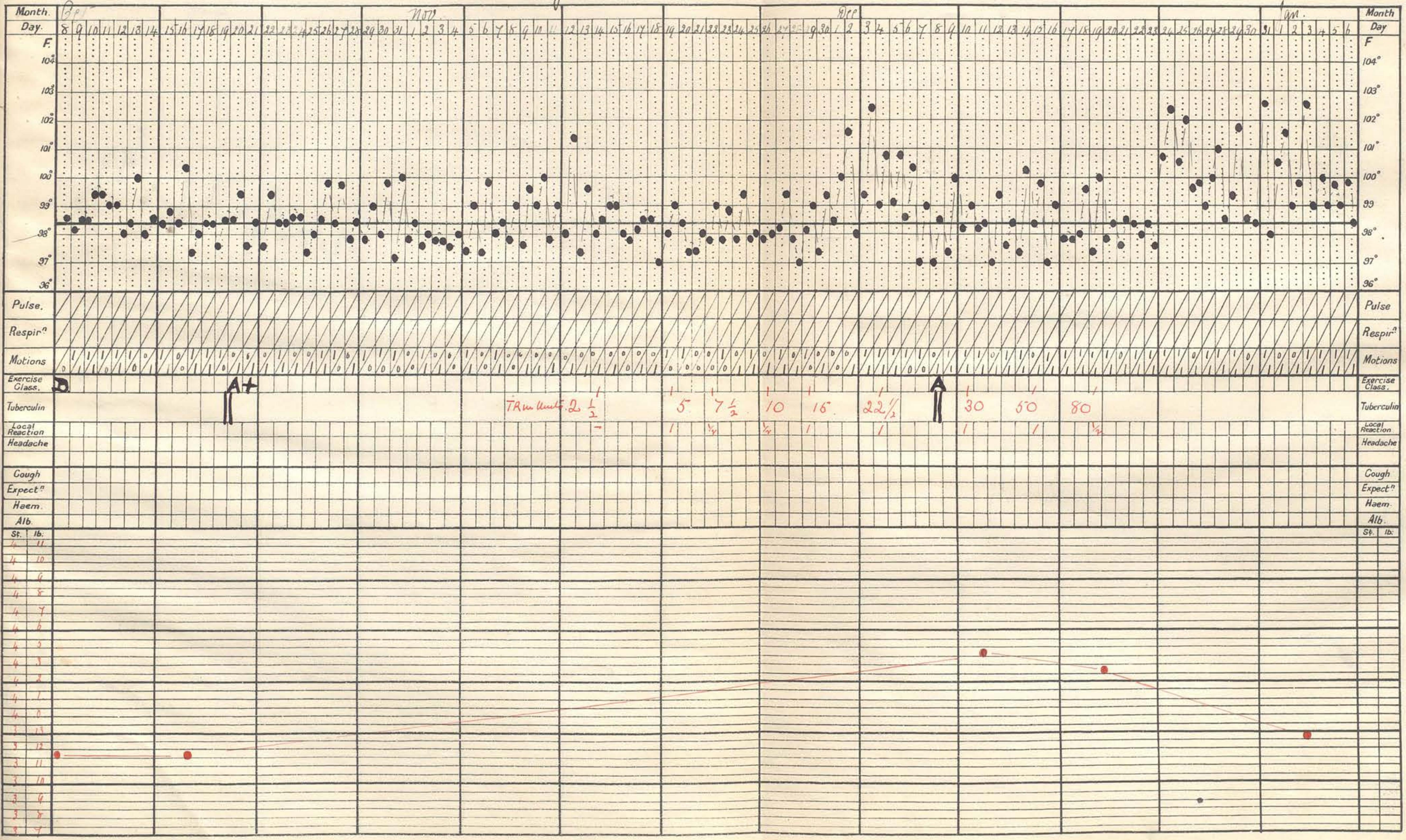
METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART.

Ward E-5 & 6.

No. 6414.

Name Edith Brown. Age 13 Born Aug. 29 - 1899. (Normal Weight 6 - 3 - 12) Admitted Oct. 8 - 1912. Disease



METROPOLITAN ASYLUMS BOARD.
QUEEN MARY'S HOSPITAL FOR CHILDREN.

TUBERCULIN CHART. 15.

Ward E 6.

No.

Name May Springall. Age 7. Born (Normal Weight) Admitted Feb 18th 1913 Disease Phthisis

Month.	Feb.							March.											
Day.	18	19	20	21	22	23	24	25	26	27	28	29	2	3	4	5	6	7	8	9	10	11
Exercise Class.	A+																					

F: 104° 103° 102° 101° 100° 99° 98° 97° 96°

Pulse. Respir.ⁿ Motions Tuberculin Local Reaction Headache Cough Expect.ⁿ Haem. Alb.

St. lb.: 3 8, 3 7, 3 6, 3 5, 3 4, 3 3, 3 2, 3 1, 3 0

Month. Day. F. Pulse. Respir.ⁿ Motions Exercise Class. Tuberculin Local Reaction Headache Cough Expect.ⁿ Haem. Alb. St. lb.