UNIVERSITY OF GLASGOW.

DEGREE OF M.D.

Title of Thesis..... The Control of Scarlet Fever and its Complications, with Special Reference to Serum Treatment.

Full Name ..... MORRISON, WILLIAM WATSON.

Address ..... SOUTHERN HOSPITAL, DARTFORD.

Year of Graduation as M.B. of Glasgow... 1916.

Other registrable qualifications ....
..... D.P.H. (Royal College of Physicians, London).

Medical appointments held ..... Bacteriologist, Cairo District, Captain R.A.M.C., Medical Officer, London County Council Fever Service.

State whether work for Thesis was done in General Practice or in Hospital, Clinic, Laboratory or other Institution, giving place of general practice or name and situation of institution:-

Thesis done in hospital, at Southern Hospital, Dartford.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

The Control of Scarlet Fever and its Complications, with Special Reference to Serum Treatment.

The control of scarlet fever has for many years been one of the most difficult problems which confronts the Public Health administrators.

In a recent address to the Public Health (1.) section of the British Medical Association, Sir George Buchanan stated that six years ago he took part in an elaborate enquiry at the Ministry of Health into the effect of isolation hospitals on the spread of scarlet fever. He states that most of the 30,000 beds in the isolation hospitals throughout England were then in use for that disease, and doubts whether these beds would have been provided unless the authorities believed that their provision would assist in the prevention of scarlet fever. He proceeds to state that this belief has not been justified. The provision of isolation accomodation has not checked the spread of scarlet fever, nor has it been responsible for any diminution in the severity or in the mortality of the disease. The only benefit so far obtained from their use has been the facilities for treatment of the individual case not otherwise available. He adds that the functions of these institutions should be to supply

the most effective treatment, to save life in the individual case, and by doing everything possible during hospital treatment and applying a follow-up system, to prevent the invalidity due to nephritis, middle ear disease and other complications. They afford an invaluable means of clinical research on a large scale which cannot elsewhere be obtained, such as the effect of sera in the treatment of infectious diseases.

During the past thirty years there has been a steady decline in the mortality of scarlet fever, but the incidence of the disease is still very high. Every seven years an epidemic wave sweeps over the community and places a great strain on the hospital accomodation of public health authorities. Even although the majority of the cases have been for the past few years of a mild type, there is no guarantee that the infective agent may not become more virulent, the case mortality again rise and scarlet fever demand extensive hospital isolation and the use of lines of treatment in accordance with more recent research. While, however, case mortality is an index of the severity over a large series of cases it takes no account of the severity of the individual case.

(2) Case mortalities and admissions of scarlet fever 1900-1929.

Year	Admissions	Case mortality	London death rate per 1000
1900	10,343	3	•16
1905	16,958	3•3	•12
1910	8,782	2•3	•28
1915	15,197	2	•25
1920	20,821	1•1	•17
1925	10,508	1•1	•19
1928	13,065	•6	•09

To combat any infectious diseases by scientific means, a knowledge of the life history of the infective agent is of supreme importance. True it is, that a measure of immunity to smallpox is obtained by vaccination without the definite infective agent being known. Typhus fever, the causative organism of which is also unknown, has been controlled, more or less, as a result of the application of the knowledge that the infective agent is carried by pediculi and suitable measures taken to destroy these parasites. In the case of scarlet fever, however, until within recent years the causal organism has been a matter of conjecture

and consequently the mode of attack against infection has been, in many cases, purely empirical.

Scarlet fever is one of the most variable of the exanthemata. It can assume one of three types:- a mild, a septic or a toxic type; the two latter forms very often proving fatal within a few days. The mild type can give rise to some of the most disabling complications such as nephritis, otitis or carditis. Also, and of great importance to the community, a mild case, which is not diagnosed and isolated, may give rise to a septic or toxic case.

It will be the purpose of this thesis

(1). To sketch the history of the disease and to mention some of the sources of infection with indications how to abolish these.

(2). To describe some of the errors in the diagnosis of the disease from my own experience and the means of avoiding these.

(3). To compare the progress and complications of cases treated with serum, with the progress and complications of cases treated without serum; and the effect of serum on the treatment of some of these complications.

(4). To summarise the effect of the use of serum in the prophylaxis, diagnosis and treatment of scarlet fever.

When the disease originated is unknown. An epidemic was described by Thucyides about the year 439 B.C. which occurred in Athens and bears a distinct resemblance, according to Malfatti, to an outbreak of scarlet fever, but this according to Rolleston,(3) was probably typhus. It is doubtful if Hippocrates recognised the disease. John Phillip Ingrassias 1510-1580 was the first medical man who described scarlet fever in the medical literature. He was professor of medicine at Naples, and in his book entitled "De Tumoribus praeter Naturam" published in 1553 he speaks of a disease popularly known as rossalia which consisted of "numerous spots, large and small, fiery and red, of universal distribution so that the whole body seems on fire" and he goes on to differentiate it from measles. Sydenham in this country in 1657 first described the disease, though, curiously enough he did not describe the angina but he was the first to call it scarlet fever, and differentiated it from measles. An epidemic in Saxony at the end of the 18th. century carried off 40,000 inhabitants. Bretonneau in the year 1824 experienced an epidemic at Tours which was attended with such a high mortality that he came to regard the disease as no less deadly than plague, typhus or cholera. According to Graves the epidemic of scarlet

fever in Dublin 1801-1804 was extremely fatal, death sometimes taking place on the second day. The epidemics which followed were mild until in the years 1831-1834 outbreaks of scarlet fever of the malignant type caused more deaths than cholera or typhus.

Dr. Cock (4) reported an outbreak of scarlet fever which occurred in the island of St. Bartholomew in the West Indies in 1829 and 1830, caused by the immigration of children from the United States. It is interesting to note that he first described the appearance of the tongue, and also that after the arrival of these children from the island of St. Bartholomew on the adjoining island of Montserrat an outbreak of scarlet fever occurred there. He also appreciated the fact that cases of malignant scarlet fever may arise from infection by a mild case.

The association of streptococci with scarlet fever and its complications has been recognised for a long time. Loeffler in 1884 isolated streptococci from throat smears and suggested that this was the etiologic factor. In 1885 an outbreak of the disease was traced to the milk supplied by a farm in Hendon, and Dr. Klein, who made the bacteriological examinations, succeeded in isolating a streptococcus from the milk and an identical streptococcus from the patients. Mervyn Gordon has more recently made an

investigation into the bacteriology of scarlet fever and found that there were two groups of streptococci present, one identical with the streptococcus pyogenes and a second streptococcus which had distinct cultural characters, such as the clotting of milk in that medium and its conglomeration in broth media. This he called the streptococcus scarlatinae or conglomerata. Dohle also reported the presence of a spirillum in the blood.

Dr. Mallory (5) of Boston City Hospital described a body which was situated between the cells of the rête mucosum. This body has an amoeboid shape and some of the bodies described have a stellate appearance reminding one of some of the stages in the life history of the malarial parasite. Other intracellular bodies have been described by other authorities. Amato did several inoculation experiments on apes which resulted in a modified form of the disease developing in these animals. Notwithstanding these observations and researches the association of the streptococci with scarlet fever as a primary entity has for long been a matter of doubt. In 1905 Jochman published a careful article weighing the evidence for the claim of the streptococcus to be the primary cause of scarlet fever, and concludes that "Streptococci are indeed the most common, and indeed the most dangerous agents of the secondary infections and complications of the disease, but are not the true causative organism of scarlet fever".

This dictum was almost universally credited when I commenced work in the infectious diseases hospitals in 1920, but within recent years, the last eight years to be precise, the truth of it has been tested again, and the recent works of Drs. George and Gladys Dick in 1923-1924 has practically proved that a streptococcus is the causal organism. (6) In 1923 Dr. and Mrs. Dick succeeded in producing scarlet fever in human volunteers by swabbing the naso-pharynx with haemolytic streptococci obtained from the pus in a finger of a nurse who was suffering from scarlet fever. This streptococcus has the property of causing haemolysis in red blood corpuscles. They then cultivated this organism, obtained a soluble toxic filtrate, and by injecting ·1 or ·2 of a 1 in 1,000 dilution determined the susceptibility or non-susceptibility to scarlet fever. This reaction is now known as the Dick reaction. I have tested several hundred cases of scarlet fever in the convalescent stage of the disease and have found that the majority of such cases have been Dick negative in their reaction. A few cases however, remain Dick positive. These cases have probably not produced sufficient immunity or may have been infected by a different strain of streptococci as it has been

demonstrated that there are four serological groups of scarlet fever streptococci which may produce varying amounts of immunity. I have also used the test in a ward of convalescent children in which scarlet fever has occurred and have been able to segregate the susceptible children. The reaction is analogous to that of the Schick test for diphtheria; it differs from it in a few details chiefly in that the reaction is, in a susceptible individual, at its maximum within six hours, whereas the reaction to the Schick test is often not at its maximum until the thirty-sixth hour. A control test with heated toxin is done on the opposite arm. (7)

The work of Dr. W. Mair on the aetiology of scarlet fever is interesting, although he does not conclude that the disease is due to the streptococcus haemolyticus. He injected a series of monkeys with the streptococcus haemolyticus and found that by the intradermal injection of serum obtained from these monkeys he was able to blanch the rash in scarlet fever. This reaction, known as the Schultz Charlton reaction is corroborative evidence of the fact that the disease is due to streptococci. We have made use of this test at this hospital in many cases of doubtful erythemata

and in a later part of this paper I shall refer to the use of this test in diagnosis. One of his penultimate remarks however, is worthy of quotation with regard to the Schultz Charlton reaction in scarlet fever, "The conclusion that sera which are capable of blanching the rash of scarlet fever can be produced in monkeys by the injection of haemolytic streptococci is in agreement with the work of Dochez and the Dicks in America, and of MacConkey and O'Brien in this country. These workers have prepared sera in horses by the injection of cultures or of culture filtrates of haemolytic streptococci. These sera possess the property of blanching the rash and are now being tried for their therapeutic effect in scarlet fever".

The serum treatment of scarlet fever has come into general use for the treatment of the toxaemia of scarlet fever, and I shall show from the records of cases which have come under my care that the serum quickly reduces the temperature, causes the rash to fade more rapidly, and if given at the onset of the illness, renders the patient less liable to complications, especially to the septic complications such as adenitis and arthritis.

Specific agglutinins are found in the serum of convalescents, especially if this is concentrated.

If these claims or even a majority of them are accepted, there appears to be no doubt that scarlet fever is due to a specific haemolytic streptococcus.

The infection of scarlet fever is spread by the distribution of articles which have been in contact with an infected person and have become contaminated by the infectious discharges from the nose, ear, or skin. The commonest articles are clothes, letters and books. Domestic animals may also spread infection. Articles of food may become contaminated, and of these the most culpable is milk. Missed and unrecognised cases, especially when they are suffering from a mucous discharge from the nose or throat are the most dangerous carriers of infection.

One epidemic reported (6) in the annual report of the State of Public Health illustrates the spread of infection by missed cases handling, and so infecting milk. The Medical Officer of Health of the Lymm Urban District informed the Ministry in his weekly return of infectious diseases that about 40 cases of acute tonsillar and pharyngeal inflammation had occurred in his district, and on the 17th. September 1923 Dr.P.G.Stock, a Medical Officer of the Ministry, made inquiry into the circumstances of this outbreak. Prior

to the outbreak the district had been apparently free from infectious disease. When Dr. Stock arrived in the district no complete list of the patients was available, but as soon as a preliminary one could be prepared it was found that some 45 cases had occurred between 4th. August and 15th. September. As many of these patients had only suffered from a mild attack and did not present typical symptoms, the illness had been regarded by the Medical Officer of Health as acute tonsillar and pharyngeal inflammation. On 12th. September however, the County Medical Officer of Health of Cheshire visited the district, and in a letter to the Ministry dated 13th. September he expressed the opinion that the patients he had seen were all suffering from scarlet fever. Further enquiries showed that all the persons affected had obtained milk from a certain dealer who purchased the whole of his supply from a farm on the outskirts of Lymm. When this farm was visited four cases of scarlet fever in different stages of the disease were found, and, as the farmer declined to comply with the instructions of the Medical Officer of Health steps were taken to obtain the necessary magistrate's order for stopping the milk supply. Warning notices were also issued to the public, notifying the prevalence of scarlet fever and recommending that all milk should be

boiled before comsumption.

One of the chief causes of the spread of the disease is the presence of mild ambulant cases. How often in practice is one told that a child has only a "stomach rash" or has had "a slight sore throat which cleared up in a few days". There can be no doubt that these unrecognised cases are the greatest carriers of infection. The matter might deserve less consideration, but considering that the mildest case may produce a virulent and perhaps a fatal form in another individual, we must exert every effort to prevent the spread of the disease by the early and accurate diagnosis of such mild cases.

It is my experience that school closure has a modifying influence on the incidence of the disease among children, and that when the children return to school the number of scarlet fever cases rapidly increase.

This is presumptive evidence that the aggregation of susceptible individuals is an important factor in the spread of the disease. The school medical officer can here help in the prevention of the disease by the early diagnosis and isolation of mild cases of scarlet fever, by testing the susceptibility

of contacts to scarlet fever by means of the Dick test, and by early treatment and isolation if necessary of all cases who show any signs of naso-pharyngeal inflammation.

Infectious diseases hospitals are often criticised adversly, and blamed on account of return cases which are a source of renewed infection. On examining the statistics of this hospital, which for the past year has treated both acute and convalescent cases, I find that out of 6,265 discharges there were 142 return cases, an infectivity rate of 2·26%. The interval that elapsed between the discharge of the infecting case and the return case was one week in fifty-one cases, two weeks in fifty-seven cases and the remainder during the third and fourth weeks. In the majority of these infecting cases a history is given that the patient after discharge developed a discharge from the nose or from the ear. This discharge may have contained the streptococcus haemolyticus and have infected the return case. Great care and frequent examination of the naso-pharynx is necessary prior to the discharge of a patient after scarlet fever, and the slightest indication of nasal catarrh should be an indication for further isolation. On the other hand many

return cases are undoubtedly due to ineffective or inefficient home disinfection of articles which have been used by the patient prior to hospitalisation, or to some new source of infection.

The old recognised method of isolating a case until all desquamation had entirely ceased has now, in most cases, been abandoned. (9). Dr. Moore of Huddersfield held that desquamation was not due to the specific organism of scarlet fever. Desquamation occurs in other diseases, such as measles and erysipelas and also in some forms of eczema, and is, in all probability, due to hyperaemia of the skin. Theoretically, the exfoliated epidermis if contaminated by discharges, either nasal or aural, and inhaled by a susceptible individual could cause scarlet fever, and consequently the greatest care and thoroughness should be employed in the ablutions of patients prior to discharge from an infectious diseases hospital. In practice however, I have found that no return case has been ascribed to desquamation alone and that the infective agent is usually present in a nasal or aural discharge.

A typical case of scarlet fever presents few difficulties to the diagnostician. The initial

vomiting, and headache, the rash with pyrexia, the faucial injection and exudate on the tonsils, and the state of the tongue all combine to give a clinical picture which can hardly be mistaken; but such a combination of symptoms and signs is often absent. Frequently the rash is very transient, disappearing within a few hours. The pyrexia may be slight or the temperature may be normal, and the faucial condition may only consist of a slight injection. In several outbreaks of scarlet fever in my diphtheria wards I have from time to time noticed that several cases of tonsillitis have preceded and accompanied outbreaks of scarlet fever. In a recent paper (10). on acute tonsillitis and some of its sequelae Drs. Glover and Griffith state that "Epidemic tonsillitis without a rash is generally attributed to strains belonging to a heterogenous group of haemolytic streptococci and that these strains cause scarlatina when their toxigenic powers are sufficiently high to overcome the antitoxic immunity of the person attacked".

This dictum from the bacteriological standpoint, explains in a measure, the synchronous outbreaks of tonsillitis and scarlet fever which I have experienced from time to time.

In the report for 1928, the Metropolitan Asylums Board give an interesting table in which are summarised the errors in diagnosis for various infectious diseases and I shall quote these for scarlet fever. Out of 13,065 cases admitted certified as scarlet fever there were 323 cases whose diagnosis had to be revised, giving an error rate of 2·47%, and it is of interest to note that

7 cases of scarlet fever were wrongly diagnosed as cases of cerebro spinal meningitis,

6 cases of scarlet fever were wrongly diagnosed as cases of chicken pox,

31 cases of scarlet fever were wrongly diagnosed as cases of diphtheria,

3 cases of scarlet fever were wrongly diagnosed as cases of enteric,

194 cases of scarlet fever were wrongly diagnosed as cases of measles,

3 cases of scarlet fever were wrongly diagnosed as cases of puerperal fever,

5 cases of scarlet fever were wrongly diagnosed as cases of whooping cough,

so that there are many pitfalls in the diagnosis of scarlet fever.

Many of the prodromal rashes of measles or chicken pox might be mistaken for the rash of scarlet fever. There are also many drug and serum rashes which simulate the rash, so that the whole clinical history must be gone into carefully. At this hospital I have found the Schultz Charlton reaction of great value in arriving at a diagnosis in doubtful cases and I shall quote a few of the cases in which it has proved itself of value.

(1).D.M. a nurse at this hospital, was admitted with a sore throat on the 11th. May 1929. Her temperature was normal, but on the 12th. May she developed an erythema which was punctate in character in the upper extremities but was papular on the back. There was very little faucial injection. The Schultz Charlton reaction was done and the rash around the injection was blanched. This confirmed the diagnosis of scarlet fever.

(2).T.E.M. aged 4 years, was admitted with scarlet fever on the 17th. May 1930. She had a temperature of 100° and there was a very faint erythema and slight faucial injection. She desquamated on the 20th. May. An erythema of doubtful origin was observed on the 6th. June, and the Schultz Charlton reaction was done but proved negative, so that we concluded that the erythema was not due to a relapse of scarlet fever.

(3).K.M. aged 5 years, was admitted on the 5th. July 1930 with a history of headache and diarrhoea. She had a blotchy rash on her chest on admission, but her temperature was normal and her fauces were clean. She was admitted to a scarlet fever ward. On the 17th. July she developed a punctate rash on t chest and there was faucial injection. A Schultz

Charlton reaction was done and was positive and a diagnosis of scarlet fever arrived at. During her convalescence she had another attack of enteritis with a blotchy erythema which was of similar character to the rash on admission.

(4).F.W.B. aged 4 years, was admitted on the 13th. June 1930 with a diagnosis of scarlet fever. The rash was described as "A somewhat discrete punctate erythema on the trunk, upper arms and thighs". Her temperature was normal the following day and there was no faucial injection. A Schultz Charlton reaction was done but there was no blanching of the erythema. The following day the rash faded, The subsequent history did not indicate that the case had been one of scarlet fever and agreed with the result of the Schultz Charlton reaction.

(5).A.M. aged 18, a ward maid, was admitted with a sore throat and a faint rash on the trunk. She had just joined the staff and had recently been vaccinated. Her temperature was 99° and there was very little faucial injection. The diagnosis was a matter of doubt. The Schultz Charlton reaction was done and was positive, and the diagnosis of scarlet fever was made. Within a few days of the disappearance of the rash she desquamated and her further progress was that of a mild case of scarlet fever.

I consider that the Schultz Charlton reaction is an aid in the diagnosis of mild cases with indefinite rashes, and in cases of erythema not due to the streptococcus haemolyticus, but to other causes such as the administration of drugs or serum, which might simulate scarlatina.

I have recently examined the records of cases of scarlet fever of this hospital and in order to evaluate the use of serum in the treatment of scarlet fever and its value in the prevention of the complications of scarlatina, I have made a comparison between the results of the serum treatment in 312 cases which were given serum, and 4990 cases which had not been so treated. I shall draw attention to the effect of serum on some of the salient features of the disease, such as the effect in length of pyrexia, period of rash, and on the occurrence of complications.

The method of administration of the serum is a matter of importance. At this hospital, the routine method has been by the intramuscular route, and I have had no alarming serum reactions by this method. The amount of serum given was usually 20c.c. to 40c.c. of Burroughs Wellcome's antiscarlatinal serum and the group of muscles selected was usually the muscles of the thigh by the fascia lata.

(11.)

Dr. Banks of Leicester City Hospital has used the intravenous method of administration of the serum and reports excellent results, but the serum reactions have been in many cases of an alarming character; rigors have occurred within one hour of administration,

the temperature has reached 107°, the colour has become poor and the pulse rapid. He reports one fatal case. I have heard of a similar occurrence where the serum was used for septicaemia in one case in private practice.

Scarlet fever treatment with serum - 312 cases.

Average period of pyrexia		2·96 days
Duration of rash		2·66 "
29 cases developed otitis	percentage	9·29
23 " " adenitis	"	7·37
6 " " arthritis	"	1·92
11 " " albuminuria	"	3·52
4 " " nephritis	"	1·28
17 " " rhinitis	"	5·44
1 " " relapse	"	·32
1 " " endocarditis	"	·32
4 " " tonsillitis	"	1·28

Scarlet fever treatment without serum - 4,990 cases.

Average period of pyrexia		4·88 days
Duration of rash		3·88 "
534 cases developed otitis	percentage	10·7
498 " " adenitis	"	9·97
105 " " arthritis	"	2·1
290 " " albuminuria	"	5·81
98 " " nephritis	"	1·96
252 " " rhinitis	"	5·04
135 " " relapse	"	2·7
19 " " endocarditis	"	·38
37 " " tonsillitis	"	·74

The pyrexial period of a case of moderate severity of scarlet fever is from three to five days. As a rule, after the administration of serum, the

pyrexia lasts twenty four hours and the temperature subsides often from 104° to 98° with dramatic celerity. The average pyrexial period of 312 cases treated with serum was 2·96 days, whereas that of the control cases was 4·88 days, and in my opinion the control cases were usually of a milder type. The pulse rate was also less, the toxaemia was lessened and the faucial condition was greatly ameliorated. The rash in scarlatina is a fair index of the severity of the attack, and this as a rule faded earlier in the serum cases than in the control cases, the average duration of the rash in the serum cases being 2·66 days, that in the control being 3·88 days.

The incidence of complications in scarlet fever is one of the most important features of the disease. True it is, that patients die early from a septicaemia due to an overwhelming infection of a virulent type, but in these days when the infection is of a milder type, a useful index of the use of a special form of treatment would be the effect of that treatment in the prevention of complications.

Goodall,(2) in a recent clinical statistical study of scarlet fever, gives a table of the incidence of complications in scarlet fever which is of interest

as it is a study of the disease prior to the use of serum during the period 1895-1914. He states that out of 21,290 patients, 8,776 had complications and 12,514 had no complications. These complications he tabulates thus:-

Complication.	Percentage of total cases of scarlet fever.
Otitis	11•3%
Albuminuria	6%
Adenitis	6%
Adenitis suppurative	1•7%
Nephritis	4•2%
Rheumatism	1•9%
Tonsillitis	1•2%
Stomatitis	•8%
Relapse	•8%
Endocarditis	•2%

In my series of cases which was much smaller than Goodall's I have found that otitis in both groups of cases was the most common complication. In the cases treated with serum the number was smaller than in that of the control series. In my series of cases treated with serum there were 9•29% cases of otitis while in the control series the percentage of cases was 10•7.

This is a natural sequel to the observation made in a previous paragraph that serum diminishes the severity and ameliorates the faucial involvement. Otitis is caused by the passage of infection through

the eustachian tube from the posterior pharynx to the middle ear.

Rolleston, (13) quoting Yearsley, states that 34% of acquired deafness follows scarlet fever, so that we can confidently anticipate a great diminution in this disability by the early use of serum in scarlet fever.

Otorrhoea is one of the chief causes of prolonged residence in fever hospitals. In some cases a patient is detained for 100 days on account of otorrhoea so that the prevention of this complication would be of great economic advantage to the community.

Otitis is more common in winter than in summer and is more prevalent in young children than in adults.

The early symptoms of otitis are pain over the affected ear and a rise of temperature. This pain in infants may simulate meningeal pain. An early examination of the tympanic membrane is indicated, and if the drumhead is found to be injected or bulging an early paracentesis should be done. This relieves tension and permits free drainage of the inflammatory products, a cardinal law in surgical technique. This treatment, followed by the use of antiseptic hygroscopic drops and the thorough drying out of the meatus will in many cases prevent extension of the process of

inflammation and lead to healing of the membrane and restoration of function.

Frequently in the course of otitis, oedema and redness develop over the mastoid, and in a few cases in front of the mastoid, owing to the arrangement of the temporal fascia. In such cases it has been the practice at this hospital to do a Wilde's incision. This consists of an incision down to the bone and a stripping of the periosteum. This simple operative procedure has in many cases been successful, the oedema has subsided, the temperature has come down to normal, the patient making a complete recovery and the dangers of a radical mastoid operation have been averted. If the temperature does not subside, or if there is delay in the healing of the incision from the presence of necrosed bone, the radical operation should be advised.

(1).E.B.S. aged 7 years, was admitted on the 5th. Dec. 1930 with a moderately severe attack of scarlet fever. On the 12th. Dec. she developed a temperature of 102° and right otalgia. The cervical glands were enlarged. The tympanic membrane was found to be red, congested and bulging. A paracentesis was done under a general anaesthetic on Dec. 22nd. and the temperature fell to normal. On the 24th. Jan. 1931 she developed oedema over the left mastoid and in front of the auricle with otorrhoea. A Wilde's incision was done on the 2nd. Feb. The temperature fell to normal and the oedema subsided. The wound over the mastoid healed and she was discharged with both drums healed and hearing normal.

(2).R.P.B. aged 4 years, was admitted on the 31st. Dec. 1930 with a fairly severe attack of scarlet fever. The rash and temperature lasted for four days. On the 16th. Jan. he developed right otalgia and a temperature of 99°. The following morning he developed purulent otorrhoea which was treated with spirit ear drops, and on the 25th. Jan. there developed redness and oedema over the right mastoid with protrusion of the auricle. A Wilde's incision was done with good results. The oedema subsided and in time the incision healed and he was discharged on the 25th. March with a healed drum and normal hearing.

(3).F.A.S. aged 8 years, was admitted with scarlet fever on the 16th. June 1930 and she had a history of otorrhoea. This ear discharge continued, and on the 18th. July she developed oedema and pain over the mastoid process, with protrusion of the auricle. Her temperature was 100° and pulse 100. A Wilde's incision was done, pus was found under the periosteum and necrosed bone. The otologist who did a radical mastoid operation found osteomyelitis of the temporal bone, a new infection superimposed on an old one, and the condition cleared up and she was discharged on the 1st. November without otorrhoea.

I consider that antistreptococcal serum, if given at the onset of the disease, by ameliorating the faucial and pharyngeal condition, is of great help in preventing the onset of otitis. The Wilde's incision is also of value in the early treatment of mastoiditis.

The early treatment of acute otitis consists in a paracentesis of the tympanic membrane on the first sign of bulging of the drumhead, the provision of free drainage and the treatment of the discharge by antiseptics containing alcohol.

The treatment, however, of chronic otorrhoea following scarlet fever is still a matter of difficulty. One author, Paton,(14) (1928) states that "The removal of adenoids results in an increased incidence of otorrhoea", and Bradley(15) states that "Tonsillectomy has not protected against otitis media".

Many otologists believe in the radical mastoid operation for all cases of chronic ear discharge. There is no doubt however, that a thorough examination of all septic foci present in the nose, throat and ear should be made, and any gross source of infection removed.

A practical point in the prognosis and treatment of chronic otorrhoea is the position of the perforation of the tympanic membrane. If the perforation is centrally placed and does not extend to the periphery of the drum, the discharge which is due to a catarrhal inflammation of the middle ear frequently (in 95% of cases according to Ascherson) clears up by zinc ionisation.(16) The marginal perforations however, usually indicate bone suppuration and in all probability will require a radical mastoid operation.

McCarrison in a lecture on "Some Surgical Aspects of Faulty Nutrition"(17) states that experimentally in rats one of the most common consequences of faulty food, deficient in Vitamin A, is pus in the middle ear.

The causes giving rise to middle ear disease interact with one another; they are squalid poverty - with its accompaniment of malnutrition - neglected catarrh, tonsils and adenoids, mouth breathing, and the after effects of measles, scarlet fever and diphtheria. He goes on to say that "My rats do not suffer from the three last; but my experience in these animals leads me to believe that the insufficient supply of Vitamin A bearing foods may be responsible for middle ear disease in children to an extent much greater than is generally believed".

The general health of the patient should be improved by rest, tonics, fresh air and sunlight, and an adequate supply of Vitamin A bearing foods.

I have found at this hospital most of the cases of chronic otorrhoea clear up under this régime without operative measures.

Nephritis and late albuminuria are very serious complications of scarlet fever. Although the prognosis of scarlatinal nephritis is in most cases good, still a considerable number of deaths fron nephritis and uraemia occur as a complication of scarlet fever. Many cases which we have diagnosed as chronic nephritis may be late sequelae of the acute nephritis which began with an attack of scarlet fever.

Some writers state that the streptococci attack the glomeruli while the toxins affect the tubules. The effect of serum on this serious complication of scarlet fever should be beneficial.

In my series of cases 1·28 per cent of the cases which had serum developed nephritis, while 3·52 per cent developed albuminuria, whereas in the control cases 1·96 per cent developed nephritis and 5·81 per cent developed albuminuria.

Dr. Goodall, in his statistics for this complication during the period 1895-1914 states that 4·2 per cent developed nephritis. Ker (18) gives a much higher figure, his percentage being 11·02. Dr. Robb (19) of Belfast stated that in one hundred cases treated with serum he had no cases of nephritis.

Nephritis is more common in children than in adults and is also more frequent during the winter months. The uncertainty of the prognosis is a matter of great importance, many of the cases having an unexpected fatal termination. Frequently an apparently mild case of scarlet fever develops nephritis.

The majority of the cases of nephritis occur during the third week of the illness and it is frequently associated with enlargement of the cervical

glands. Von Pirquet regarded this occurrence as an anaphylactic phenomenon in the third week of the disease during which the toxins damaged the renal tissue.

The use of alkalis as a preventative measure to the onset of nephritis was first described by M. Fischer (20) in 1911. Dr. Osman of Guy's Hospital has advocated, and Dr. Carter (21) of the Park Hospital has tested its value in the prevention of nephritis in cases of scarlet fever. He found that by the use of alkalis from the onset of the fever only ·6% developed nephritis, while in a control series of 316 cases 5·5% developed this complication.

The other preventative measures to this complication are avoidance of chill to the back which undoubtedly lowers the resistance of the body and allows the toxins to act on the kidney.

Nephritis is often ushered in by fever, vomiting, and oedema of the face. On examination of the urine there is suppression, blood and albumen are found, and epithelial casts are present. There is always a diminution of the chlorides and the reaction is usually acid. These symptoms usually subside within three weeks and the condition clears up entirely. On the other hand, the case may not respond to treatment, suppression of urine may persist

and the patient may develop uraemia with serious results.

In seeking a treatment for any complication we naturally look to the cause for an indication of the therapeutic course to adopt. In several cases where scarlet fever antitoxin was given after the onset of nephritis, no beneficial results occurred and the usual lines of treatment such as complete rest in bed, a bland diet with plenty of fruit juices, attention to the bowels by means of saline aperients, and the use of radiant heat have been adopted.

If uraemic symptoms develop I have found that venesection has been of great value. A high rectal enema followed by a rectal saline which contains 6% glucose often prevents the onset of ketosis and supplies some of the basal caloric requirements of the body. Lumbar puncture has been found to be useful in several cases, the removal of 20c.c. or 30c.c. of fluid in many cases ameliorating the condition, the fluid in most cases being under considerable pressure.

(1).T.Y. aged 7 years, was admitted on 7th. Feb. with a moderately severe attack of scarlet fever. His temperature was 100°. The symptoms lasted three days and thereafter the temperature became normal. After three weeks apyrexia he suddenly had a rigor and vomited. His temperature was found to be 102? On the 1st. March blood and albumen were present in the urine and there was suppression. The following day oedema of the face was noticed and his temperature remained high for two days. A light salt free diet was ordered, hot packs

and saline aperients were given and the condition cleared up within three weeks.

(2).M.D. aged 9 years, was admitted with a mild attack of scarlet fever on the 23rd. Oct. 1930. His temperature on admission was normal. On the 2nd. Nov. he developed earache and on the following day ear discharge developed. On the 6th. Nov. he had headache, sickness and vomiting and the temperature suddenly rose to 102°. He had oedema of the face and on examination of the urine, blood and albumen were found to be present. The ordinary treatment for nephritis was adopted but he did not react to this, and on the 28th. Nov. he developed twitchings of the facial muscles, became cyanosed and was semi-comatose. Venesection was done and rectal salines with glucose given but he remained semi-comatose. On the 30th. Nov. lumbar puncture was done, 25c.c. cerebro-spinal fluid removed, and the following day he was able to recognise the nurse. From this date he gradually improved, the urine became normal in quantity, and the blood and albumen disappeared. He made a complete recovery and was discharged.

From these cases I conclude that venesection is of great use in uraemia, that lumbar puncture relieves the cerebral congestion, consequently the headache is diminished, and that high rectal enemata followed by rectal saline and glucose combine to eliminate some of the poisons, to dilute the poisons of uraemia and supply some of the necessary calories to the body.

Lymphadenitis is a frequent complication of scarlet fever. It may occur at two periods in the disease - at the onset, and after three weeks illness. At the onset

of the disease it is caused by the absorption of septic material from the fauces or from the buccal mucous membrane, and the degree of this involvement depends on the severity of the lesions in the throat or mouth and consequently is more severe in septic cases. A late adenitis occurs about the third week and, although important in itself, is also important from the fact that it frequently is a precursor of nephritis. In my series of cases early adenitis was less frequent among the cases which had had antitoxin than in those which had not had it. The inflammatory process occasionally goes on to suppuration, but more often it subsides and the glands become normal, unless there is a specific or tubercular infection present.

The presence of enlarged glands three weeks after a pyrexial illness with sore throat should make one suspect that the case has been one of scarlet fever which has been missed. Desquamation should be looked for and the urine should be examined for albumen and casts. If there are signs that the gland is suppurating there should be no undue haste to incise it until fluctuation is present, since early incision very often protracts the recovery of the gland to normal.

Rheumatism is one of the less common complications of scarlet fever. Goodall, in his statistical survey gives the figure of 1·9 as the percentage of cases from 1895-1914 which developed this complication. In my series of cases, 1·2% developed arthritis among those treated with serum, while 2·1% had arthritis among the controls. Scarlatinal rheumatism affects the smaller joints, chiefly the wrists and the interphalangeal joints, and in my experience, suppuration is rare except in very septic cases.

(1). M.K. aged 8 years, was admitted with a moderate attack of scarlet fever on the 5th. June 1930. Her temperature was then 101°, but it was normal on the 7th. June. On the 8th. June however, her temperature rose to 100°, and on the 9th. it was 104°. She had severe pains in the wrists and in the interphalangeal joints. The treatment was Mist. Sodii Salicly. and 30c.c. antistreptococcal serum, and by the 14th. June her temperature was normal and the pain had gone from her joints.

(2). O.J. aged 6 years, was admitted on the 17th. Nov. 1930 with a mild attack of scarlet fever. Her temperature was 101° on admission, but was normal the next day. She made good progress until the 16th. Dec. when she developed pains in the left hip and her temperature rose to 101°. On the 17th. Dec. she had pain in the right thigh. Lin. Methyl Salicyl. was ordered and by the 23rd. Dec. the pain had gone and her temperature was normal. On the 9th. Jan. 1931 she developed pain in her left shoulder. Mist. Sodii Salicyl. and Lin. Methyl Salicyl. were ordered and by the 13th. Jan. the pains had quite gone.

Scarlatinal rheumatism almost always ends favourably within a few days after which the swelling of the joints subsides. Any or all joints may become affected by pain and swelling. Many cases of torticollis occurring during scarlet fever are caused by an infection of the joints of the cervical vertibrae.

Despite the absence of heart involvement the cases of scarlatinal rheumatism bear a strong resemblance to the type of acute rheumatism seen in this country.

Suppuration in joints may be due to an ordinary synovitis going on to suppuration or from embolism during a septicaemia.

Scarlet fever may and does cause endocarditis and pericarditis. The cardiac complications rarely lead to the sudden and dramatic fatalities which occur from time to time in cases of diphtheria from involvement of the neuro muscular mechanism.

Cases however, of sudden death do occur. I recollect one case of a child who was convalescent from scarlet fever. She had had a liberal tea when she suddenly became restless and pallid, then became cyanosed and died before medical aid arrived.

The early toxaemia in scarlet fever frequently leads to a myocarditis with dilatation of the cardiac chambers and results in a relative incompetence of the

valves, so that a mitral systolic murmur is commonly heard in the early stages of the illness. This usually disappears however, when the pyrexial period is over.

By means of antistreptococcal serum we are able to diminish the toxaemia and so lessen the frequency of this myocarditis. I find from the records that ·32% of the cases treated with serum developed a systolic mitral murmur, whereas ·38% of the control cases had this murmur of relative incompetence.

Although we cannot attribute every mitral systolic murmur to the presence of endocarditis, we have to conclude, if the murmur persists for some time unchanged, or if a presystolic murmur develops that endocarditis is present as a result of scarlet fever and that permanent damage has been done to the valves.

Pericarditis occurs from time to time, and in cases of large pericardial effusion with cardiac distress the advisability of puncture and removal of some of the pericardial fluid must be considered.

(1). D.F. aged 9 years, was admitted on the 19th. April 1930 with scarlet fever. She developed pain in the wrists and thighs on the 24th. April. On the 27th. April she developed pain over the praecordium and a definite friction rub was heard over an area of 2 inches below the left nipple, due to pericarditis. On the 29th. April the praecordial pain was less but the dullness had increased. These signs and symptoms gradually diminished with ordinary treatment and she was discharged fit on the 2nd. June.

(2). G.S. aged 7 years, was admitted with a moderately severe attack of scarlet fever on the 26th. Nov. 1930. She was given 20c.c. antistreptococcal serum on the 27th. Nov. She developed cervical adenitis on the 29th. Nov. and her temperature remained elevated. On the 3rd. Dec. she complained of pain in the right wrist, in the knees and in the ankles. There was swelling of the affected joints and pain on movement, and on the 6th. Dec. a systolic mitral murmur was present. The pulse was rapid - 108 per minute - but of good tension. With rest and treatment of Mist. Sodii Salicyl. the murmur was less marked by the 28th. Dec. and her progress continued favourably. On the 13th. Jan. the murmur disappeared and the remainder of her convalescence was uneventful.

(3). W.O. aged 8 years, was admitted on the 22nd. Jan. 1930 with scarlet fever. On the 24th. Jan. he developed a systolic apical murmur. His temperature remained at 102° and on the 30th. Jan. he was given 20c.c. antistreptococcal serum. His temperature subsided but the murmur persisted and he was discharged with a systolic murmur fully compensated.

Rhinorrhoea is one of the complications of scarlet fever which deserves more attention. Nasal discharges are frequently the cause of return cases. It has been my experience that most of the outbreaks of faucial and laryngeal diphtheria in scarlet fever wards are due to cases of rhinorrhoea which harbour the diphtheria bacillus. Such cases spread the infection of diphtheria by droplet infection, and cause an outbreak of diphtheria among the susceptible patients in the ward.

The nasal discharge may consist of mucus, be muco-purulent, or sanious. The nostrils become excoriated, an offensive discharge, often bloody, trickles over the lips and face causing irritation and impetiginous sores to develop on the face.

Careful examination of the septa narium in these cases frequently shows the presence of a membrane, and bacteriological examination of the nasal discharge demonstrates the presence of diphtheria bacilli. The treatment by antitoxin invariably clears up the condition and the case ought to be isolated to prevent further spread of the diphtheria bacilli.

Relapse of scarlet fever as a complication is not very common. Goodall states that it recurred in •8% of his cases. It is not such a frequent occurrence in scarlet fever as in typhoid or in the kindred affection due to streptococci erysipelas, in which despite the use of serum, relapses are common. In my series of cases treated with serum, •32% developed a relapse while in the control cases 2•7% developed this complication.

Generally, in relapse of scarlet fever we find individual symptoms less well marked than in the primary attack, but this is not always the case.

Some of these cases may terminate fatally with pneumonia or with nephritis even when the primary attack was mild.

The question of error in the original diagnosis must have attention, but discounting these discrepancies there are undoubtedly many cases of relapse of scarlet fever.

Relapses may be caused by an infection by another member of the streptococcal group to that of the original infection, as it has recently been shown that there are four groups of haemolytic streptococci which cause scarlet fever.

(1). P.de F. aged 8 years, was admitted on the 12th. July 1930 with scarlet fever. She had a general punctate erythema, faucial injection and a temperature of 101°, and within seven days she desquamated. On the 24th. Aug. 1930 she developed a temperature of 103·4°, had severe faucial symptoms and a general punctate erythema. This was diagnosed as a relapse of scarlet fever from which she made an uneventful recovery.

(2). E.M. aged 9 years, was admitted on the 17th. Sept. 1930 suffering from a sore throat. She had a temperature of 99°and a general punctate erythema, and the fauces were injected. This erythema faded after 24 hours, and desquamation was present on the 5th. day. On the 12th. day she developed a temperature of 101° and a sore throat. When examined she had a bright general punctate erythema and faucial injection. This subsided within four days and she made a complete recovery.

Pyogenic infections of the skin such as boils, furunculosis and paronychia occur from time to time during convalescence. Such complications are probably due to lessened resistance on the part of the debilitated patient and also to the exposure of the new skin after desquamation.

Pulmonary complications in scarlet fever are more common in septic cases than in mild cases and usually consist of broncho-pneumonia. The prognosis depends on the strength of the heart as it does in primary pneumonia. In one case which I shall record antistreptococcal serum was beneficial in the treatment.

G.G. aged 4 years, was admitted with scarlet fever on the 14th. July 1930. She also had pain in the left side of the abdomen. Her temperature was 103°, pulse 120 and respirations 38. On the 18th. July she had severe cough and dyspnoea dullness and bronchial breathing over the left base behind. On the 19th. July 20c.c. antistreptococcal serum was given, and on the 20th. the respiratory distress was lessened and her temperature was 101°. On the 22nd. July her temperature was normal and she made a complete recovery.

Pleurisy and empyema occur from time to time.

Gastrointestinal complications are rare in scarlet fever. Cases of streptococcal peritonitis occurring during the course of scarlet fever have been reported. One case reported in the M.A.B. annual report

1925 by Dr. Inkster, describes the occurrence of a streptococcal peritonitis which she concluded, on account of the absence of focal infection to have been a specific manifestation of the scarlatinal organism.

A case in my series however, had symptoms of severe colitis with blood and mucus in the stools, which occurred on the fourth week of illness.

L.F. aged 7 years, was admitted with a moderately severe attack of scarlet fever on the 21st. April 1930. On the 27th. May he developed a temperature of 102° with symptoms of diarrhoea, and blood and mucus in the stools. He had a remittent temperature for seventeen days. The line of treatment followed was 30c.c. antistreptococcal serum, glycerine enemas and pituitary extract. The bacteriological report on the urine and faeces was negative for streptococcus haemolyticus and for the coli typhoid group.

(22)

Henoch, in his lectures on infectious diseases states that profuse diarrhoea occasionally sets in at the commencement of the disease in malignant cases, so suddenly and severely that it induces a state of collapse resembling cholera.

Jaundice occurs from time to time in scarlet fever. It is not of grave significance and is usually caused by catarrh of the bile ducts or by a mild interstitial hepatitis.

P.G. aged 4 years, was admitted with scarlet fever on the 29th. Oct. 1930. He was given 30c.c. anti-streptococcal serum. He vomited one evening and on the following day he had signs of jaundice with bile in the urine. This cleared up within thirteen days.

Diseases of the blood are rare as complications of scarlet fever. Anaemia is frequent however, after such complications as nephritis but improves under appropriate treatment with iron. Several cases of purpura fulminans have been reported from time to time, and one case is of interest which developed this disease during her convalescence from scarlet fever at this hospital.

J.V.B. aged 8 years, was admitted on the 7th. June 1930 with scarlet fever and had an uneventful illness with the exception of the development of bilateral cervical adenitis on the 23rd. June. On the 27th. June she developed petechiae on the buttocks and a severe haemorrhage from the vagina. On the 28th. there was extensive haemorrhage into the right thigh and left knee and extensive ecchymosis of the right upper eyelid. These extravasations of the blood became so tense that blisters and bullae developed on the surface. Despite treatment with serum and calcium salts she died on the 30th. June. At the autopsy a general anaemia and blanching of the heart, liver and spleen was found.

J. D. Rolleston and Mc.Cririck report a similar case in the British Journal of Children's Diseases 1910. (23) Henoch who first described the disease as "purpura fulminans" collected several cases, and he concludes his

lecture by stating that "How the haemorrhagic diathesis comes to arise after scarlet fever we do not know. Perhaps it is owing to the occurrence of molecular changes in the walls of the small vessels producing a liability to rupture".

From these observations I consider that scarlet fever is still a disease with which the public health authorities have to deal. True it is, that the case mortality has diminished and that the majority of the cases are of a much milder type than that which prevailed ten or twenty years ago, still what guarantee has the community that a severe epidemic of septic or toxic scarlet fever may not occur which will tax the resources of any public health authority.

The utility of the isolation hospital in the prevention and in the treatment of complications is well known. In addition they can become centres of research into infectious diseases and other diseases of childhood.

I have stressed the importance of the early diagnosis of scarlet fever and the use of the Schultz Charlton reaction in the doubtful exanthemata, and the value of the Dick test in the recognition of susceptible individuals.

The serum treatment of scarlet fever has not met with the great success which followed the use of antidiphtheritic serum in diphtheria. Serum, however, has a definite place in the therapeutics of the disease, and if given at the onset in severe cases, it causes an immediate fall of temperature, it diminishes the toxaemia and causes the rash to fade at an early date.

The severity and the number of the complications are lessened by the early administration of serum in severe cases.

The treatment of the complications when they have arisen is not affected by the administration of serum, since the streptococci first cause toxaemia which damages the tissues and when the damage has occurred the antiserum is of very little avail.

REFERENCES.

(1) Buchanan, Sir George.
"Some Recent Policies Regarding Particular Diseases."
British Medical Journal 1931. p.283.

(2) Annual Report.
Metropolitan Asylums Board 1929. p. 108.

(3) Rolleston, J. D.
"The History of Scarlet Fever"
British Medical Journal 1928. p.926.

(4) Cock.
Edinburgh Medical & Surgical Journal, January 1832.

(5) Mallory, F.B.
"Protozoon-like bodies Found in Four Cases of Scarlet Fever." Journal of Medical Research 1904, Vol.V p.483.

(6) Dick, G.F. and Dick, G.H.
Journal of American Medical Association 1924. LXXXiii. p. 84.

(7) Mair, W. M.D.,
On the "Nature of the Schultz Charlton reaction in Scarlet Fever." Annual report of Metropolitan Asylums Board 1924. p.157.

(8) State of the Public Health. 1924. p.49.

(9) Moore, Dr. S.
"The Infectivity of Desquamation in Scarlet Fever."
"Public Health" 1908. p.148.

(10) Glover, Alison J. and Griffiths, F.
"Acute Tonsillitis and some of its sequelae."
British Medical Journal 1931. p.521.

(11) Banks,H., Stanley, P., Mackenzie, J.C.H.
"Intravenous Antitoxin in Scarlet Fever."
"The Lancet" 1929. p.381.

(12) Goodall, E.W.
"Scarlet Fever, Diphtheria and Enteric Fever 1895-191
"A Clinical Statistical Study" p.16.

(13) Rolleston, J. D.
"Acute Infectious Diseases." p. 286.

(14) Quarterly Journal Medicine 1928. LXXXV. p.119

(15) Report of St. Andrew's Institute 1926. iii. p.119.

(16) Ascherson, N., F.R.C.S.
"The Investigation of the Source of Chronic Ear Discharge."
Clinical Journal, 18th March, 1931.

(17) McCarrison, Robert.
"A Lecture on Some Surgical Aspects of Faulty Nutrition."
British Medical Journal 1931. p.970.

(18) Ker.
"Infectious Diseases" p.117.

(19) Robb, A. Gardner, M.B. O.P.A.
"Specific Antitoxin in Scarlet Fever."
British Medical Journal 1926. p.11.

(20) Fischer, M.
"Oedema and Nephritis."

(21) Annual Report. Metropolitan Asylums Board 1927. p.77.

(22) Henoch.
"Lectures on Children's Diseases." Vol.ii. p.52.
New Sydenham Society 1889.

(23) Idem. p.379.

APPENDIX.

A. The Schultz-Charlton Test.

The reagent for this test consists of scarlet fever antitoxin, and that used in the cases quoted was made at the Belmont Laboratories of the London County Council. The serum is injected intradermally into an area of the erythema, and the amount injected consists of ·2 c.c. A positive result is denoted by the blanching of the rash, and occurs within 8 to 24 hours after the injection.

B. The Dick Test.

This test for susceptibility to scarlet fever consists in the intradermal injection of ·2 c.c. of scarlet fever toxin, and the site selected is usually the flexor aspect of the left forearm. ·2 c.c. of heated toxin is injected into the skin in the same region of the right forearm as a control. The results are read the following day, and consist of positive and negative reaction. The susceptible patients show a red flush at the site of the injection lasting some days. Those who are immune show no reaction. Patients who give a reaction with the heated toxin are considered to be immune unless the reaction with the unheated toxin is much greater than with the heated toxin.

C. The intramuscular injection of serum.

The skin over the lateral aspect of the thigh is thoroughly disinfected and cleansed with spirit, then tincture of iodine is applied. The serum is injected deeply into the muscles of the thigh.

D. Temperature charts.

1 - 5	To show the effect of serum on temperature and pulse in scarlet fever.
6 - 8	Cases of otitis media showing effect of paracentesis and Wilde's incision.
9 - 10	Scarlet fever with acute nephritis and uraemia.
11	Scarlet fever with acute rheumatism treated with anti-streptococcal serum.
12	Scarlet fever complicated by rheumatism and pericarditis.

D. (contd.)

13	Scarlet fever complicated by acute rheumatism and endocarditis, treated with anti-streptococcal serum.
14	Scarlet fever complicated by transient toxic myocarditis, treated with anti-streptococcal serum.
15 & 16	Scarlet fever with relapses.
17	Scarlet fever with pneumonia, treated with anti-streptococcal serum.
18	Scarlet fever complicated by colitis, treated with serum.
19	Scarlet fever - complication purpura fulminans.

(1)

London County Council.

Form 305.

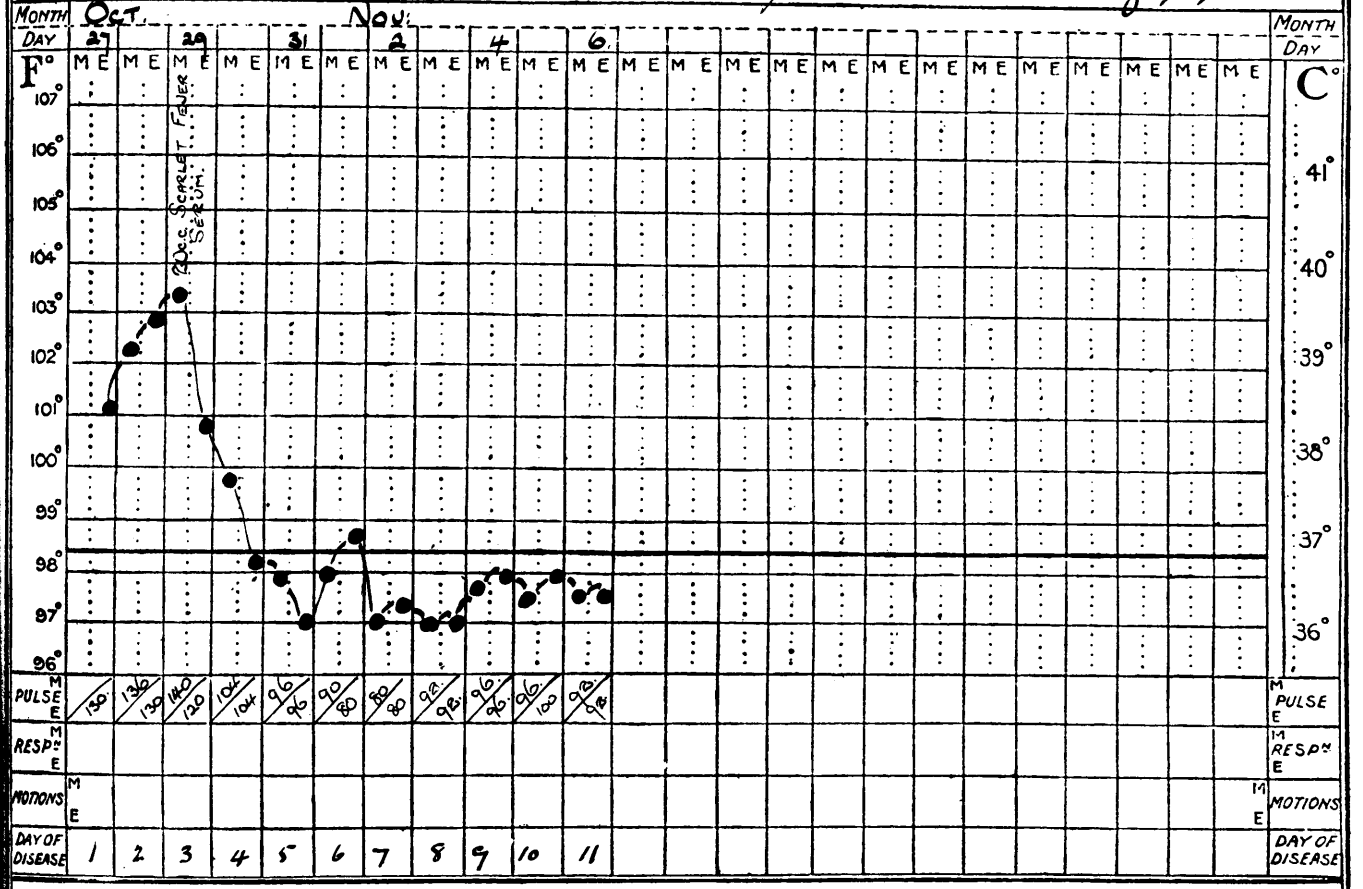
Southern HOSPITAL.

PATIENT'S NAME J. C. AGE 8.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever: Treatment with serum on third day of illness causing rapid diminution of pyrexia



URINE.

DATE																							
No. of Oz. in 24 hrs																							
REACTION																							
SP. GR.																							
ALBUMEN																							
BLOOD																							
UREA																							
Microscop' Appearance Etc.																							
COUGH Simple																							
Spasmodic																							
Whoop																							

(2)

London County Council.

Form 305.

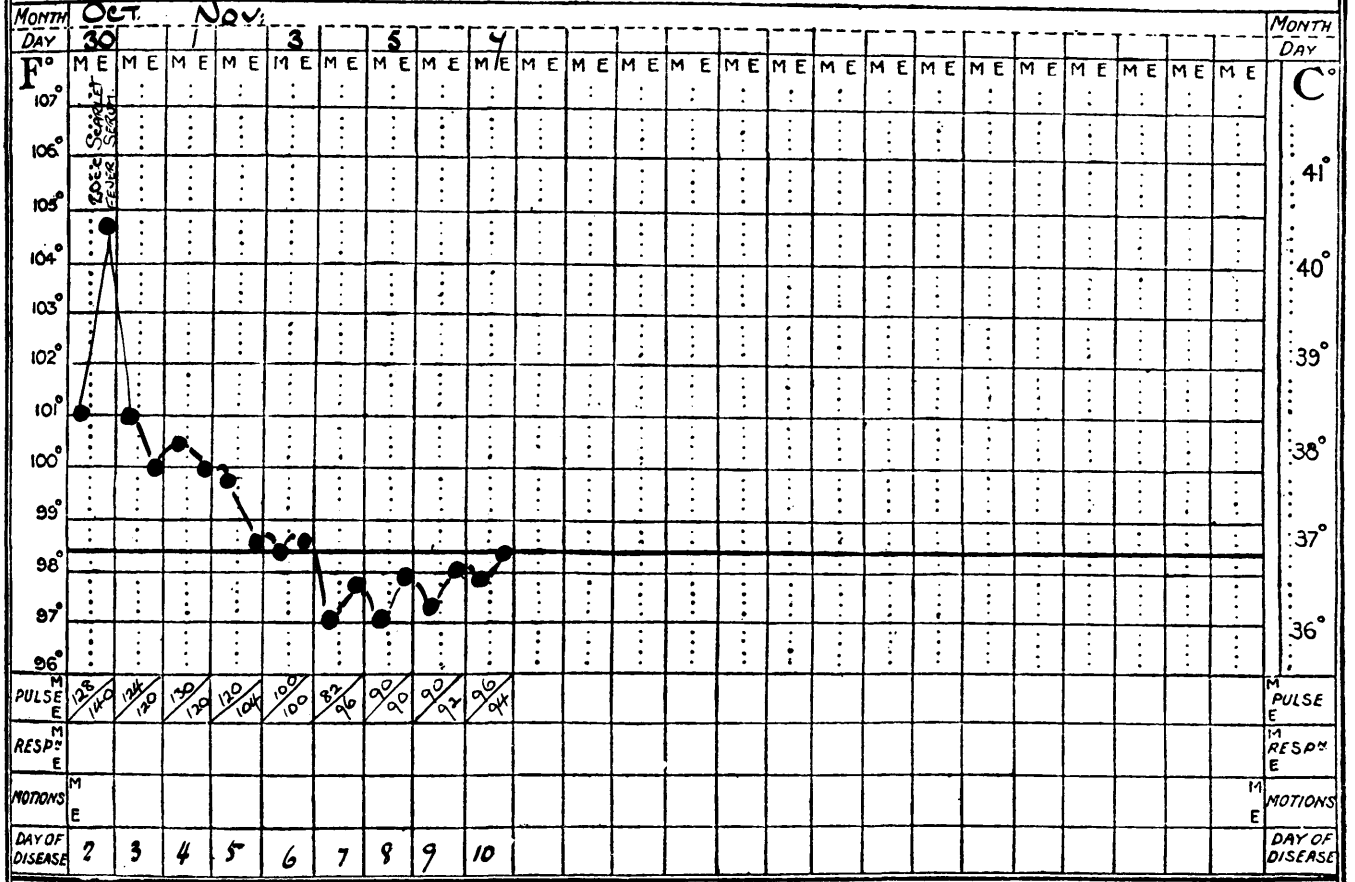
Southern HOSPITAL.

PATIENT'S NAME L. H. AGE 6.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. To show effect of administration of antitoxin in pyrexia.



URINE.

DATE																								
N° of oz. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
" Spasmodic																								
" Whoop																								

Form 305.

(3)

London County Council.

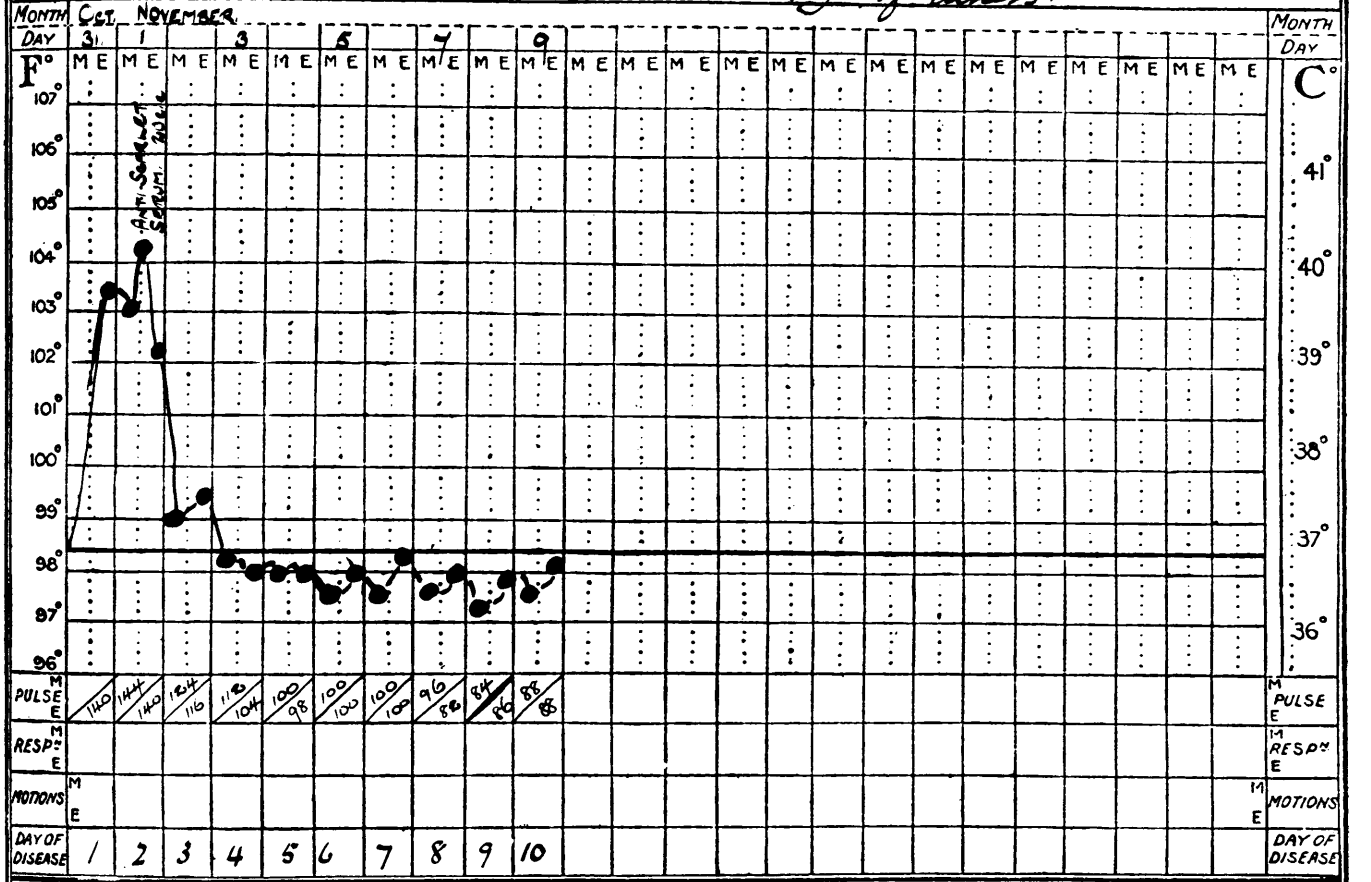
Southern HOSPITAL.

PATIENT'S NAME L. C. AGE 9

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever: To show effect of scarlet fever antitoxin on temperature and pulse. Serum given 2nd day of illness.



URINE.

DATE																								
No of 03. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
Spasmodic																								
Whoop																								

Form 305.

(4)

London County Council.

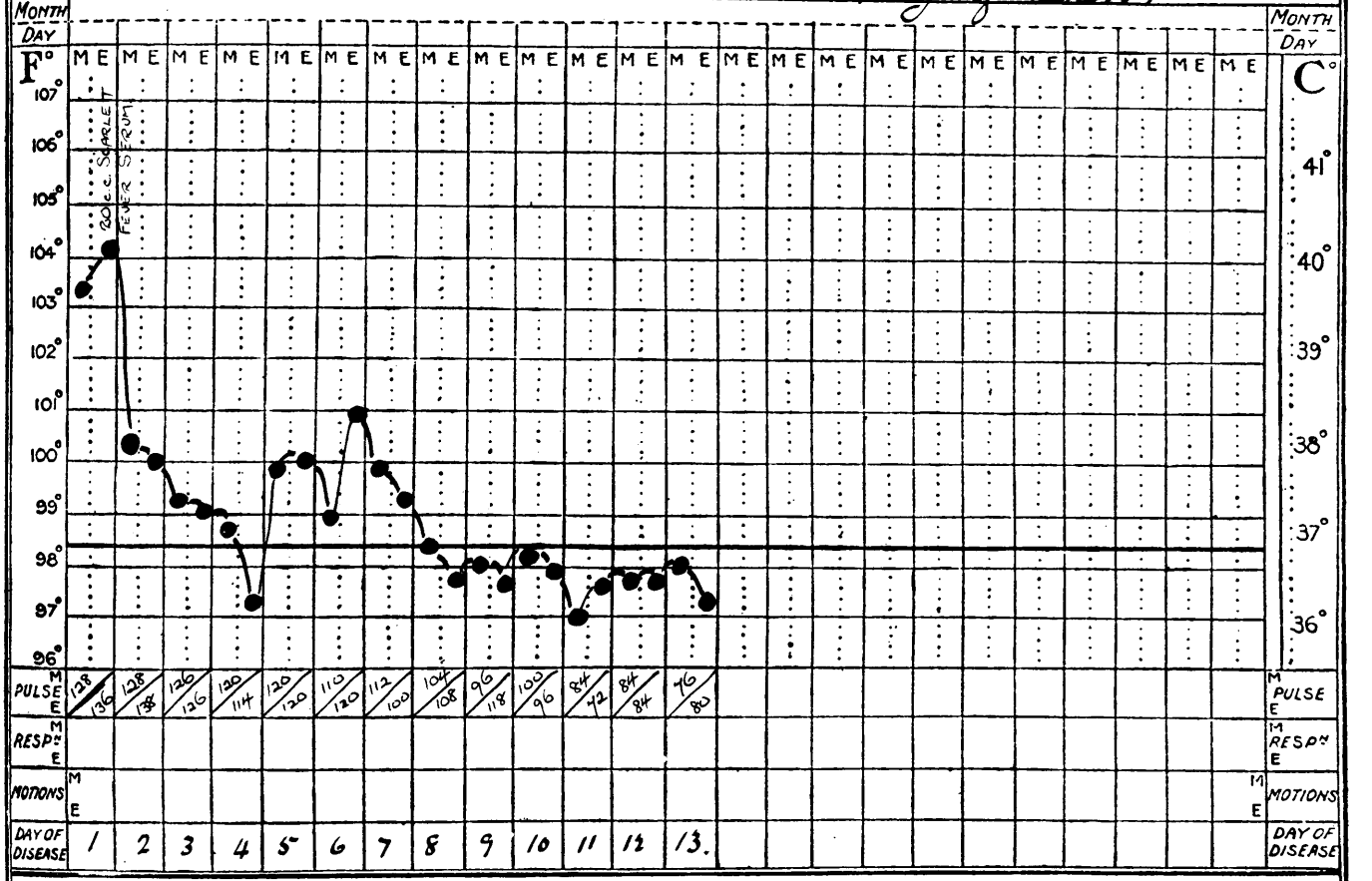
Southern HOSPITAL.

PATIENT'S NAME J. G. AGE 3½.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. To show effect on temperature and pulse of 20 c.c. Scarlet fever antitoxin Given on 1st day of illness.



URINE.

DATE																								
No of 03. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
" Spasmodic																								
" Whoop																								

Form 305.

London County Council.

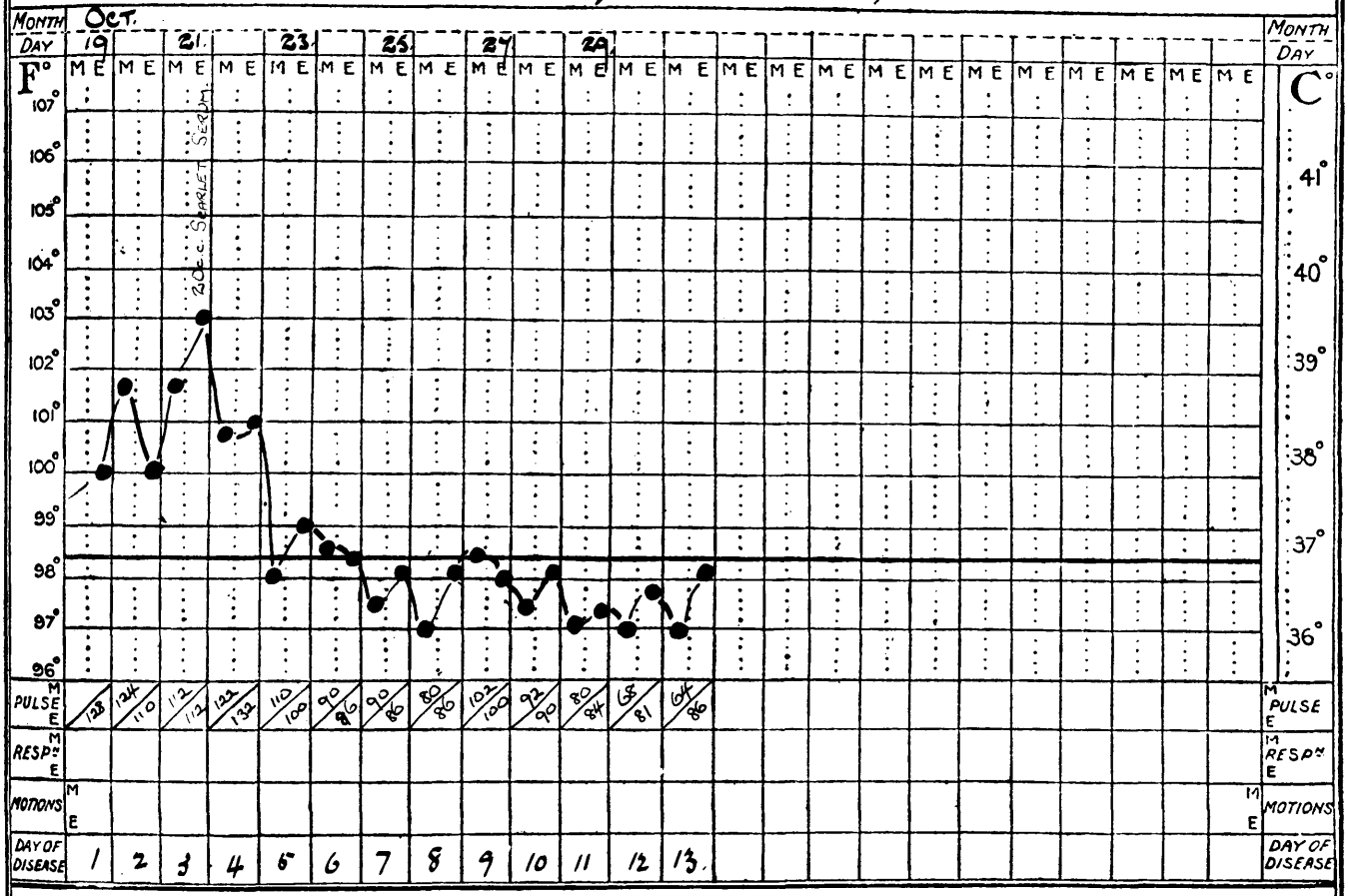
(5) Southern HOSPITAL.

PATIENT'S NAME L. W AGE 3½ years.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. To illustrate effect of scarlet fever antitoxin on temperature and pulse.



URINE.

DATE	
No of 03. in 24 hrs	
REACTION	
SP. GR.	
ALBUMEN	
BLOOD	
UREA	
Microscop' Appearance Etc.	
COUGH Simple	
Spasmodic	
Whoop	

London County Council.

Form 305.

(6).

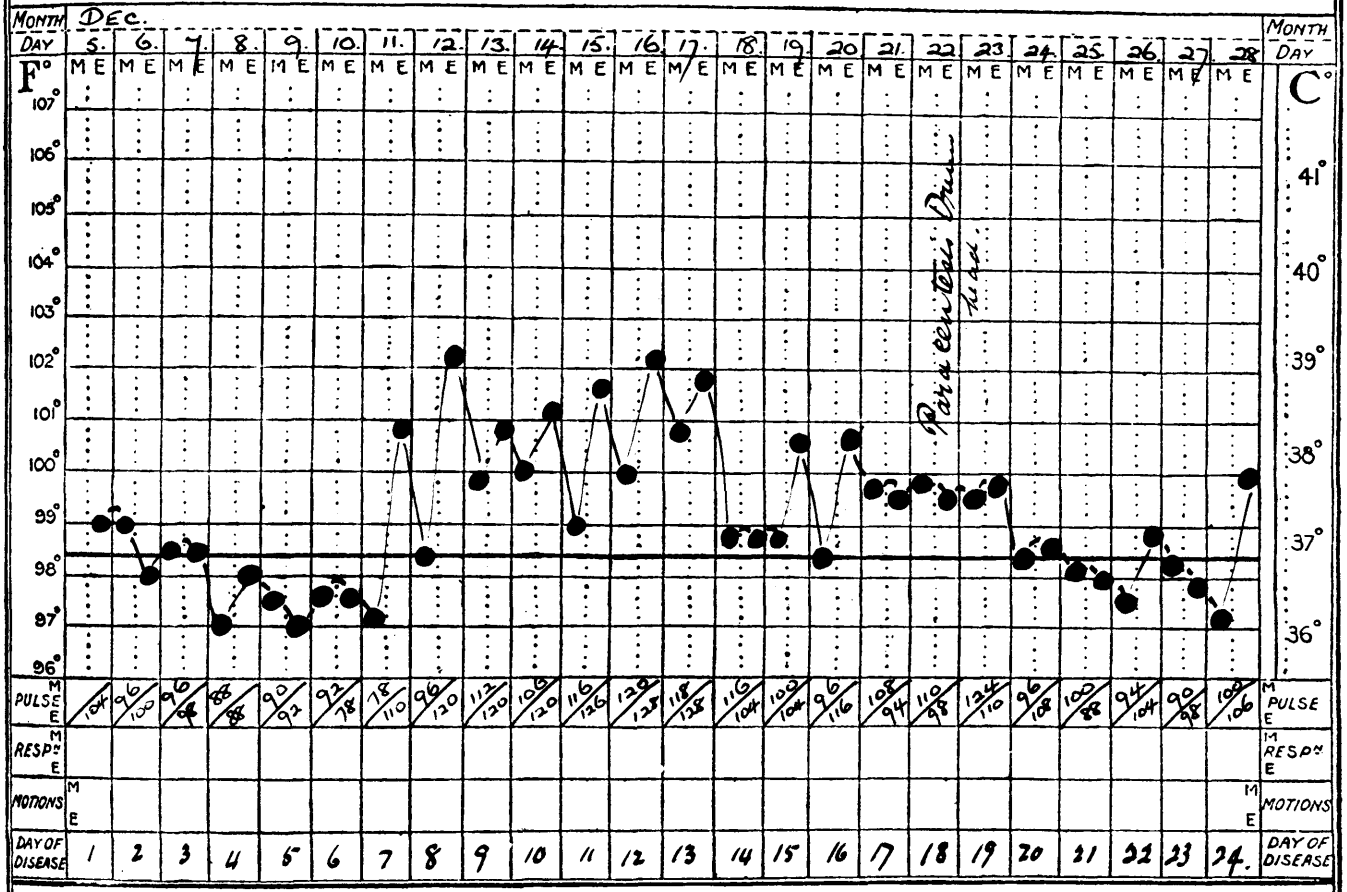
Southern HOSPITAL.

PATIENT'S NAME E. B. T. AGE 7

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Otitis Media. Treatment by paracentesis and Wilde's Incision.



URINE.

Date																								
No of oz. in 24 hrs																								
Reaction																								
Sp. Gr.																								
Albumen																								
Blood																								
Urea																								
Microscop' Appearance Etc.																								
COUGH Simple																								
" Spasmodic																								
" Whoop																								

London County Council.

Form 305.

61

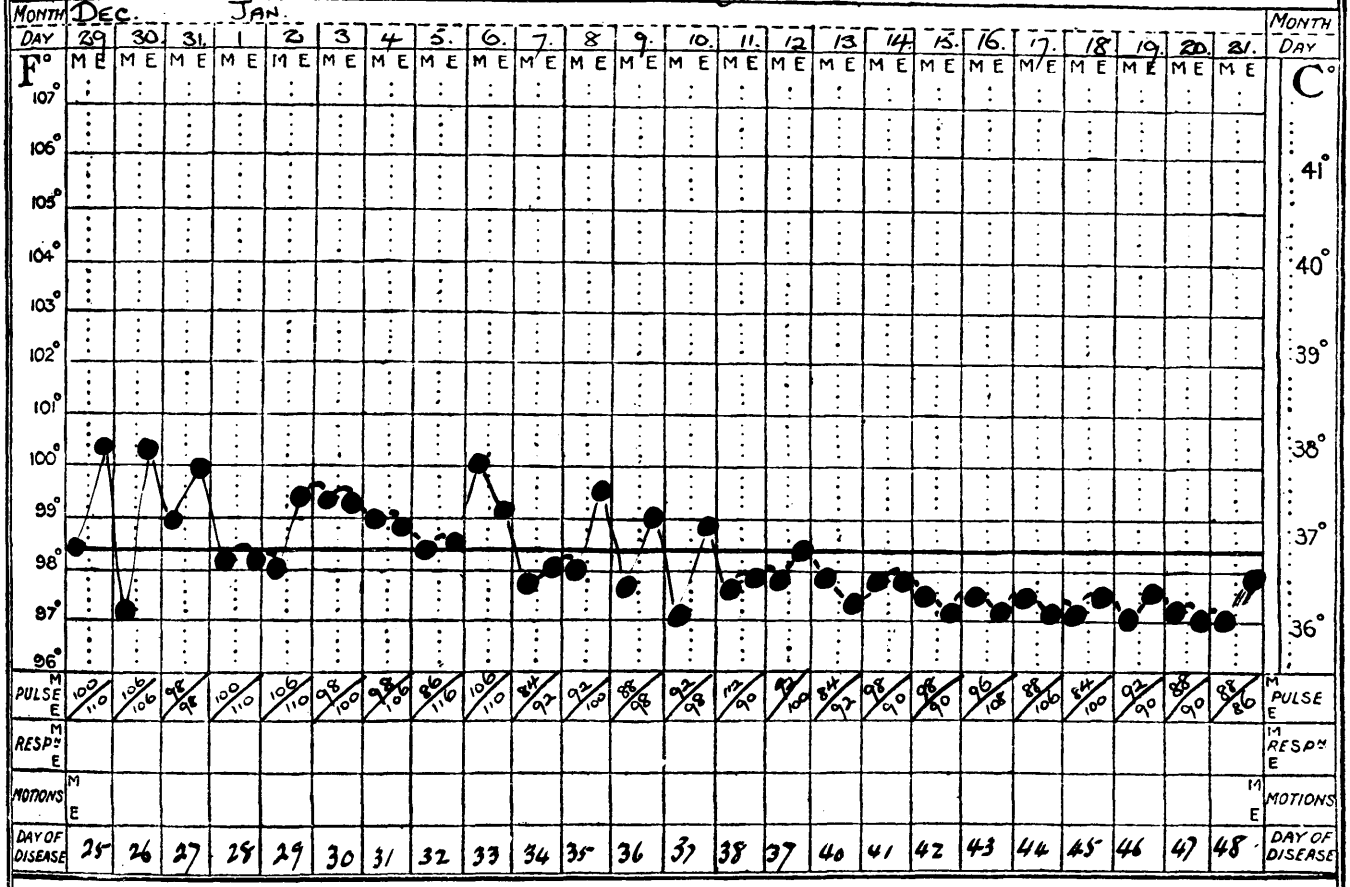
Southern HOSPITAL.

PATIENT'S NAME E.B.J. AGE 7.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Otitis media
Treated by Paracentesis and Wilde's incision



URINE.

DATE																								
N° of 03. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
" Spasmodic																								
" Whoop																								

London County Council.

Form 305.

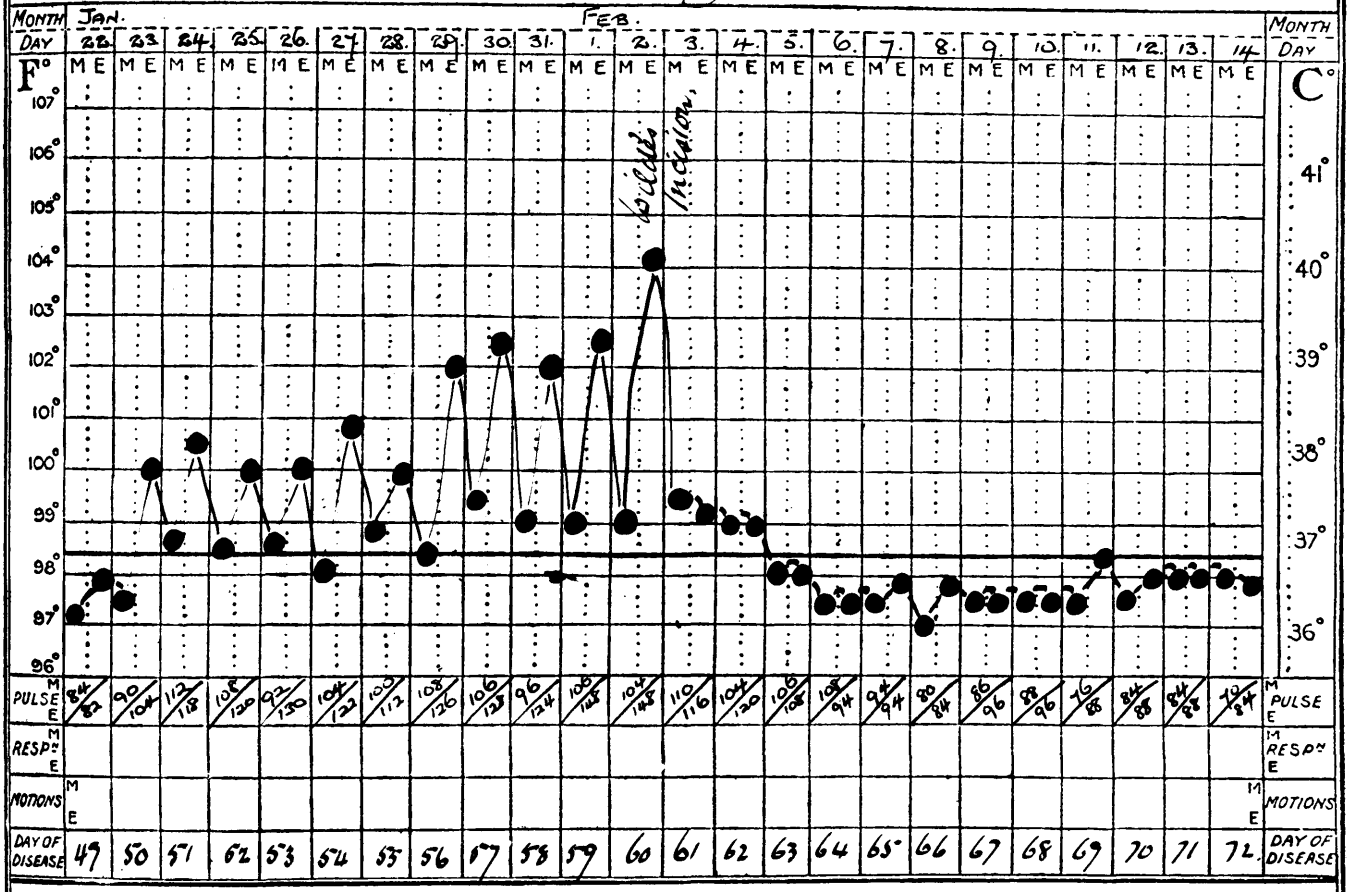
(6) Southern HOSPITAL.

PATIENT'S NAME E.S. AGE 7.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. Otitis media
Treated by Paracentesis and Wilde's incision



MONTH	JAN.										FEB.														MONTH
DAY	22	23	24	25	26	27	28	29	30	31	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14	DAY
F° / C°	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	F° / C°
PULSE	84/82	90/104	112/118	108/120	92/130	104/122	100/112	108/126	106/128	96/124	108/148	104/148	110/116	104/120	106/108	108/94	94/94	80/84	86/96	88/96	76/88	84/88	84/88	79/84	PULSE
RESP^N																									RESP^N
MOTIONS	M E																							M E	MOTIONS
DAY OF DISEASE	49	50	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	DAY OF DISEASE

URINE.

DATE																									
N° of oz. in 24 hrs																									
REACTION																									
SP. GR.																									
ALBUMEN																									
BLOOD																									
UREA																									
Microscop' Appearance Etc.																									
COUGH Simple																									
Spasmodic																									
Whoop																									

London County Council.

Form 305.

(4)

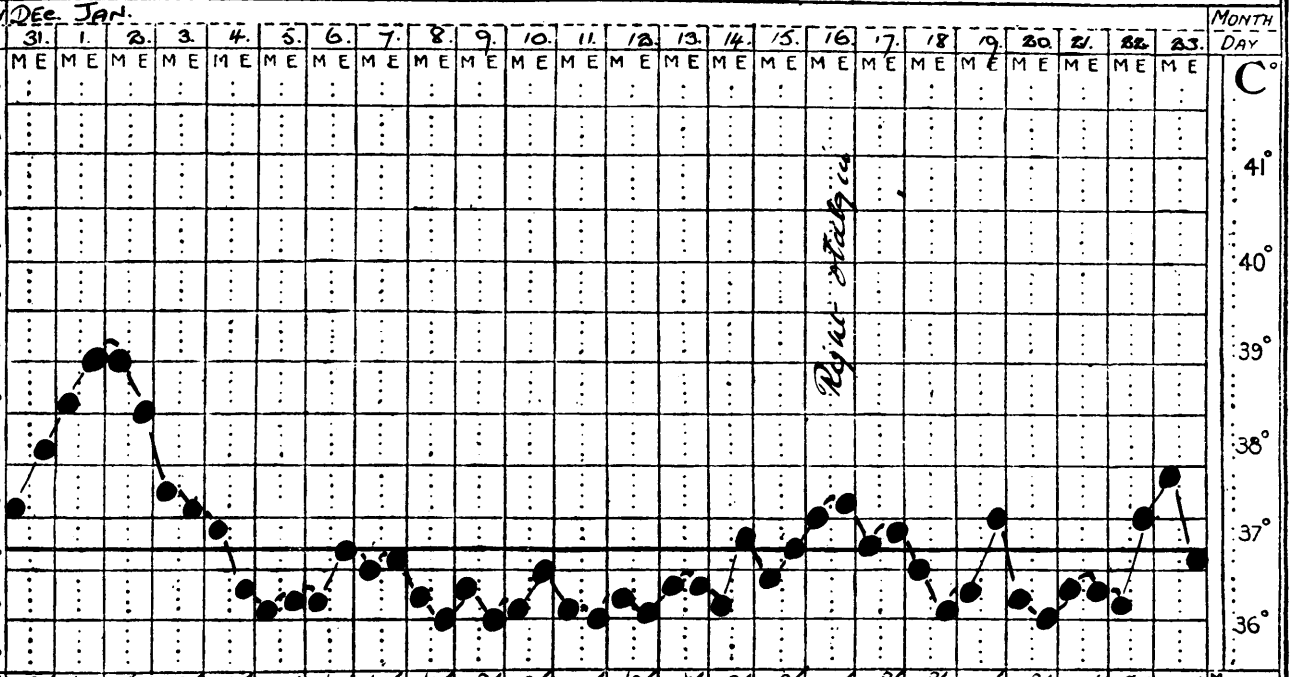
Southern HOSPITAL.

PATIENT'S NAME R.P.B AGE 4.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Complication Otitis media. Treated by Wilde's incision



MONTH	DEC. JAN.																								MONTH
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.	DAY
F° / C°	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	F° / C°
PULSE M	120	126	128	100	108	92	96	96	96	100	90	82	96	94	80	110	110	100	100	92	100	96	98	94	PULSE M
PULSE E	128	128	124	112	96	100	104	100	104	96	88	92	100	88	116	97	122	112	90	108	104	110	100	98	PULSE E
RESP^N M																									RESP^N M
RESP^N E																									RESP^N E
MOTIONS M																									MOTIONS M
MOTIONS E																									MOTIONS E
DAY OF DISEASE	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	DAY OF DISEASE

URINE.

DATE																								
N° of 03. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
Spasmodic																								
Whoop																								

London County Council.

Form 305.

(4)

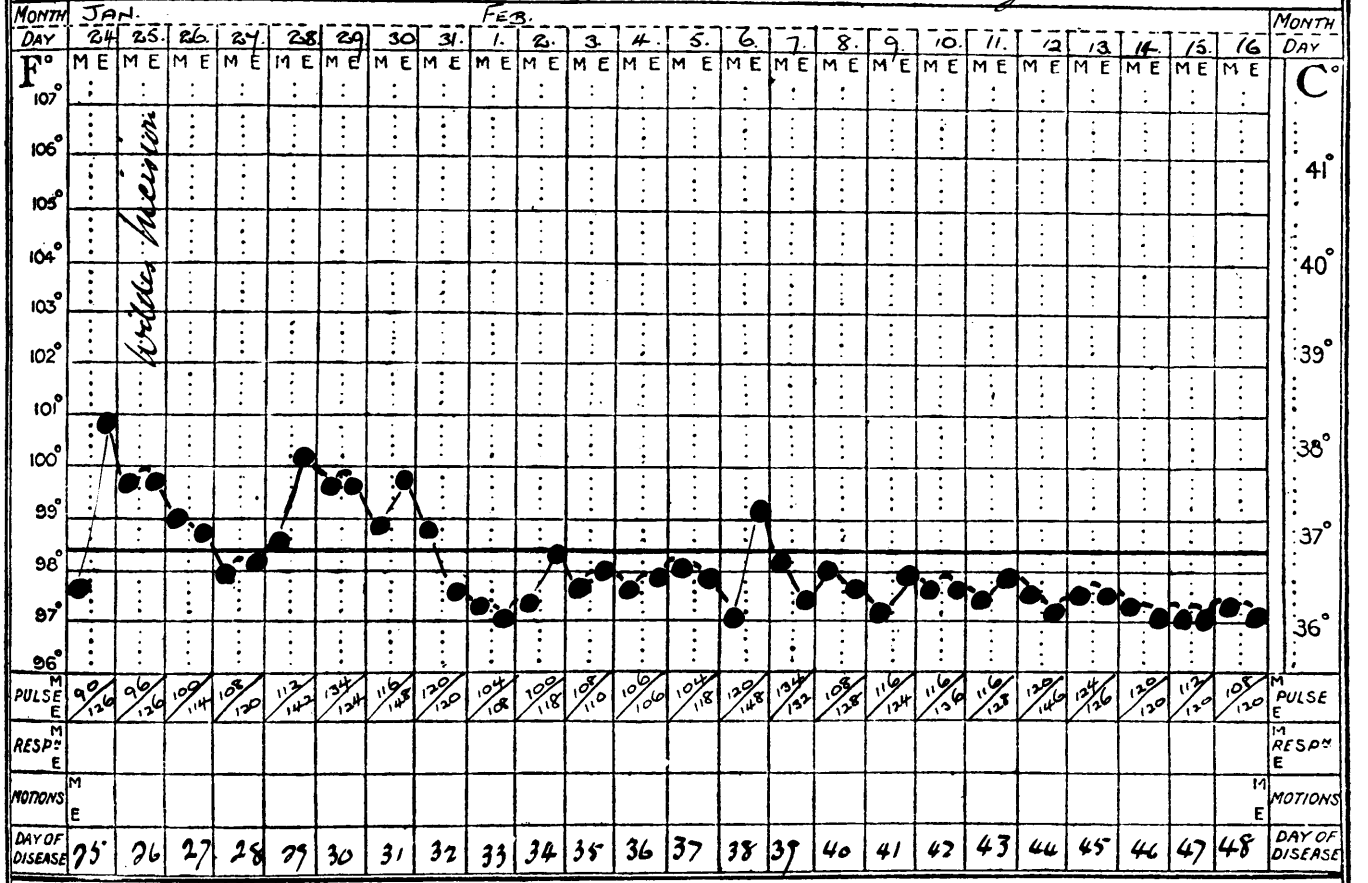
..... HOSPITAL.

PATIENT'S NAME R.P.B. AGE 11

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Complication Otitis Media
Treated by Wilde's Incision



URINE.

DATE																								
N° of oz. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
" Spasmodic																								
" Whoop																								

London County Council.

Form 305.

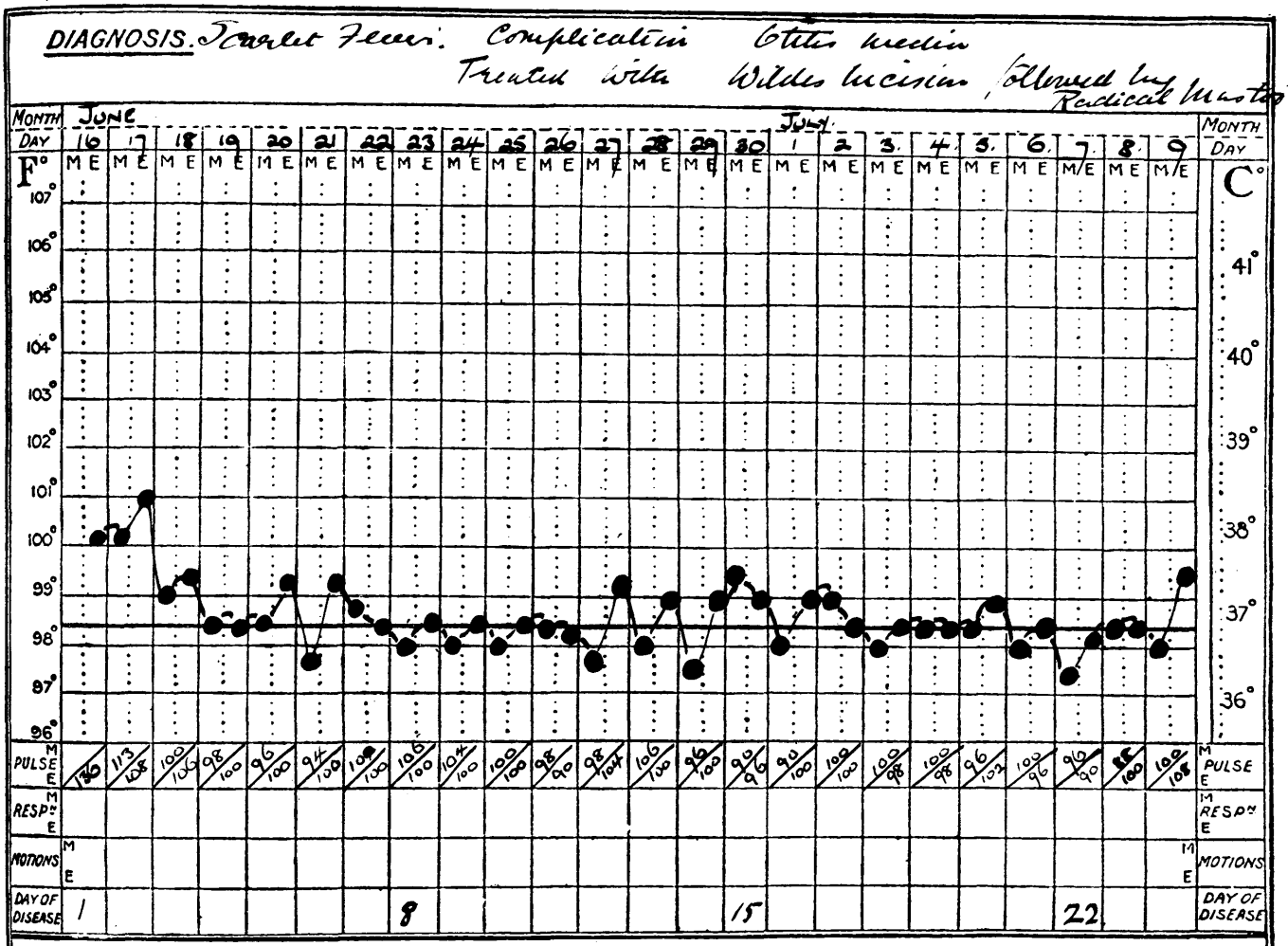
(8) Southern HOSPITAL.

PATIENT'S NAME M. A. J. AGE 8.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Complication Otitis media
Treated with Wildes incision, followed by Radical Mastoid



URINE.

DATE																										
No. of 03. in 24 hrs																										
REACTION																										
SP. GR.																										
ALBUMEN																										
BLOOD																										
UREA																										
Microscop' Appearance Etc.																										
COUGH Simple																										
" Spasmodic																										
" Whoop																										

London County Council.

Form 305.

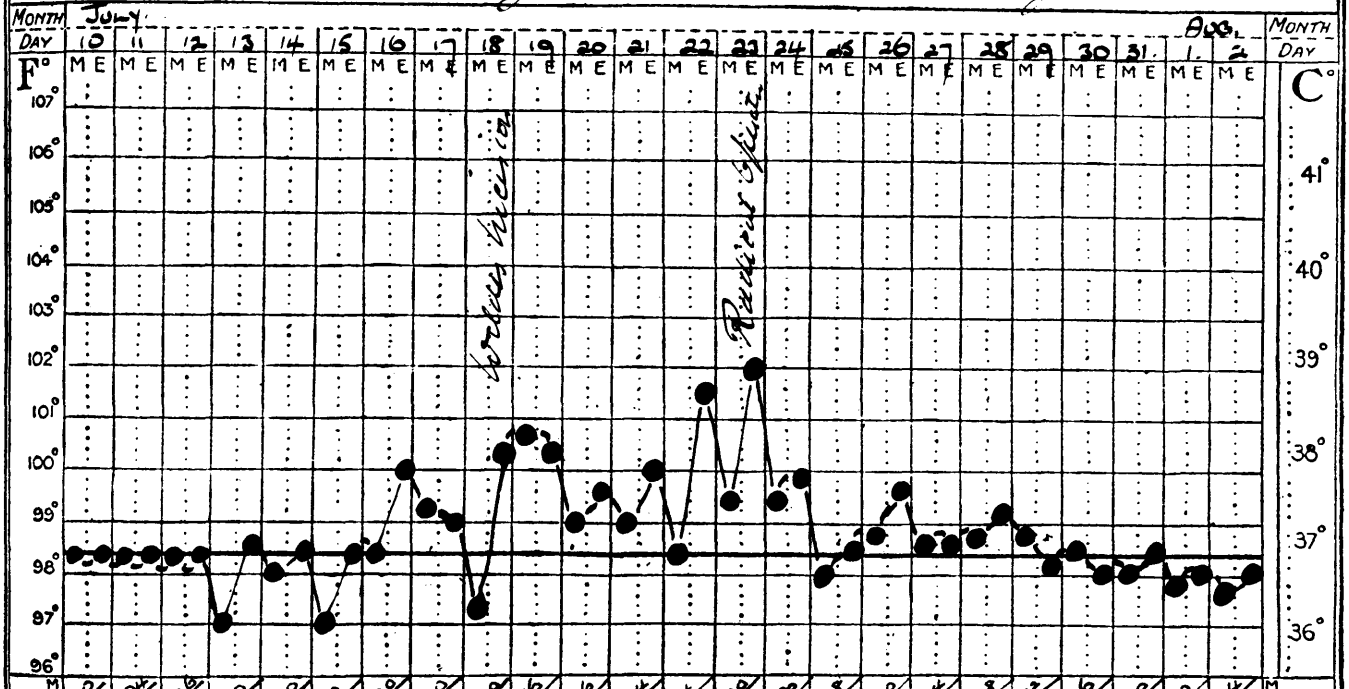
(8) Southern HOSPITAL.

PATIENT'S NAME F. A. S. AGE 8.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. complication Otitis media
Treated by Wilder Incision, followed by Radical mastoidectomy



MONTH	July																										Aug.		MONTH
DAY	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1	2					DAY
PULSE M	100	104	106	100	100	90	100	100	100	116	116	124	114	120	120	108	100	104	108	112	96	100	90	94					PULSE M
PULSE E	100	100	100	102	88	96	120	118	120	120	120	126	126	140	120	108	106	104	108	96	100	100	106	96					PULSE E
RESP^N M																													RESP^N M
RESP^N E																													RESP^N E
MOTIONS M																													MOTIONS M
MOTIONS E																													MOTIONS E
DAY OF DISEASE	25			28							35							42						48					DAY OF DISEASE

URINE.

DATE																													
N° of 03. in 24 hrs																													
REACTION																													
SP. GR.																													
ALBUMEN																													
BLOOD																													
UREA																													
Microscop' Appearance Etc.																													
COUGH Simple																													
Spasmodic																													
Whoop																													

London County Council.

Form 305.

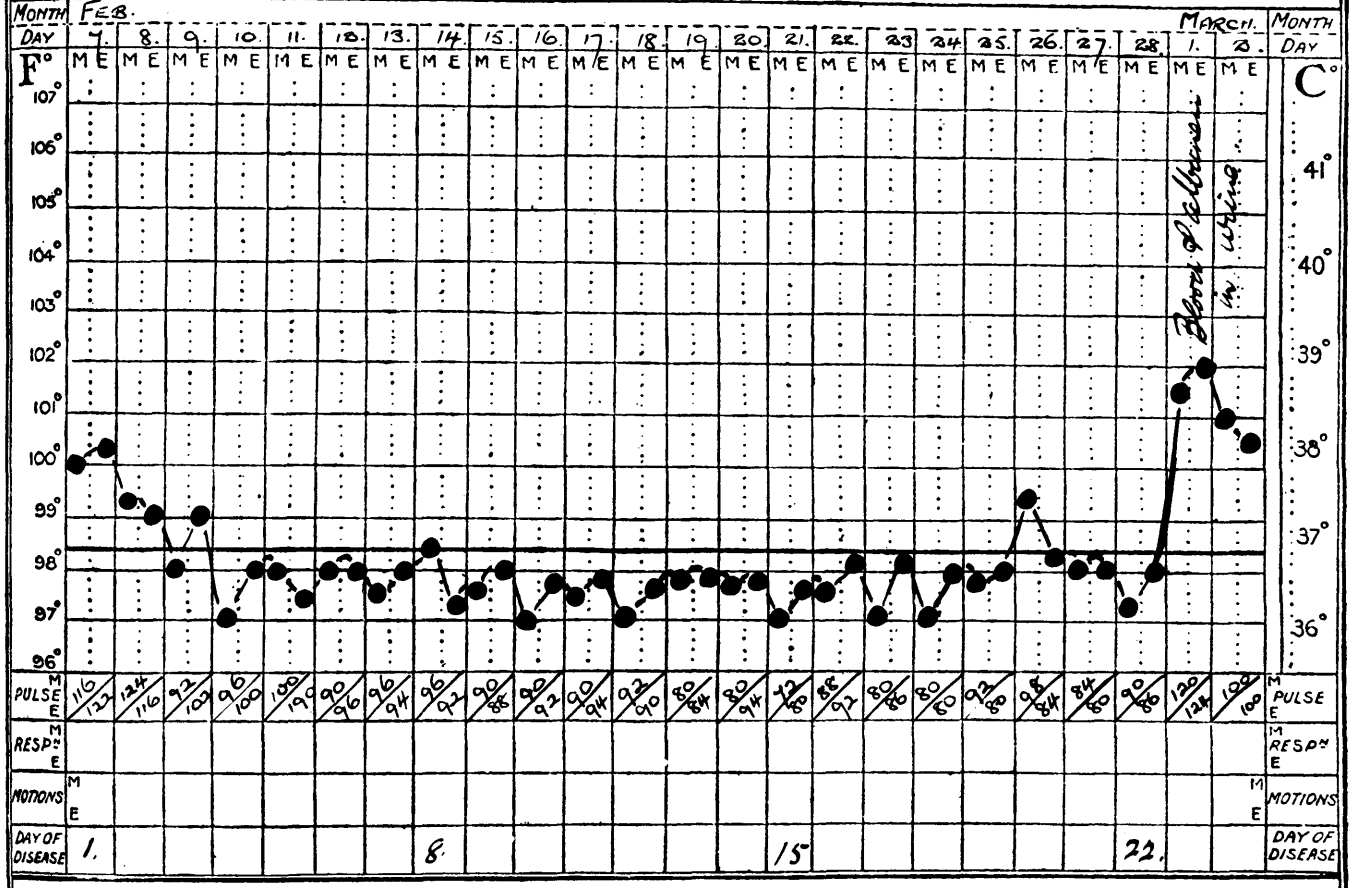
(9) Southern HOSPITAL.

PATIENT'S NAME T. Y. AGE 7.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Complication, acute nephritis at the third week ushered in by pyrexia



Month	Feb.																						March.		Month
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.	Day
F° / C°	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	M E	F° / C°
Pulse	116/122	124/116	92/102	96/100	100/100	90/96	96/94	96/92	90/88	90/92	90/94	92/90	80/84	80/94	72/80	88/92	80/86	80/80	92/80	98/84	84/80	90/86	120/124	100/100	Pulse
Respn																									Respn
Motions	M E																						M E		Motions
Day of Disease	1.							8.							15.							22.			Day of Disease

URINE.

Date	Feb. 8.	9.	10.	11.	12.	13.	14.	15.	16.	17.	18.	19.	20.	21.	22.	23.	24.	25.	26.	27.	28.	March 1.	2.	3.
No of Oz. in 24 hrs																						3xi	3xviii	3xxx.
Reaction	Ac.	Ac.	Ac.	Ac.		Ac.		Ac.		Ac.		Ac.		Ac.		Ac.		Ac.		Ac.		Ac.	Ac.	Ac.
Sp. Gr.																								
Albumen	Nil.	Nil.	Nil.	Nil.		Nil.		Nil.		Nil.		Nil.		Nil.		Nil.		Nil.		Nil.		Tr.	Cloud	Cloud
Blood																						+	++	+
Urea																								
Microscop' Appearance Etc.																								
Cough Simple																								
" Spasmodic																								
" Whoop																								

London County Council.

Form 305.

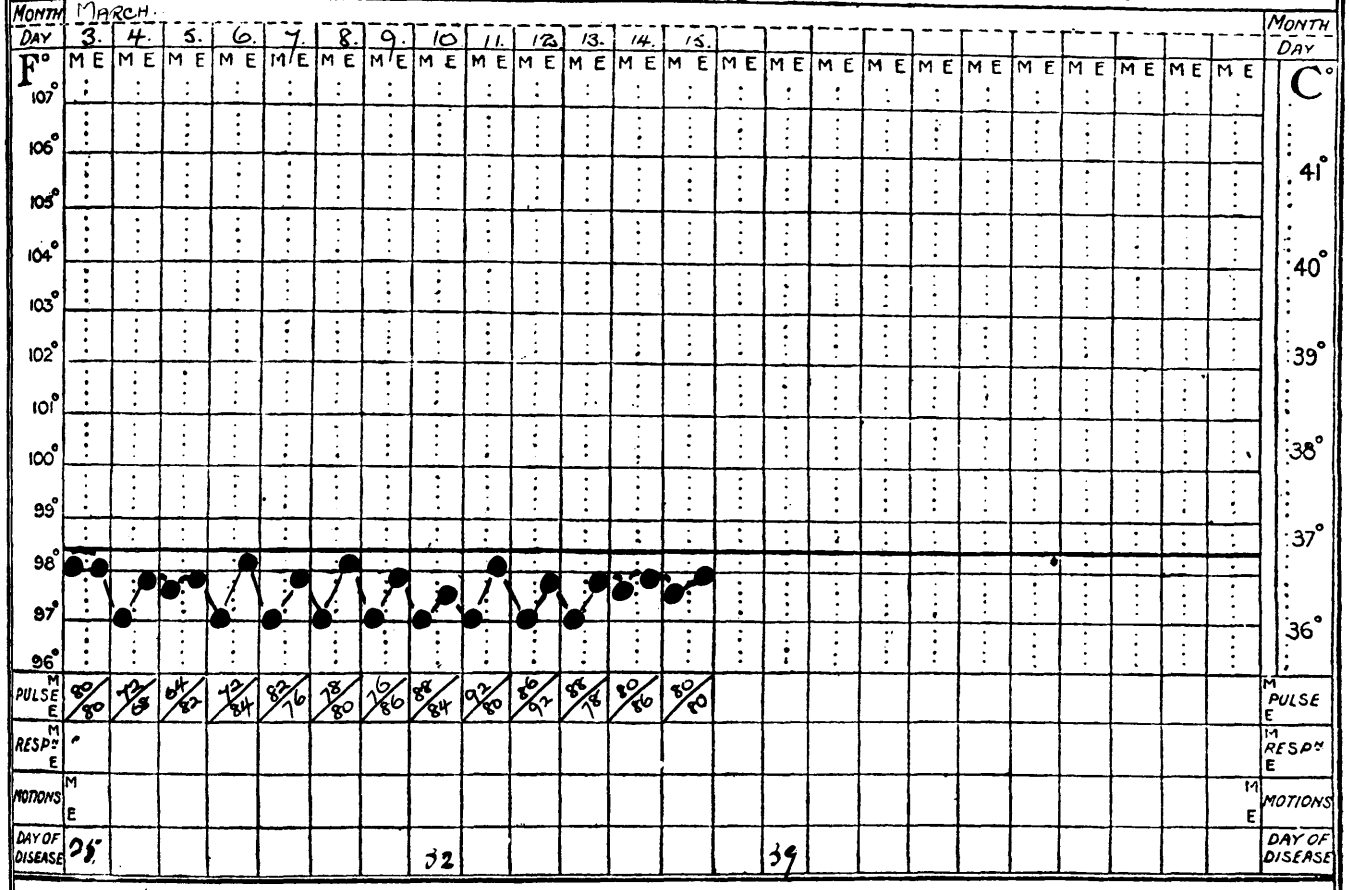
(9) Southern HOSPITAL.

PATIENT'S NAME J. Y. AGE 7

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Complication: acute nephritis at the third week of illness ushered in by pyrexia



URINE.

DATE	MARCH 4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.
No of 03. in 24 hrs	3xxxx	3xxxx	3xxxx	3xxxx	3xxxx	3xxxx	3xxxx	3xxxx	3xxxx	3xxxx		
REACTION	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.
SP. GR.												
ALBUMEN	Cloud	Cloud	Cloud	Cloud	Cloud	TRACE	TRACE	TRACE	TRACE	F.T.	NIL.	NIL.
BLOOD	NIL.	NIL.	NIL.	NIL.	NIL.	NIL.	NIL.	NIL.	NIL.	NIL.	NIL.	NIL.
UREA												
Microscop' Appearance Etc.												
COUGH Simple												
" Spasmodic												
" Whoop												

London County Council.

Form 305.

(10

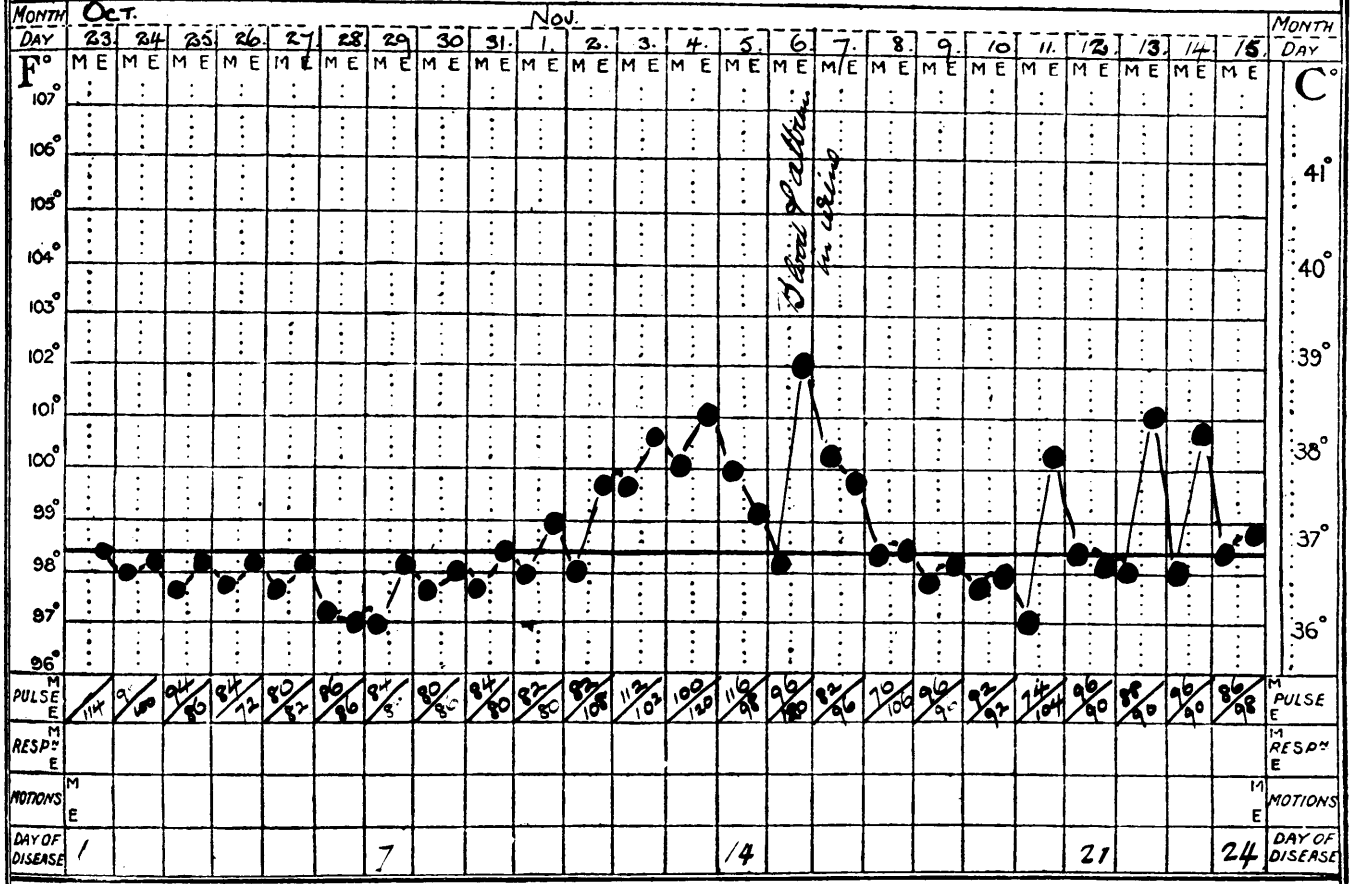
Southern HOSPITAL.

PATIENT'S NAME M. B. AGE 9

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever, complicated by acute nephritis and uraemia.



URINE.

DATE	Oct. 24		26		28		30		Nov. 1.		3.		5.	6.	7.	8.	9.	10	11.	12.	13	14	15	16
No of Oz. in 24 hrs															3vii	3xii	3iii	3iii	3iss	3vii	3xviii	3xiv	3xx	3xviii
REACTION	Ac.		Ac.		Ac.		Ac.		Ac.		Ac.		Ac.	Ac.		Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.	Ac.
SP. GR.																								
ALBUMEN	Nil.		Nil.		Nil.		Nil.		Nil.		Nil.		Nil.	+	Cloud	++	+	+	+	+	+	+	+	+
BLOOD														+	+	+++	+	+	+	TR.	TR.	TR.	TR.	+
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
Spasmodic																								
Whoop																								

Form 305.

London County Council.

(10)

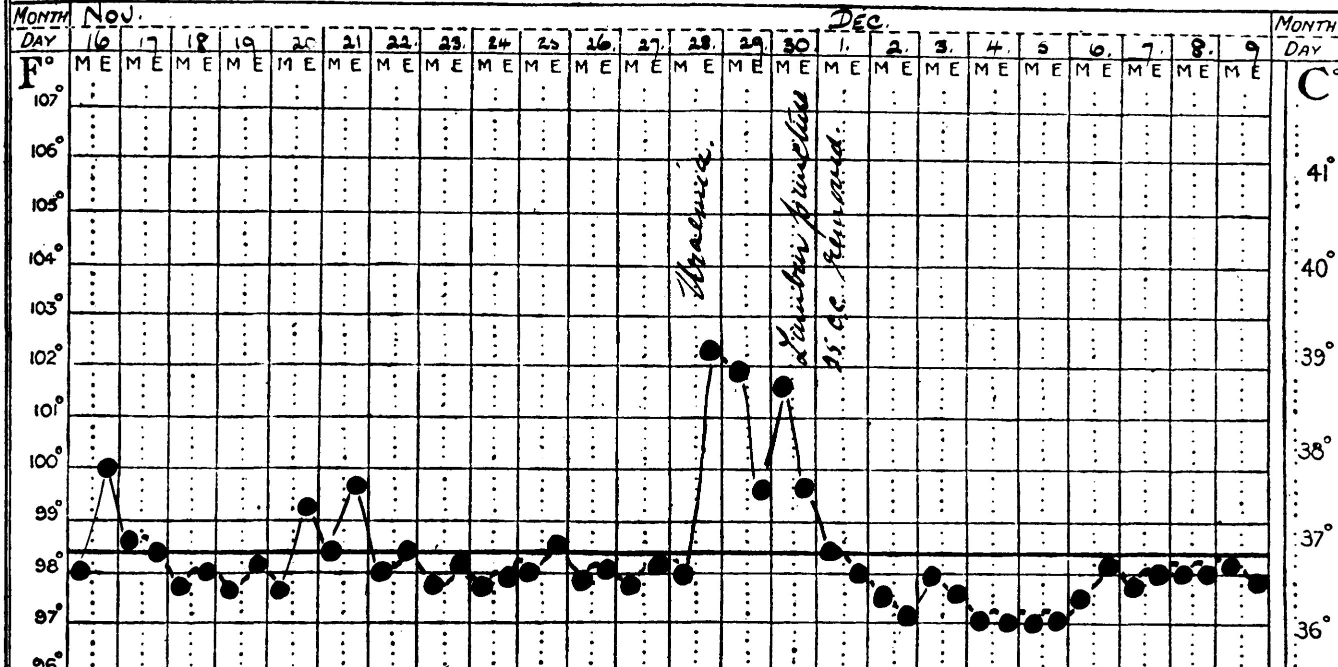
Southern HOSPITAL.

PATIENT'S NAME M. D. **AGE** 9

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Complicated by acute nephritis and uraemia



Month / Day	Pulse (M / E)	Day of disease
Nov. 16	92 / 106	2
17	92 / 92	
18	88 / 88	
19	84 / 86	
20	82 / 86	
21	82 / 90	7
22	86 / 84	
23	80 / 90	
24	80 / 90	
25	80 / 88	
26	84 / 80	
27	82 / 88	
28	120 / 126	14
29	130 / 100	
30	128 / 112	
Dec. 1	120 / 80	
2	100 / 76	
3	100 / 70	
4	84 / 76	21
5	78 / 80	
6	100 / 104	
7	100 / 68	
8	92 / 90	
9	90 / 90	26

RESP^N. — MOTIONS —

URINE.

Date	No. of oz. in 24 hrs	Reaction	Sp. Gr.	Albumen	Blood	Urea	Microscop' Appearance Etc.
Nov 17	℥xxii	Ac.		+	+		
18	℥xxx	Ac		+	+		
19	℥xxv	Ac.		+	+		
20	℥xxx	Ac.		+	+		
21	℥xxvi	Ac		+	+		
22	℥xxvi	Ac		+	+		
23	℥xl	Ac.		+	+		
24	℥xl	Ac		+	+		
25	℥xl	Ac		+	+		
26	℥xxxv	Ac		+	+		
27	℥xxvi	Ac.		+	+		
28	?	Ac.		+	+		
29	℥vi	Ac.		++	++		
30	?	Ac.		++	++		
Dec 1.							
2	℥i	Ac.		Tr.	Tr.		
3	℥v+	Ac.		Tr.	Tr.		
4	℥x+	Ac.		+	+		
5	℥xii	Ac.		+	+		
6	℥xviii	Ac.		+	Tr.		
7	℥xxii	Ac		+	Tr.		
8	℥xxi	Ac		+	Tr.		
9	℥xxv	Ac.		Tr.	Ft. Tr.		
10	℥xl	Ac.		Ft. Tr.	Ft. Tr.		

COUGH Simple — Spasmodic — Whoop —

London County Council.

Form 305.

(11)

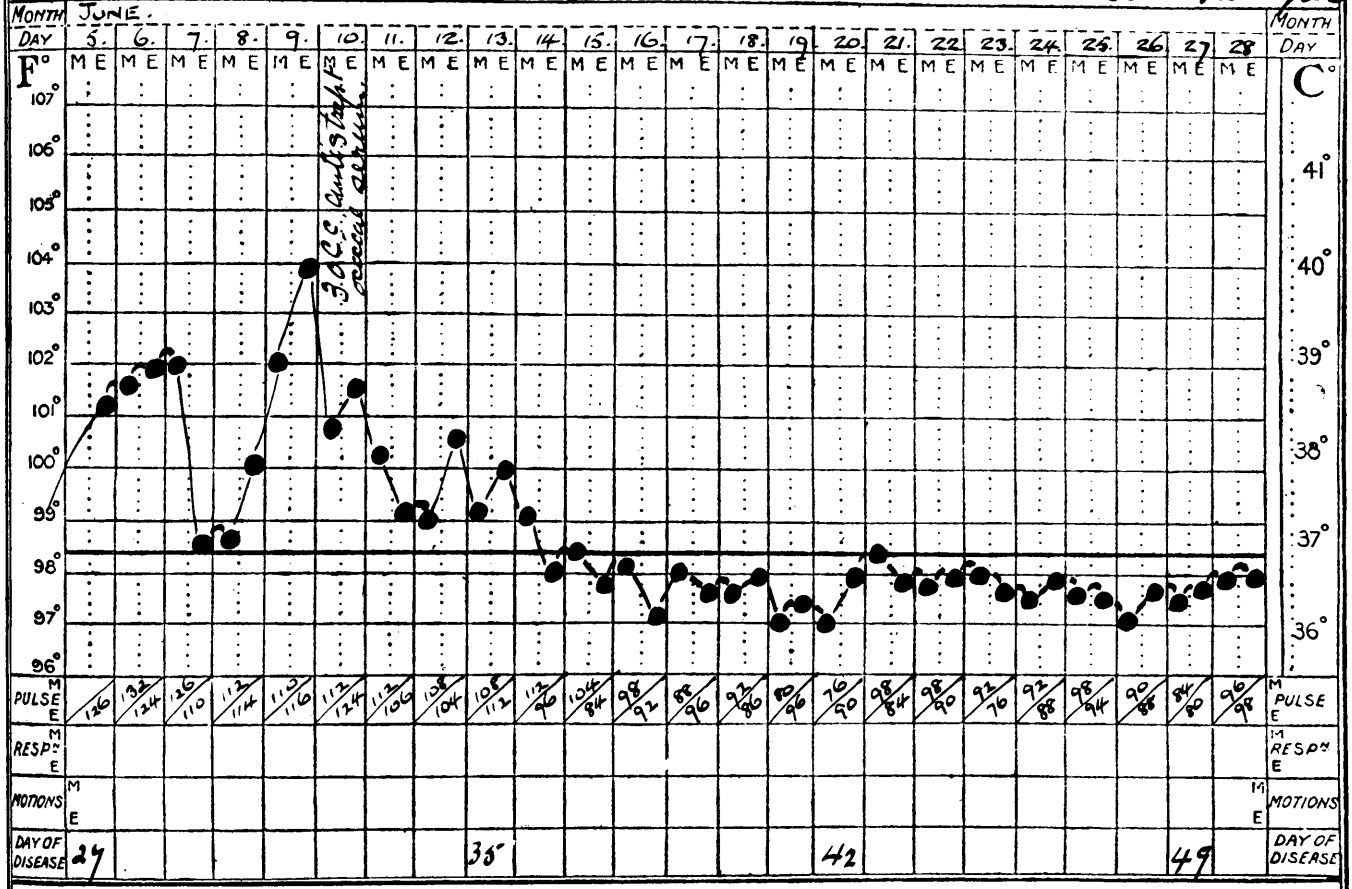
Southern HOSPITAL.

PATIENT'S NAME M. K. AGE 8.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. complicated by Rheumatism.
Treated with Antistreptococcal Serum and Sod. Salicylate.



URINE.

DATE																								
No of 03. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
" Spasmodic																								
" Whoop																								

London County Council.

Form 305.

(12).

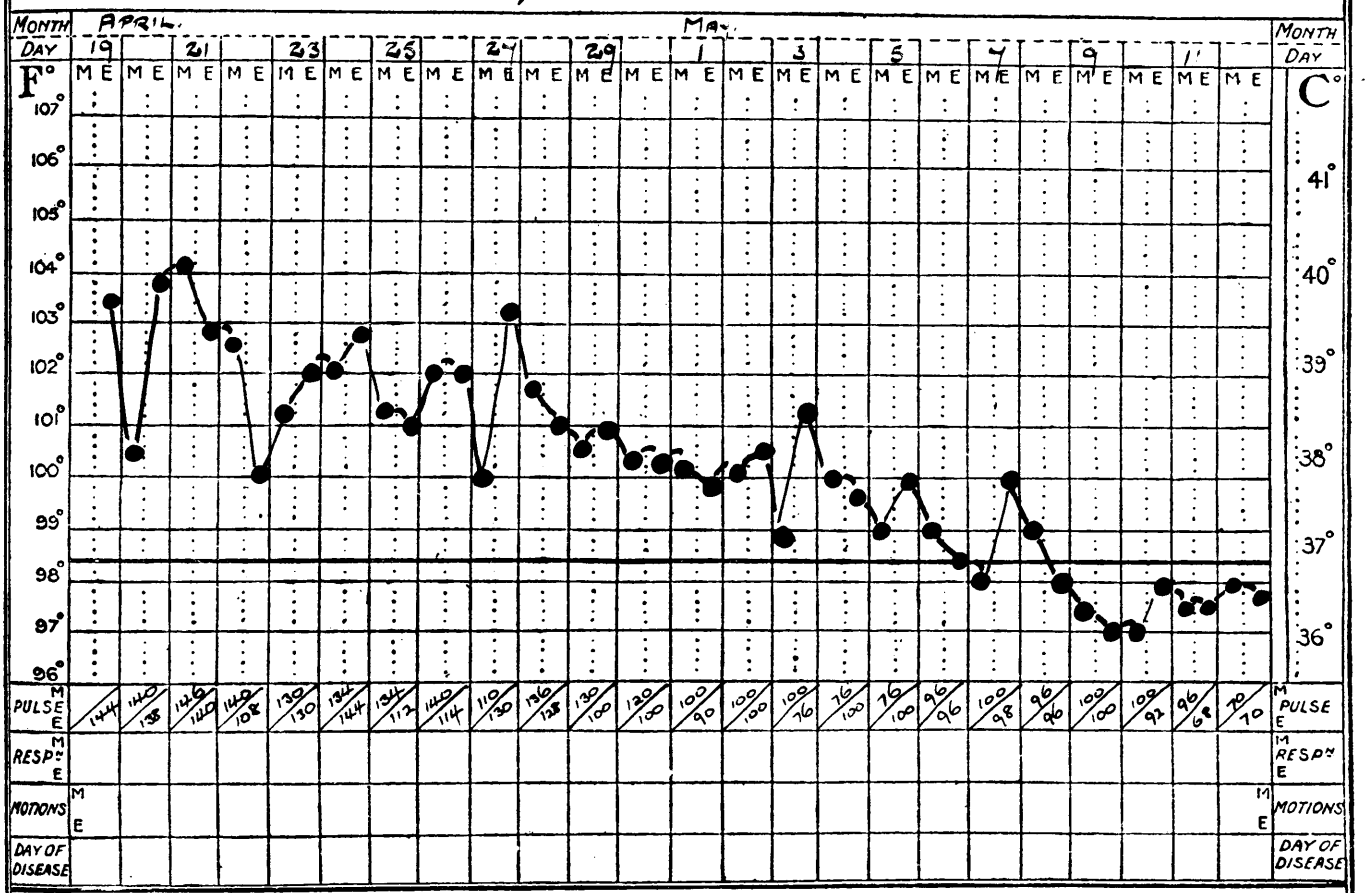
Southern HOSPITAL.

PATIENT'S NAME D. Y. AGE 9.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. Complicated by rheumatism and pericarditis?



URINE.

DATE																									
No of Cs. in 24 hrs																									
REACTION																									
SP. GR.																									
ALBUMEN																									
BLOOD																									
UREA																									
Microscop' Appearance Etc.																									
COUGH Simple																									
Spasmodic																									
Whoop																									

London County Council.

Form 305.

(13)

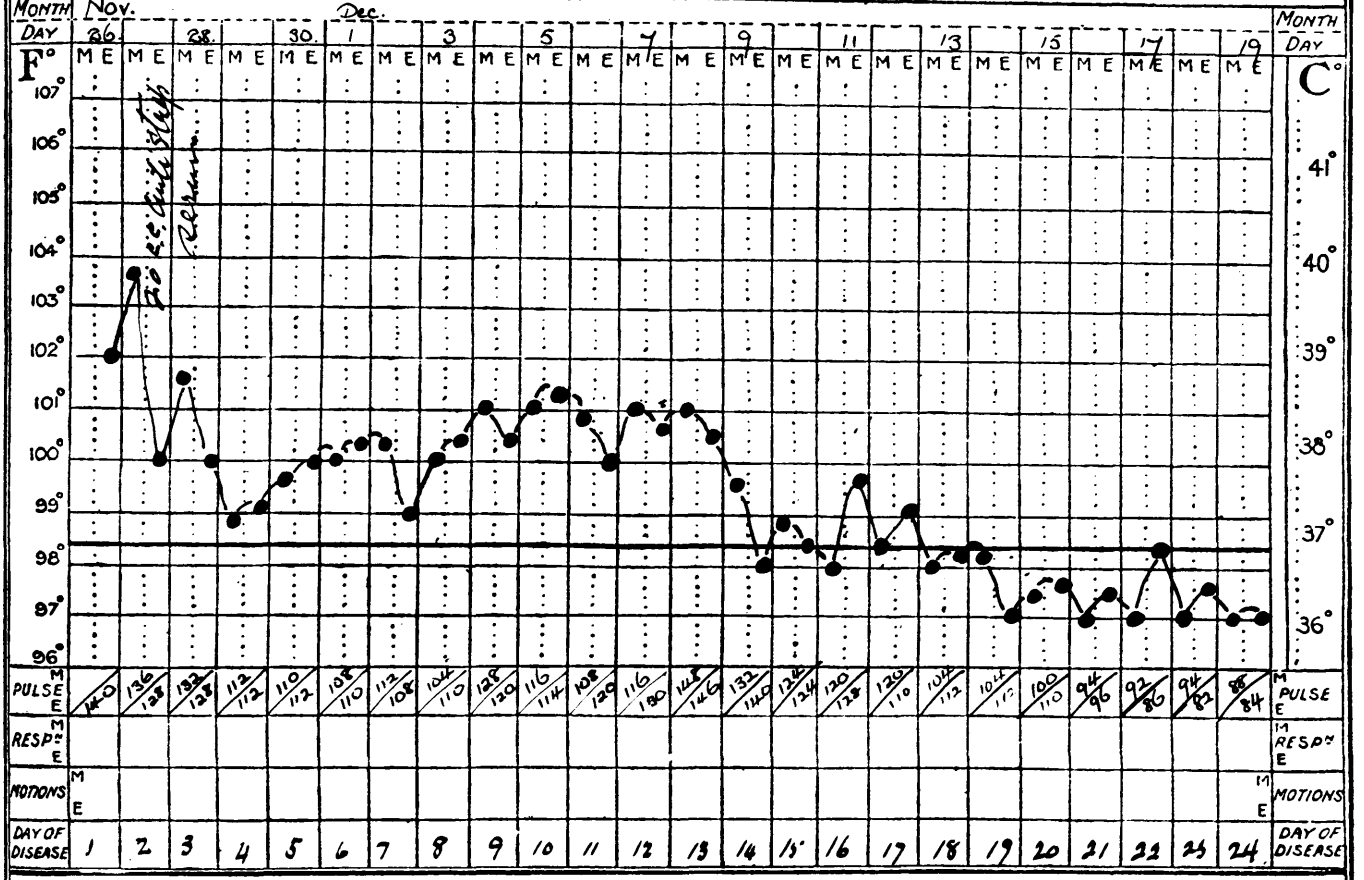
..... HOSPITAL.

PATIENT'S NAME G. S. AGE 7.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. Complicated by acute rheumatism and endocarditis. Treated with serum.



URINE.

Date																								
No of Os. in 24 hrs																								
Reaction																								
Sp. Gr.																								
Albumen																								
Blood																								
Urea																								
Microscop' Appearance Etc.																								
Cough Simple																								
" Spasmodic																								
" Whoop																								

London County Council.

Form 305.

(14.)

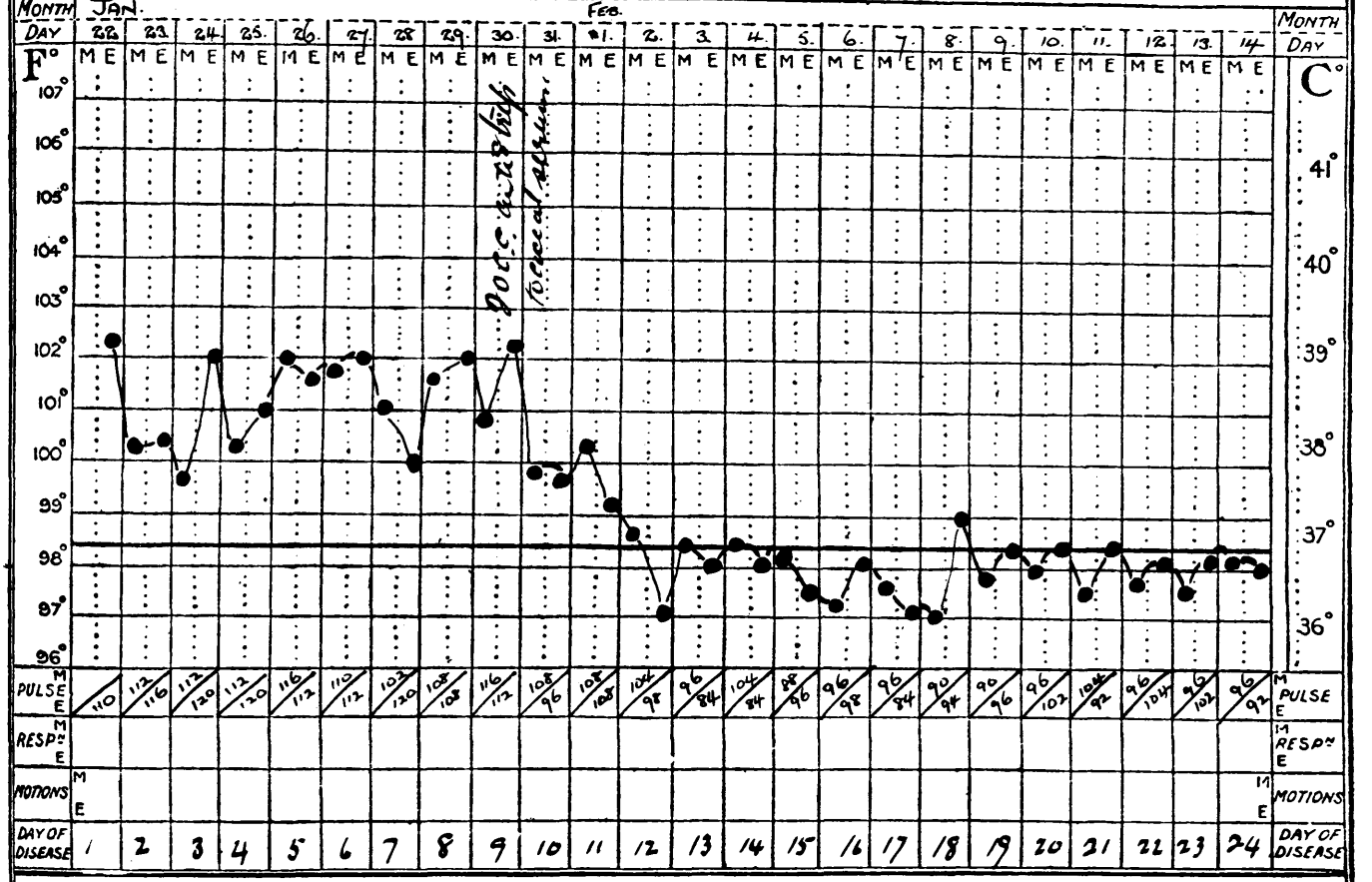
Southern HOSPITAL.

PATIENT'S NAME W. O. AGE 8.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. complicated by relative incompetence of mitral valve. treated with anti streptococcal serum.



URINE.

Date																								
No of ozs in 24 hrs																								
Reaction																								
Sp. Gr.																								
Albumen																								
Blood																								
Urea																								
Microscop' Appearance Etc.																								
Cough Simple																								
Spasmodic																								
Whoop																								

London County Council.

Form 305.

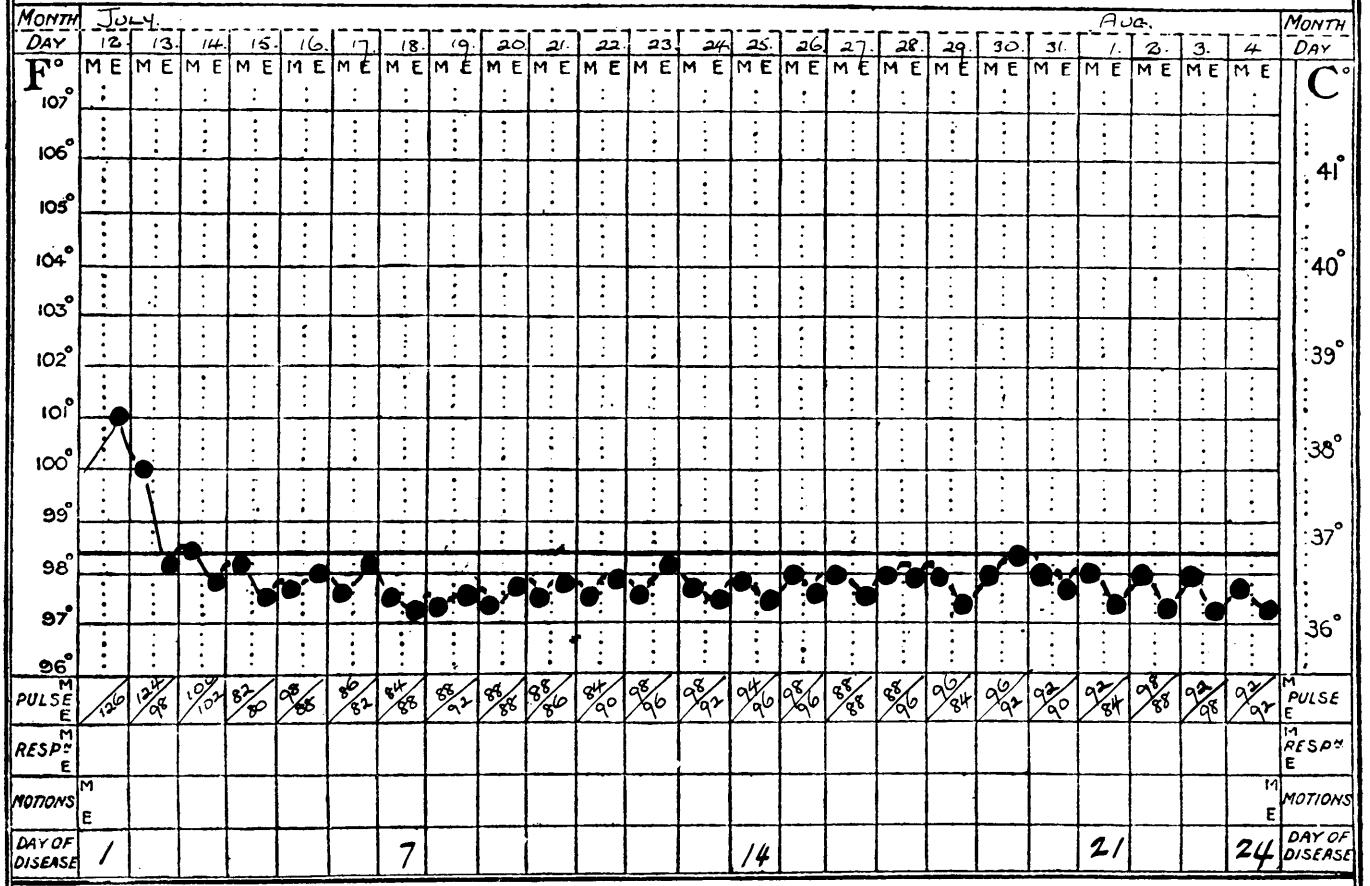
(15.) Southern HOSPITAL.

PATIENT'S NAME P. de Y. AGE 8.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. Complication Relapse Scarlet Fever. 24th Aug 1930.



URINE.

DATE																							
No of oz. in 24 hrs																							
REACTION																							
SP. GR.																							
ALBUMEN																							
BLOOD																							
UREA																							
Microscop' Appearance Etc.																							
COUGH Simple																							
Spasmodic																							
Whoop																							

London County Council.

Form 305.

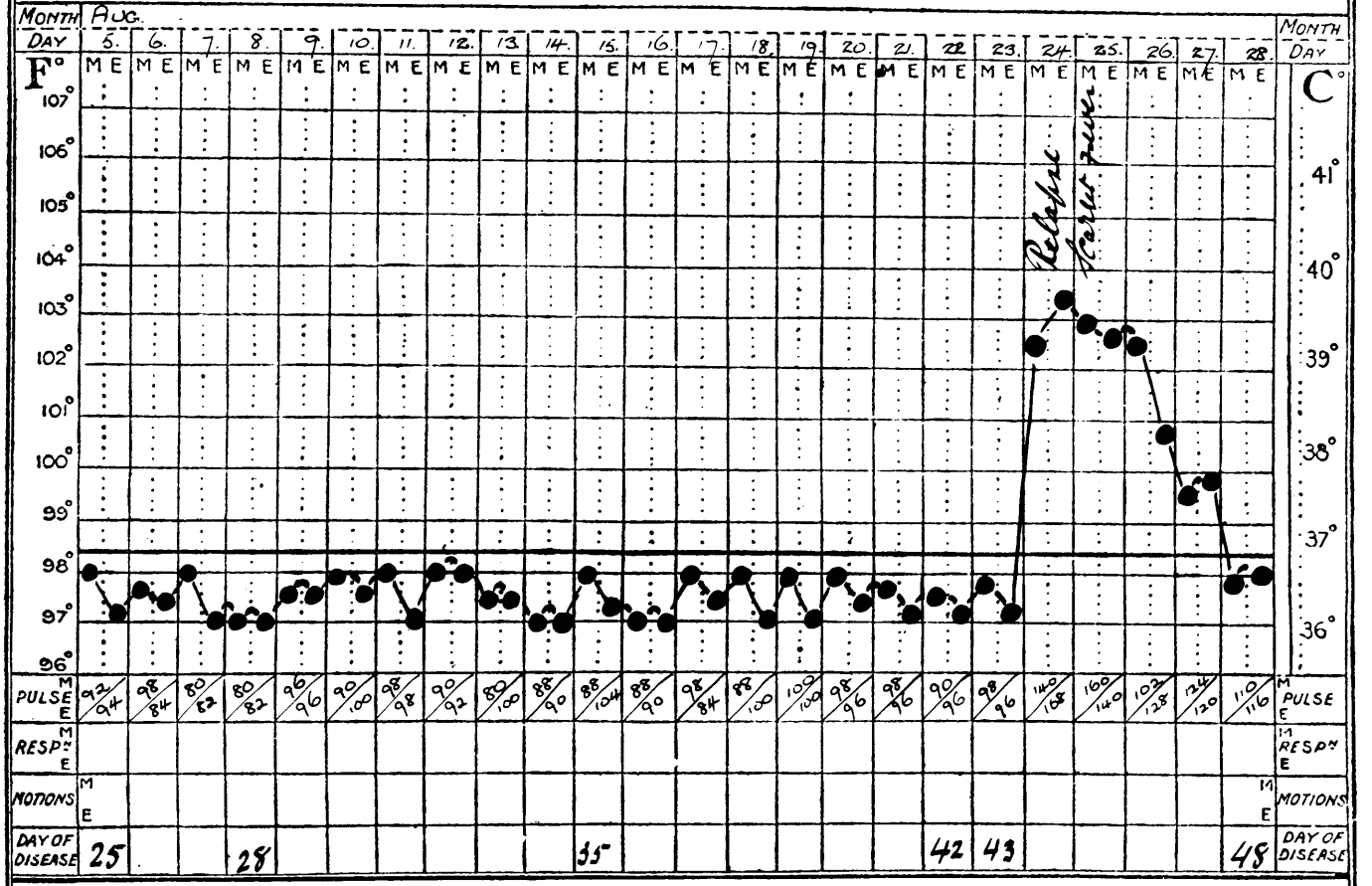
(15.) Southern HOSPITAL.

PATIENT'S NAME P. de F. AGE 8.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS.



URINE.

DATE																								
No of oz. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
" Spasmodic																								
" Whoop																								

London County Council.

Form 305.

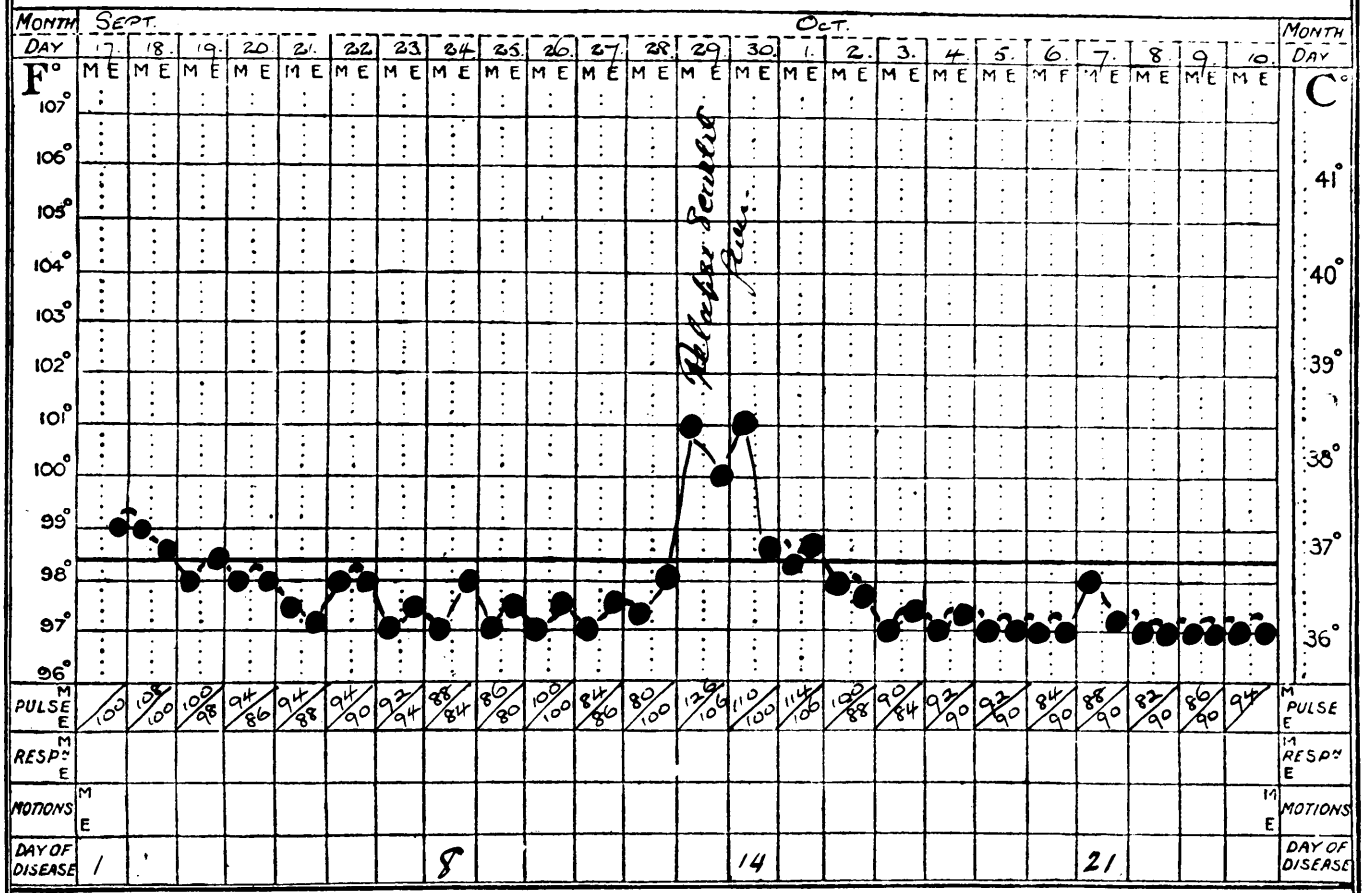
(14) (16). Southern HOSPITAL.

PATIENT'S NAME E. M AGE 9

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Complications Relapse Scarlet Fever.



URINE.

DATE																								
N° of oz. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
Spasmodic																								
Whoop																								

London County Council.

Form 305.

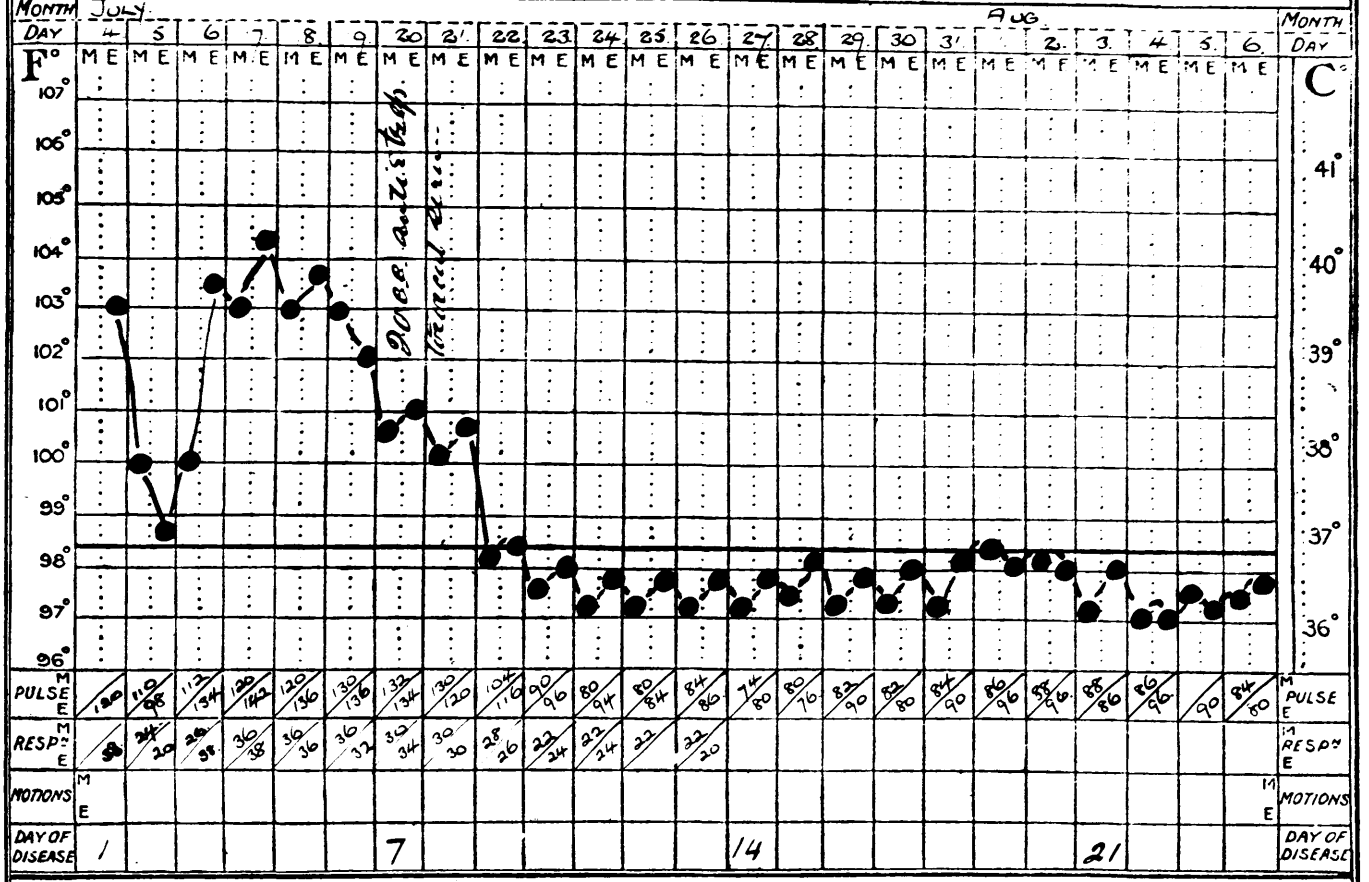
(17). HOSPITAL.

PATIENT'S NAME G. G. AGE 4.

CLINICAL CHART.

100,000—(H. 18485 24A) 18.7.30

DIAGNOSIS. Scarlet fever – Complicated Pneumonia treated with anti-streptococcal serum.



URINE.

DATE																								
No of cs. in 24 hrs																								
REACTION																								
SP. GR.																								
ALBUMEN																								
BLOOD																								
UREA																								
Microscop' Appearance Etc.																								
COUGH Simple																								
" Spasmodic																								
" Whoop																								

London County Council.

Form 305.

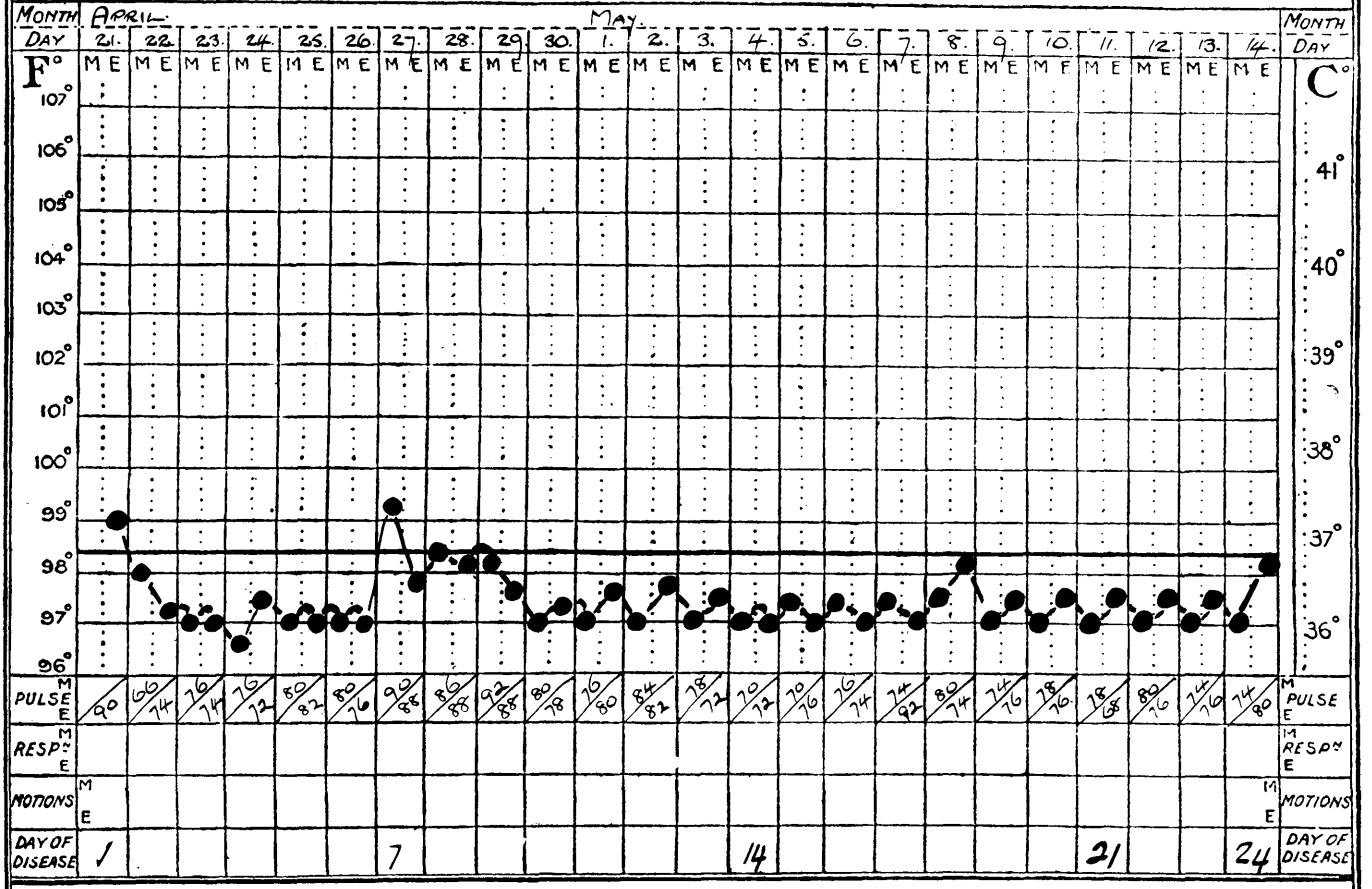
18. Sutton HOSPITAL.

PATIENT'S NAME L. Y. AGE 7.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. complicated by colitis
Treated with antistreptococcal serum



URINE.

DATE																									
No. of oz. in 24 hrs																									
REACTION																									
SP. GR.																									
ALBUMEN																									
BLOOD																									
UREA																									
Microscop' Appearance Etc.																									
COUGH Simple																									
" Spasmodic																									
" Whoop																									

London County Council.

Form 305.

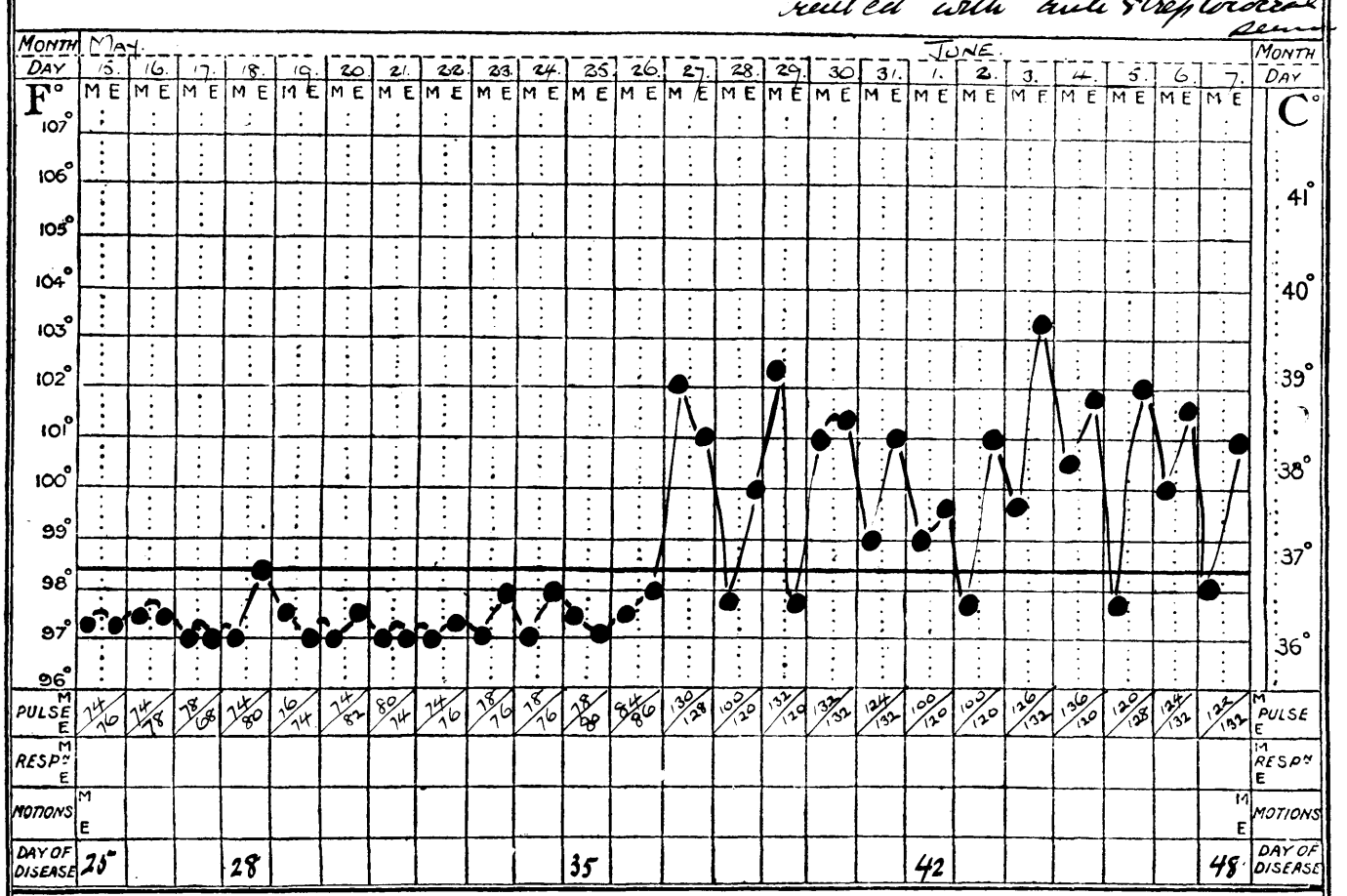
(18) Southern HOSPITAL.

PATIENT'S NAME L. Y. AGE 7.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. complicated by colitis
Treated with anti-streptococcal serum



URINE.

DATE																									
No of o3. in 24 hrs																									
REACTION																									
SP. GR.																									
ALBUMEN																									
BLOOD																									
UREA																									
Microscop' Appearance Etc.																									
COUGH Simple																									
" Spasmodic																									
" Whoop																									

London County Council.

Form 305.

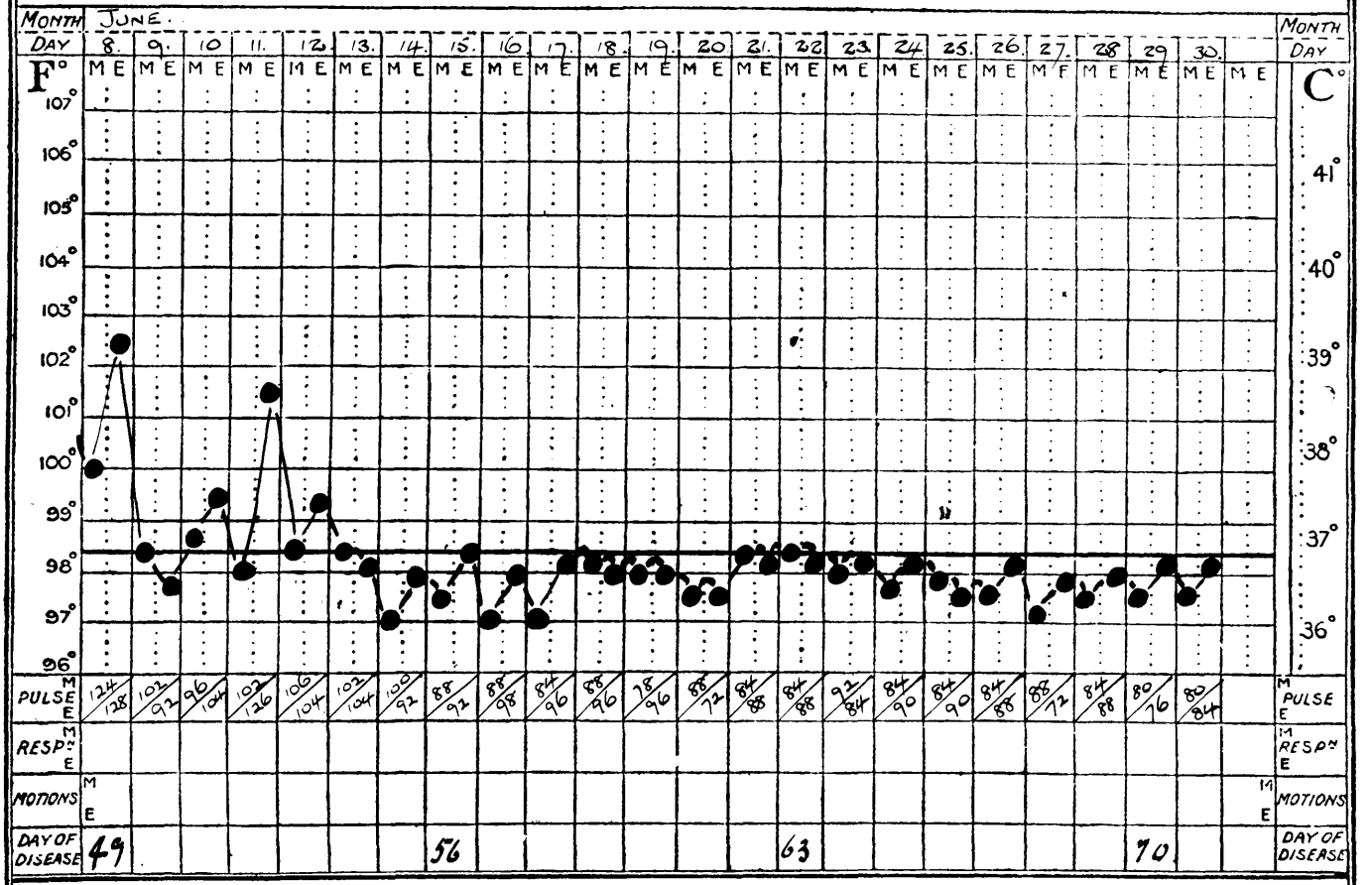
(18.) Southern HOSPITAL.

PATIENT'S NAME L. F. AGE 7.

CLINICAL CHART.

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet Fever. Complicated by Colitis. Treated with Anti-Streptococcal Serum.



URINE.

DATE																							
No of 03. in 24 hrs																							
REACTION																							
SP. GR.																							
ALBUMEN																							
BLOOD																							
UREA																							
Microscop' Appearance Etc.																							
COUGH Simple																							
Spasmodic																							
Whoop																							

London County Council.

Form 305.

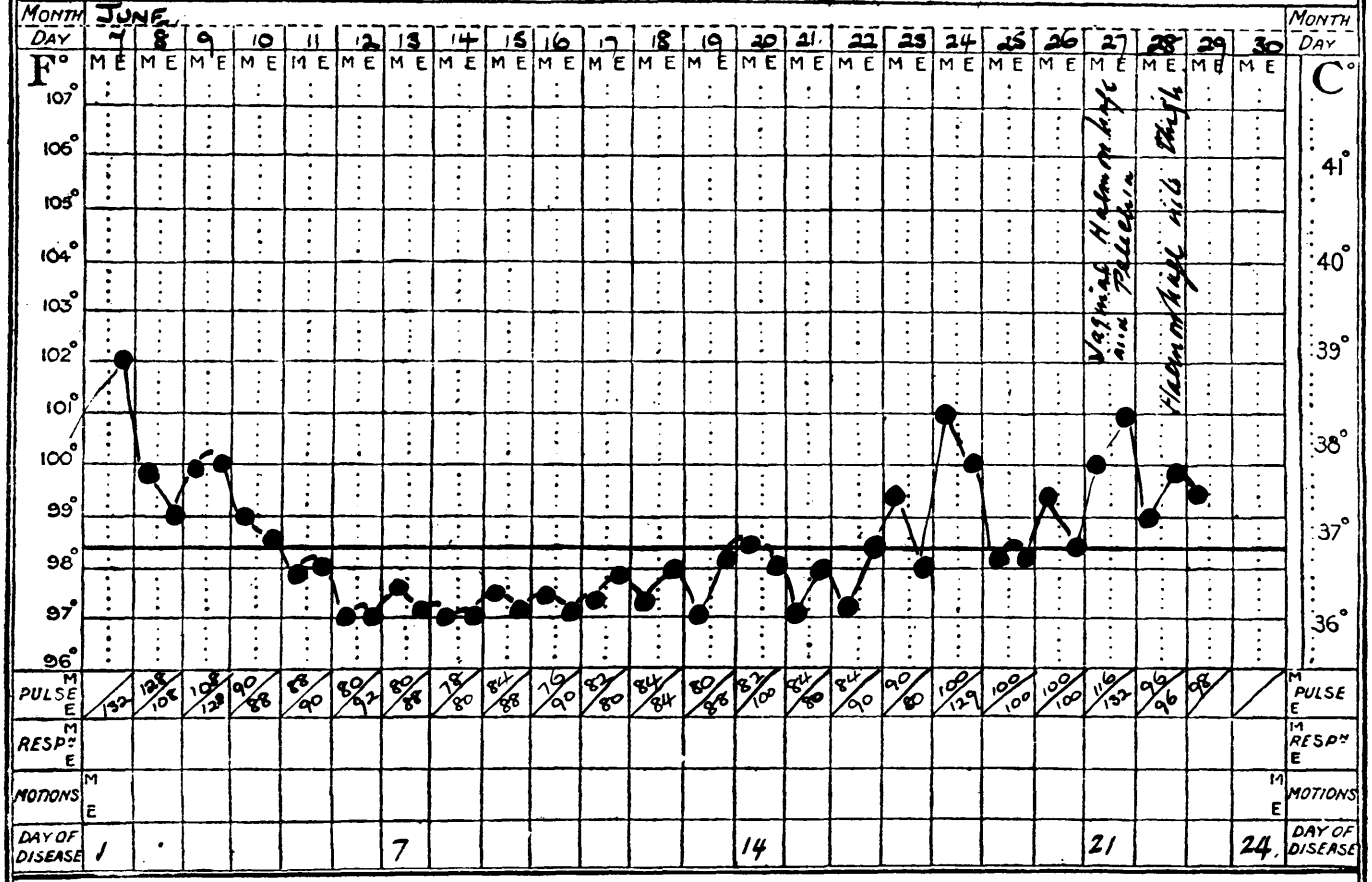
(19.) Southern HOSPITAL.

PATIENT'S NAME J. B. AGE 8

CLINICAL CHART

100,000—(H. 18485/24A) 18.7.30

DIAGNOSIS. Scarlet fever. Complication Purpura fulminans. Treated with serum. Fatal termination.



URINE.

DATE																									
N° of oz. in 24 hrs																									
REACTION																									
SP. GR.																									
ALBUMEN																									
BLOOD																									
UREA																									
Microscop' Appearance Etc.																									
COUGH Simple																									
" Spasmodic																									
" Whoop																									