THE CLINICAL ASPECTS

of

MUSTARD GAS POISONING.

by

J.M.Mackintosh M.A.,M.B.,Ch.B.,D.P.H.

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ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
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PREFACE.

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The Clinical Study presented in this Thesis is the result of observations extending over a period of six months, and of a much more prolonged analysis of records. The opportunities were exceptional, as the Hospital to which I was attached was early recognised as a special "Gas-Centre". The conditions of study, on the other hand, were particularly unfavourable for systematic research.

The investigations on which this study is founded proceeded on three lines: statistical, pathological, and clinical.

For the purposes of the first investigation I personally examined more than fifteen hundred cases of Gas Poisoning, and analysed the records of fully two thousand. From this preliminary analysis I selected for further study the fifteen hundred cases in which the causal agent was proved to be Mustard Gas (Dichlorethyl-sulphide.)

The microscopic pathology of fatal cases in this series was studied by Dr.W.W.Ingram, the Pathologist to the Hospital; and, in addition, valuable assistance in the interpretation of Morbid changes was kindly given by Dr./

Dr. Shaw Dunn, to whom many of the sections were sub-:mitted. Pathological histology, however, is not within the scope of this study. Reference is made to the findings of these observers, and also to the work of Warthin and Weller, only for the necessary elucidation of clinical problems. I have confined myself, as far as possible, to the results of my own observations: the records of gross pathological changes are derived entirely from my own notes taken at autopsy. In supplement to the somewhat meagre records of clinical pathology which it was possible for me to take under the stress of war conditions, I have to acknowledge my indebtedness to the work of Dr. Matthew J. Stewart, Dr. G.R. Herriman, and Dr. E.B. Krumbhaar.

The main body of these observations, however, was conducted at the bedside. During the whole period under consideration I was in personal charge of wards which received all severe cases of Mustard Gas Poison-:ing, and the case records and analyses are the result of my own work. The inspiration, the guidance, and the vital force necessary to carry on this work were largely provided by Dr. C.M. Wilson, the Officer in charge of the Medical Division. His wide experience and clinical ability/

ability were of inestimable value to me at all times; and his faculty for logical reasoning and deduction saved me from many a false step.

In January 1920, Dr. Wilson and I published some of our earlier results in the Quarterly Journal of Medicine. This paper, however, dealt chiefly with the Military aspect of the subject, and was intended to be of assistance in the assessment of pensions. At the time of publication much of my later material had not been arranged or studied, so that the article in question bears little resemblance to the present account, either in form, or in interpretation of results.

A great deal of experimental and chemical research has been accomplished on the subject of Mustard Gas. In this account hardly any reference has been made to these two branches of study, as they have not an immediate bearing on my subject. I have endeavoured to transgress as little as possible into criticism of work already complete; my object has been constructive rather than critical - to establish a closer connection between the laboratory and the clinical work.

The discussion of Functional phenomena is the outcome of much general reading and thought on a subject which has been to me of intimate personal interest; but I owe a special debt to the writings of McDougall and Rivers. I have/

have dealt, perhaps a little more fully than a strict-:ly clinical study would warrant, with the psycho-path-:ology of war-neurosis in general; yet I feel that secure foundations must be laid, because the psycholo-:gical terms and definitions in current use are liable to such variety of interpretation. Moreover, a clinical survey is in a sense a human document; it must consider the mind as well as the body, the patient as well as the process.

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Note on Arrangement of Sections.

As Mustard Gas poisoning is a complex in which a variety of distinct lesions ~~are~~ *is* presented by one patient, I have thought it preferable to place the greater part of my illustrative material in an appendix. This has been done for convenience of reference, and in order to avoid undue repetition in the text. The case numbers quoted in the text, therefore, refer to the numbered illustrations in the Appendix.

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S E C T I O N I.

INTRODUCTORY.

INTRODUCTION.

Dichlorethylsulphide, the active agent in Mustard Gas, was first made by Victor Meyer in 1886. Meyer described the toxic properties of the liquid and its vapour, and concluded that the most severe action develops only after entrance to the blood. Little attention was paid to this discovery until the recent war, when the search for ruthless methods of destruction led German Scientists to its employment on a large scale. Since that time (1917) an enormous amount of experimental work has been done on the chemical and toxic properties of the substance. The results of these researches cannot be detailed here, but a knowledge of the peculiar characteristics which make this substance so potent a factor in the production of casualties is essential to a clear understanding of the significance of the lesions in the human body.

Mustard Gas[1] - I shall use the familiar term throughout - "is a solution of dichlorethylsulphide in carbon tetrachloride in the proportion of about 8 to 2. The latter is a useful solvent, and aids in producing a wide distribution of the toxic fluid, when the explosion of the gas-shell takes place. The combination is colourless, but becomes yellow on exposure to light.

It has a rather pungent odour, from which the common term is derived, resembling oil of mustard. It is an oily liquid which evaporates slowly, clings to garments, and - like petrol, for instance - penetrates rapidly through cloth and even leather." Two characteristics differentiate sharply its mode of action from that of the other gases of warfare: its insidiousness and the persistence of its toxic properties after dispersion. By 'insidiousness' I mean that its effects are not produced immediately on exposure, but only after a more or less prolonged latent period; thus a man may remain unwittingly even in the presence of high concentrations. Its properties of persistence are even more dangerous, for clothing, equipment, huts, dugouts, and even large tracts of ground may retain contamination for several days.

"I was staying in a dug-out near Brigade Headquarters ... One night some men came in and said they had been through a gas-shell bombardment during the day . . . I noticed a queer smell like garlic off their clothes" - this man came under my care suffering from severe Bronchopneumonia following exposure to Mustard Gas. Again,

"I was sent up with a working-party early in the morning; after the sun was up, I felt a 'nippy smell' (He was a Scotsman). There was no shelling at the time . . . ."

This/

This N.C.O. was admitted suffering from Bronchitis and severe burns.

These two examples serve to illustrate the fact that the tremendous casualty-producing power of Mustard Gas lies in its remote rather than immediate action; for during a bombardment the troops are aware of their danger and take the necessary precautions. In many cases, indeed, exposure takes place quite unperceived by the subject, and the onset of erythema or eye irritation is the first indication to the patient that he has been exposed to Gas.

For the purposes of clinical description the lesions produced by Mustard Gas fall naturally into three main divisions: ocular, cutaneous, and respiratory. In subsequent sections these divisions are dealt with separately. It is only in exceptional cases, however, that the gassed patient presents an isolated group of lesions - such as Burns without any involvement of the eyes or the chest. In the great majority of cases all three divisions are implicated, and react upon one another; they cannot be entirely separated; they build up the picture of a single disease - Mustard Gas Poisoning.

I shall introduce the subject, therefore, by considering the condition as a clinical unity, using the classification/

classification of degree rather than type:

The Mild Case presents the following general characters: There is a somewhat prolonged latent period after exposure (12-48 hours) and the initial symptoms are relatively slight. The patient first complains of irritation and watering of the eyes and a sensation of heat in the skin; later there is dryness of the throat and cough; there is neither shock nor vomiting. By the end of 24 hours the lesions have reached their maximum - erythema of the skin with perhaps slight necrosis in the genital region; marked injection and watering of the eyes; and a red, dry, glistening appearance of the throat. There is usually a thin mucous discharge from the nose.

By the end of 72 hours the prognosis can be established with certainty from examination of the skin and of the temperature chart [redacted]. In 70 per cent of cases of this type convalescence is completely established in a week. In the remainder retention in hospital beyond this period is due either to the slow healing of somewhat deeper burns in the genital region or to the development of functional conditions such as photophobia, aphonia, or cardiac irritability.

The Severe Case: The great majority of the more serious cases of Mustard Gas Poisoning present, to a marked/

marked degree, lesions of eyes, skin and respiratory tract. Five cases, of which two were fatal, developed severe skin lesions without any involvement of eyes or chest; and twenty, of which four were fatal, had severe respiratory lesions with only a trifling erythema of the skin. The worst eye lesions were associated with Bronchopneumonia rather than Burns, because of the protection of the Respirator in the latter cases.

With the involvement of two great systems - cutaneous and respiratory - it is a matter of no little difficulty to determine the part played by each in the production of the typical disease. Considered apart, the temperature chart of a case of Burns bears little resemblance to one of Bronchopneumonia, but in the combination of the two the 'respiratory' influence predominates; the hurried respirations, cough, and cyanosis fill the picture; Burns are probably represented only by a pulse unusually high for pneumonia. The accompanying chart, taken from actual cases, shows the characteristic types of pyrexia in cases of (a) uncomplicated Burns (b) uncomplicated pneumonia and (c) a combination of pneumonia with extensive burns. This chart shows well the comparatively slow evolution of skin lesions. The following is the history of a fairly typical "combined" case:

Three or four hours after exposure the patient becomes suddenly/

DISEASE.

Notes of Case.

Name

Age

Diet

Case Book N°

Date of admission.

Result

Time.	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
Bowels. *Day of Dis.*																					
	2	3	4	5	6	7	8	9	10	11	12	13	14	15							

Temperature (Fahrenheit)

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

Normal Temperat. of body.

(a) A case of uncomplicated burns —●—

(b) A case of uncomplicated bronchopneumonia —●—

(c) A case of Pneumonia with extensive Burns ---o---

Day of Dis.

Pulse.

Resp.

Date.

42° 41° 40° 39° 38° 37° 36° 35°

Temperature. (Centigrade)

Entered at Stationers Hall

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Gould's Clinical Chart

suddenly aware of a sensation of discomfort and heat in the skin, quickly followed by smarting of the eyes and dryness of the throat. As this initial erythema of the skin develops there is a considerable degree of shock, with giddiness, nausea, and vomiting. By the end of 24 hours the initial shock passes off, and the characteristic features of severe Mustard Gas poisoning are developing rapidly. The patient lies on his back, breathing noisily. There is considerable nasal discharge and obstruction, and the mouth is held open. The cough is short, hoarse and painful, and sputum is expelled with difficulty. Respirations, pulse and temperature are moderately raised. The eyes are almost closed by the red and swollen lids, and there is a purulent discharge from the inner canthus. The face shows a diffuse erythema with perhaps minute excoriations around the mouth and nose. Over the body generally the reddening is irregular, and close inspection reveals the presence of minute vesicles around the hair follicles of the genitals and axillae.

During the second day crops of vesicles may appear on the erythematous areas, and the genital regions show a deeper necrosis, with oedema of the scrotum. The respirations are not greatly above normal, and the pulse shows a falling rate with improvement in strength and volume/

volume.

From the beginning of the third day the development of pulmonary infection engages the physician's attention. Respirations and temperature rise rapidly, and some degree of cyanosis always appears.

No fresh developments occur until about the sixth day (4th to 7th day) when there is commonly a striking exacerbation of the symptoms. This is due, I think, largely to the fact that tissue necrosis has reached its height, and separation of sloughs with consequent septic absorption has begun. In grave cases death often takes place about this time. In favourable cases the acute stage of Bronchopneumonia lasts till about the 12th day (11th to 14th day) when definite improvement sets in - this I take to be the onset of resolution. The temperature rarely settles completely, however; much more frequently there is a "smoulder" of a week or so caused by septic absorption from denuded areas of skin and bronchial wall. In more chronic cases a prolonged, swinging temperature of the septic type indicates the presence of a necrotic focus. The cutaneous lesions rarely give trouble after the 12th day.

The Fulminating Case is very rare; its distinguishing characteristic is that death takes place before the development of any serious degree of secondary infection. In this/

this series five cases occurred which might be described as fulminating; they had many features in common.

There is a short latent period (1 - 4 hours), followed by initial symptoms of extraordinary severity. Nausea, vomiting and a severe degree of shock mark the onset; the subjective sensations are intense heat and burning of the skin, acute pain in the eyes, and a terrible feeling of oppression and constriction in the chest. Vesicles appear on the skin within a few hours; and deep cyanosis, diminished expansion of the chest, with rapid, shallow and heaving respirations point to intense damage to the respiratory tract. The heart beat is feeble and rapid, and in two cases acute dilatation was noted. The physical signs are those of oedema of the lungs, but the typical sputum appearances are obscured by purulent secretions from the nasopharynx. Toxaemia is entirely absent: all my cases were rational and clear-headed until within three hours of death. Cyanosis and dyspnoea increase rapidly and the right heart becomes en-:gorged with blood. Death takes place before any recog-:nisable infection, even of the skin, has had time to develop.

I shall not refer again in detail to the fulminating case. The cause of death is a matter of uncertainty, but certainly secondary infection plays a minor part. I con-:clude/

conclude that the principal factors involved are shock, pulmonary oedema, and the reaction of the pulmonary tissues to the irritant.

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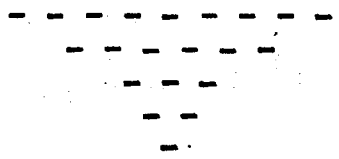
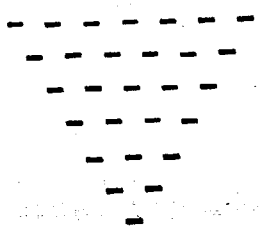
Notes and References - Section I.

(1) Medical Research Committee (No. 18.) Report on the Medical Aspect of the production of Mustard Gas.



SECTION II.

OCULAR LESIONS.



OCULAR LESIONS.

Incidence: The eyes were affected in 1119 (75 per cent.) of my cases. Of these 352 (23.5 per cent.) presented a severe conjunctivitis with lacrimation, photophobia, and pur-:ulent discharge. There were only 30 examples of definite corneal injury, demonstrable by fluorescin. Blepharitis was extremely common, and a very intractable form of ulceration of the lid margins was a feature of the severer cases. No cases in this series developed hypopyon, or signs of injury to the deeper structures of the eye. (1)

Clinical Appearances: In all cases the eye symptoms develop rapidly within 12 hours of gassing. The patient first complains of a sensation of heat and of the presence of a foreign body; actual pain is not common. Later there is a more or less profuse lacrimation and some degree of photophobia. In mild cases the appearances at the end of 24 hours are erythema and puffiness of the lids; the bulbar conjunctiva is markedly hyperaemic over an area corresponding with the palpebral fissure, but elsewhere is not much affected. The condition is one of congestion rather than inflammation, as there is no abnormal discharge from the eye. The pupil reflexes are normal and vision is unimpaired. During the second day the lids became still more puffy, giving the patient a drowsy appearance. The

lid/

lid margins show a bright red rim, contrasting with the dusky colour of the lids. This condition yields readily to treatment, and no true conjunctivitis super-:venes. In a small proportion of cases, however, lacrimation and photophobia persist after all physical signs have disappeared; the condition becomes one of 'functional photophobia'.

In the more severe cases the picture of a simple congestion is quickly obscured by the appearance of a sero-purulent discharge. On the second morning the lids are found to be firmly sealed by yellow secretion; there is considerable oedema, and examination and cleans-:ing of the eye are difficult, and painful to the patient. Blepharospasm is a marked feature and lacrimation is profuse and distressing. Conjunctivitis of all degrees of severity is found. In the lighter cases there is intense congestion in the palpebral fissure, and dilated and tortuous vessels are seen running to the corneal margin. The inner edges of the lids show minute shallow excoriations which become covered with crusts. Towards the more severe end of the series there is a very striking chemosis, in some cases completely concealing the pupil. Yet even in the most acute degrees, when the greatest difficulty is encountered in washing out the secretion, the corneal surface,

exposed/

exposed in the depths of this bulging, oedematous mass, is clear, brilliant and entirely uninjured. The pupil reacts normally to light and accommodation, and the visual acuity, if one takes into consideration the lacrimation and the oedema, is remarkedly little reduced.

The above description applies to all but the very worst cases, within 48 hours of onset. If careful and systematic cleansing is adopted, the outlook is very favourable. In the great majority of cases each day shows a steadfast improvement until at the end of 10-14 days little remains beyond blepharitis and some morning secretion between the lids. Lacrimation and photophobia are apt to be troublesome for some weeks, and in a number of cases a chronic eczematous condition of the eyelids persists.

In 20 of my cases, due partly to the severity of the initial lesion, and partly, I fear, to lack of sufficient vigour in the early treatment, true ulceration of the cornea developed. In most cases the necrosis was superficial, taking the form of minute punctate dots which gave the injured surface a mottled, steamy appearance. These cases improved rapidly under atropine, and regeneration took place without permanent impairment of vision. Three cases, however, developed fairly deep well-defined ulcers - one towards the periphery in both eyes, and two centrally in one eye only.

Vascularization/

Vascularization of the cornea took place, and a fibrous scar resulted from the healing process, leading to considerable reduction of visual acuity.

In a small proportion of cases direct contact with the liquid or high vapour concentration gives rise to early and severe burning of the cornea. This type, first demonstrated to me by Col. Elliott, [2] contrasts strongly with the types above described.

At the end of 24 hours the caustic effect of the vapour is already evident. Across the palpebral aperture, on both sides of the cornea there stretches a dead white raised band of solid oedema. This, as Col. Elliott points out, compresses the vessels, impairs the circulation, and acts as a menace to the nutrition of the cornea. The exposed portion of the cornea - usually a well-defined band situated more or less centrally - is grey and hazy and shows a blurred 'window-reflex'. As a result of irritation and congestion the pupil is contracted. Above and below the band of oedema the protected portions of bulbar conjunctiva show swelling and intense congestion. Secondary infection is very liable to take place, and deep ulceration of the cornea may develop. In favourable cases the vascular injection of the upper and lower portions slowly passes off, while centrally the solid oedema is gradually absorbed. Finally the corneal epithelium regains its normal lustre,

and/

and in the palpebral fissure oedema gives place to hyperaemia. Thus the final stage of this type is very similar to the early appearance in a mild case.

The process of resolution is very slow: in one of my cases the dead white band of oedema was still well marked on the 10th day; both corneae were hazy and anaesthetic, and their surfaces stained with fluorescin (case 25). In another case corneal ulceration was still active in the fifth week (case 33).

Sequelae: Apart from corneal ulceration, a number of cases do occur in which the normal condition of the eye is not re-established. There remains a chronic hyperaemia of the conjunctiva, with local thickenings and new vessel formation. The blepharitis also is apt to become chronic, and several of my patients suffered subsequently from recurrent hordeoli.

Warthin (3) states that even in mild cases serious refractive errors and reduction of vision occur. I have not been able to confirm this, even in severe cases, provided that no corneal lesion was present.

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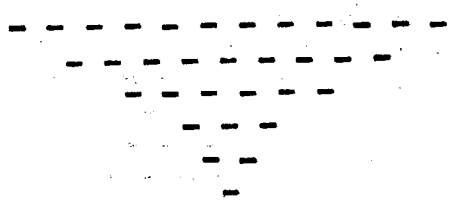
Notes and References.

(1) Several cases of Panophthalmitis following mustard gas lesions have been recorded.

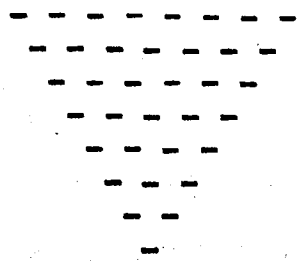
(2) Col. T.R. Elliott. D.S.O. Consulting Physician to Boulogne Base.

(3) Warthin and Weller: "Medical Aspects of Mustard Gas".



S E C T I O N III.

CUTANEOUS LESIONS.



CUTANEOUS LESIONS.

Incidence: Some degree of skin involvement was present in all my cases, the existence of at least an erythema being considered essential for a diagnosis of Mustard Gas. Excluding mild Erythema, however, the skin was implicated in 543 (36 per cent.) of my cases. Of these only 161 were classified as severe - that is, the damage to the skin gave rise to bacterial infection and pyrexia of more than a week's duration. Cutaneous lesions, uncomplicated by damage to the respiratory tract, were responsible for death in 2 cases; and in other 8 they played a large part in the production of the fatal issue.

Bodily distribution: Mustard Gas has remarkable powers of penetration, and ordinary clothes afford little protection from its attack on the underlying skin. Moreover, in the regions covered by clothing the resultant burn is liable to be more severe, on account of deficient evaporation and more prolonged contact with the contaminated material. In the third place, the gas has a special selective action on surfaces covered with sweat or sebaceous material. Consequently the genitals, the inner sides of the upper thighs, the axillae, and to a less extent the flexures, are peculiarly liable to attack. The reasons for this selective action are:- (a) that the

oily/

oily globules of the vapour tend to coalesce when directed in a fine spray upon a moist surface, and so greater concentration is secured.

(b) That the liquid,[2] thus concentrated, passes readily through the hair follicles, sebaceous and sweat glands. It is to its solubility in fatty substances that its destructive action in these areas is due.

The following table gives an account of the frequency with which the various portions of the body were affected. The predominance of lesions in the moist areas is clearly shown.

TOTAL NUMBER OF CASES 543.

Site of Lesion	Number of cases.	Per cent.
Scalp.	19	3.5
Face and neck.	121	22.3
Arms and hands.	96	17.5
Axillae.	140	25.8
Trunk - chest and back.	141	25.8
Genital Region.	318	58.5
Buttocks.	86	15.8
Thighs.	77	14.2
Legs and feet (many isolated burns)	102	18.7

Morbid Anatomy: In a severe case of Burns lesions of all degrees/

degrees from an erythema to a deep necrosis extending into the subcutaneous tissue are found. In my fatal cases the principal lesions were of the back and the genitals. The former resembled an ordinary burn of the second degree:- the entire epithelial surface was destroyed, and the denuded areas were irregularly con-:gested and bathed with thick, greyish pus; here and there gangrenous sloughs still adhered to the surface. Portions of skin less severely affected showed deep erythema, or bronze pigmentation and flaky desquamation. In 3 cases the skin of the scrotum was gangrenous and gave off a foul odour of putrefaction. The most import-:ant internal lesions were of the kidneys: all cases showed some degree of cortical congestion, and in three there was the fine stippling of acute haemorrhagic nephritis.

The microscopic pathology of cutaneous lesions has been studied exhaustively by many observers, notably Warthin and Weller. I have not had the opportunity of examining more than two or three sections, so it would be outside my province to attempt more than a brief summary of Warthin's findings: (1)

"Mustard Gas is an escharotic, specific in its action upon the epidermis and the tissues of the corium, particularly on the endothelium of the vessels. The

lesion/

lesion . . . differs from a heat burn in the absence of thrombosis, in the greater degree of fluid exudat-:ion, in the greater moistness of the affected area, and in the fact that the necrosis as shown by the loss of nuclei requires hours, even days, for its complete development. The vessels in the affected area are severely damaged and collapsed, and there is a local anaemia in the earlier stages with marked fluid exudat-:ion and leucocyte migration. It is non-haemorrhagic . . . There is no deep oedema; it is confined to the epidermis and the papillary layer of the corium in the early stages . . .

The slow healing is probably chiefly due to the vessel injury and the relatively slight leucocytic demarcating infiltration. Regeneration of the epidermis, after complete necrosis, takes place from the epithelium of the sebaceous and sweat glands."

Clinical Appearances: In the more severe cases the development of erythema of the skin generally precedes the ocular symptoms. This initial erythema is widespread, and irregular in its distribution. It is bright red in colour, but rather less brilliant than the Scarlatinal rash: it imparts, however, a very similar hot, prickly feeling to the touch. There is no sharp border limiting the affected areas, but these fade gradually into the surrounding normal skin. The subjective sensations in mild/

mild cases are not marked, but at most there is a complaint of heat rather than pain or itching. In severe cases the development of erythema is accompanied by symptoms of shock, and giddiness and vomiting are prominent, but not invariable, features.

The progress of the initial erythema varies according to the concentration of the gas and the period of exposure. Generally speaking, the deepest local lesions are due to direct contact with the liquid, while the most widespread involvement is carried by exposure to high concentrations of the vapour.

In mild cases, then, the inflammatory reaction is superficial, and the erythema gradually fades, and disappears without trace, within a week of exposure.

In cases of moderate severity the affected areas gradually become dark brown or bronzed. This phenomenon, according to Warthin, is due to pigment formation in the upper layers of the corium. The epidermis thus affected becomes indurated, giving to the fingers the sensation as though it had been roughened by frost. This dark colour-:ation persists for several weeks, until the stained cuticle desquamates. In some cases the epidermis comes away in large flakes, leaving delicate skin of normal character beneath. This is the type most commonly observed by the French and German authorities. In my cases there occurred/

occurred more frequently a fine, branny desquamation during the second week, which left behind a red, glist-:ening, slightly moist surface. This moist skin is very liable to become infected, and not infrequently falls a prey to furunculosis.

In the severest type of case the initial erythema gives place within 24-48 hours to a bluish discolouration with swelling and oedema of the epidermis. The discoloured areas are not co-extensive with the erythema; they tend to occupy the central portion of an area, and show a preference for surfaces exposed to pressure or irritation (e.g. the sacrum, buttocks, scapular region etc.) This form of lesion is similar to that which occurs in the moist parts of the body, and is commonly the precursor of necrosis. Vesicles begin to develop on these dark areas from the second day. In many of my cases they appeared in successive crops, and often showed a linear arrangement which was probably determined by scratching. In some areas necrosis develops without previous vesication. The vesicles, which may vary from the size of a pin's head to that of a large plum, are filled at first with clear serous fluid. Some become rapidly tense and liable to burst, while many remain flaccid. There is no umbilication. The contents of the non-infected vesicle have no action on either skin or conjunctiva. If the blisters are allowed to remain unpunctured, their contents often coagulate/

coagulate and so provide an excellent medium for bacterial growth. The base of the ruptured vesicle is at first pink, moist, and slightly excavated. It soon becomes dirty grey and covered with necrotic slough. These areas tend to run together and large infected tracts resembling 2nd degree burns are rapidly produced. In favourable cases necrosis is complete by about the 6th day, and separation of the sloughs begins. This is the critical period for the patient, on account of absorption from the infected surfaces. Separation is generally complete, and some progress made in regeneration, by the 21st day. In the case of the deeper lesions, where the true skin has been damaged, the healing process is much more slow, and leaves a delicate fibrous scar of low vitality.

The above is a description of the course of the disease in typical cases, but various modifications occur. Brown or bluish discolouration and even vesication may arise without previous erythema, in which case the course of the disease is more acute. Again, during the acute stage of necrosis, large tracts of damaged skin may be removed en masse by simple friction; or (as in case 36), a delirious patient may greatly aggravate his condition by tearing off bandages.

In certain areas, as already mentioned, there are important variations in degree. The genital regions suffer very severely, especially the head of the penis and the pendent parts/

parts of the scrotum. In a well-marked case there is tense oedema of the scrotum by the end of the second day. Close inspection reveals minute vesicles situate at the mouth of hair follicles. On the 3rd day these have already broken down, giving place to small, shallow ulcers which rapidly coalesce and become infected. Pain is not severe if the parts are well-supported and kept at rest, but the slightest manipulation is painful on account of the great tension, and the liability to fissures. Gangrene of these tissues is very prone to supervene. As regards the penis, the lesion is seen at its worst in cases where there is a short prepuce not completely covering the meatus. A long prepuce affords a certain amount of protection. In the circumcised the glans suffers severely, but good results are obtained on account of the free drainage. In the first case a severe purulent balanitis is the almost invariable result. Examination at the end of 48-72 hours shows a greatly inflamed and oedematous prepuce which can be drawn back only with great difficulty, and agonizing pain to the patient. Underneathy the site of the original projection of the prepuce one finds an annular ulcer on the glans. The meatus itself is commonly involved, and ulceration takes place on its inner aspect, causing great pain and difficulty in micturition. Three of my cases required catheterization under cocaine during the acute stage. The subsequent

ulcerative/

ulcerative process is very severe, resulting in some cases in deep necrosis with consequent cicatrization. Three of my cases required circumcision on this account.

The axillae suffer in a similar fashion but to a less extent. Linear cicatrices are not uncommon, and in some cases are found in all the flexures. But permanent deformity is very rare.

In the scalp Mustard Gas Burns give rise to a very intractable condition resembling seborrhoeic eczema. Large numbers of thick moist scales collect, and on removal leave a raw irritated surface. The growth of hair does not seem to be affected.

The Symptoms present few special features:

Shock is a dramatic manifestation of the onset of severe Burns. The development of the initial erythema is quickly followed by giddiness, nausea and vomiting. The face is anxious and covered with a cold sweat, and tremors of the fingers and twitching of the lips are noted. The pulse becomes rapid, small, and weak. In most cases improvement gradually sets in, (1-6 hours) and the patient feels fairly comfortable. But in two of my cases vomiting continued until the 3rd day, when death occurred from collapse.

Pain is not nearly so common as one might expect; in many instances even extensive vesication develops without any degree/

degree of discomfort. In the later stages of desquamation there is often a good deal of distress from itching. Yet a number of the most severe cases pass through the whole course of the disease without more than trifling discomfort during daily dressing.

Toxaemia: After the initial shock has passed, the patient complains of little until about the 6th day, when the sloughs begin to separate. Up to this time also the temperature chart gives little cause for anxiety. At this period, probably on account of imperfect leucocytic demarcation, symptoms of septic absorption set in rapidly. Pulse and temperature rise together, and albumin appears in the urine. In the grave cases a definite nephritis occurs, and the patient's resistance to infection gradually diminishes. The urinary condition may yield to treatment, but necrosis of the skin continues with renewed vigour and gangrene supervenes. Death takes place within a week of the onset of acute symptoms.

Complications and sequelae.

Nephritis is the most dangerous complication. Three of the four cases in my series developed this condition between the 4th and the 6th day. The onset was insidious, the first sign of a grave renal lesion being the smoky tint of/

of the urine. Vomiting and oedema did not occur till near the end. The fourth case developed on the 14th day, the sudden onset being marked by rigor and vomit-:ing. This patient ultimately recovered after a long illness. (Cases 31, 32, 37 and 38).

Furunculosis is a fairly common sequel, even in mild cases. The principal situations are the neck, the back, and the buttocks. This condition was well-marked in 18 cases.

Bedsores developed in 2 cases - in one as a terminal phenomenon. The other case (case 38) was one of pro-:longed septic infection with great emaciation; the bedsore did not appear on the site of a burn.

Abscess formation from involvement of the lymphatic glands was present in 8 cases. Two of these were in the groin, and six in the axilla.

Pyuria occurred in three cases; in two of these it was associated with severe septic burns of the genitals; the third was a case of septic absorption from very extensive burns.

Femoral Thrombosis developed in two patients. In one the initial fever, due to Bronchopneumonia and severe burns, began to subside on the 8th day but did not settle satis-:factorily. A long smoulder led to a sharp rise of tem-:perature on the 31st day accompanied by the signs of venous thrombosis/

thrombosis in the left thigh and leg. The left calf measured one inch more than the right. The condition yielded readily to treatment by citric acid. (The temperature chart of this patient is shown on the following page).

Prognosis in a case of extensive burns must necessarily be guarded. Most of the lesions, individually, did well; but the development of nephritis was a possibility which could not be overlooked. If the critical 7th day is safely passed, one may confidently look forward to a favourable result. In cases of moderate severity healing is usually complete by the end of the fourth week. Our after-histories of cases transferred to England show that no invaliding disability results from skin lesions alone.

----------oOo----------

DISEASE.

Notes of Case.

Name Pte. G.

Age

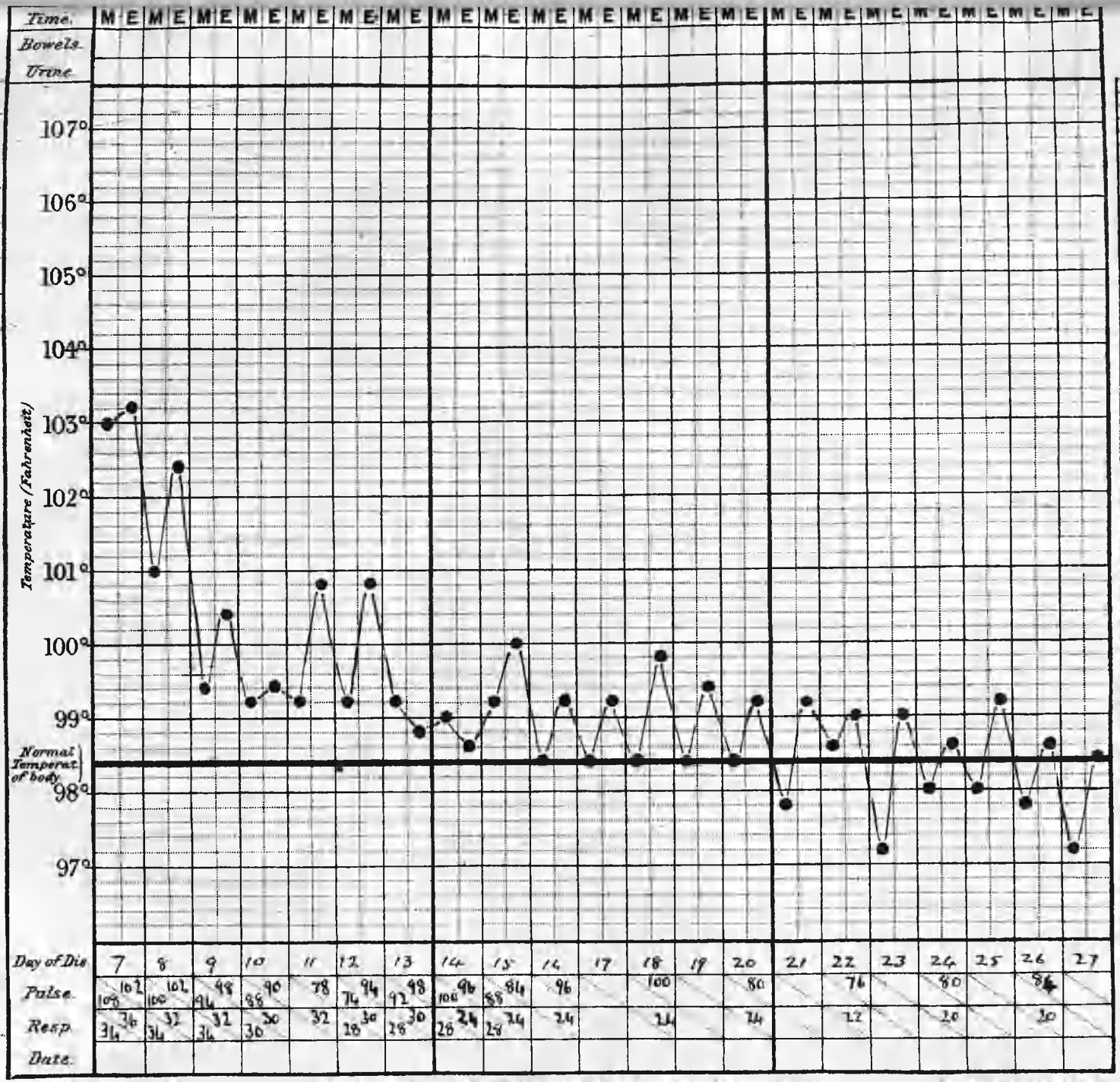
Diet

Case Book N°.

Severe burns on Genitals. Broncho-pneumonia till 13th day. Venous thrombosis Left Thigh from 31st day. Treated with Citric Acid grs. XXX four-hourly.

Date of admission.

Result



Entered at Stationers Hall. Printed and Published by Wodderspoon & Co. 6 Gate Street Kingsway W.C. 2. Gould's Clinical Chart

DISEASE.

Notes of Case.

Name

Age

Diet

Case Book N°

Date of admission.

Result

Time.	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	M E
Bowels.																									
Urine.																									
Day of Dis.	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45							
Pulse.		80	90	80	106	108	110	104	104	116	100	104	96	88	76	76	84								
Resp.		22	22	20	20	24	22	22	24	20	22	22	20	18	18	18	20								
Date.																									

Temperature (Fahrenheit)

107° 106° 105° 104° 103° 102° 101° 100° 99° *Normal Temperat. of body.* 98° 97°

Temperature (Centigrade)

42° 41° 40° 39° 38° 37° 36° 35°

Pain and swelling Left Thigh and Leg

Urine first Temperature fell evacuated

On 6th day.

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DISEASE.

Notes of Case.

Name

Age

Diet

Case Book N°

Date of admission.

Result

Bowels.
Urine.

Temperature (Fahrenheit)

107° 106° 105° 104° 103° 102° 101° 100° 99° *Normal Temperat. of body.* 98° 97°

Pain and swelling Left Thigh and Leg

Inserted Temperature Till evaporated on 60th day

Temperature (Centigrade)

42° 41° 40° 39° 38° 37° 36° 35°

Day of Dis	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45
Pulse		80	90	80	106	108	110	104	104	116	100	104	96	86	76	76	84	
Resp.		22	22	20	20	24	22	22	24	20	22	23	20	18	18	18	20	
Date.																		

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Gould's Clinical Chart

DISEASE.

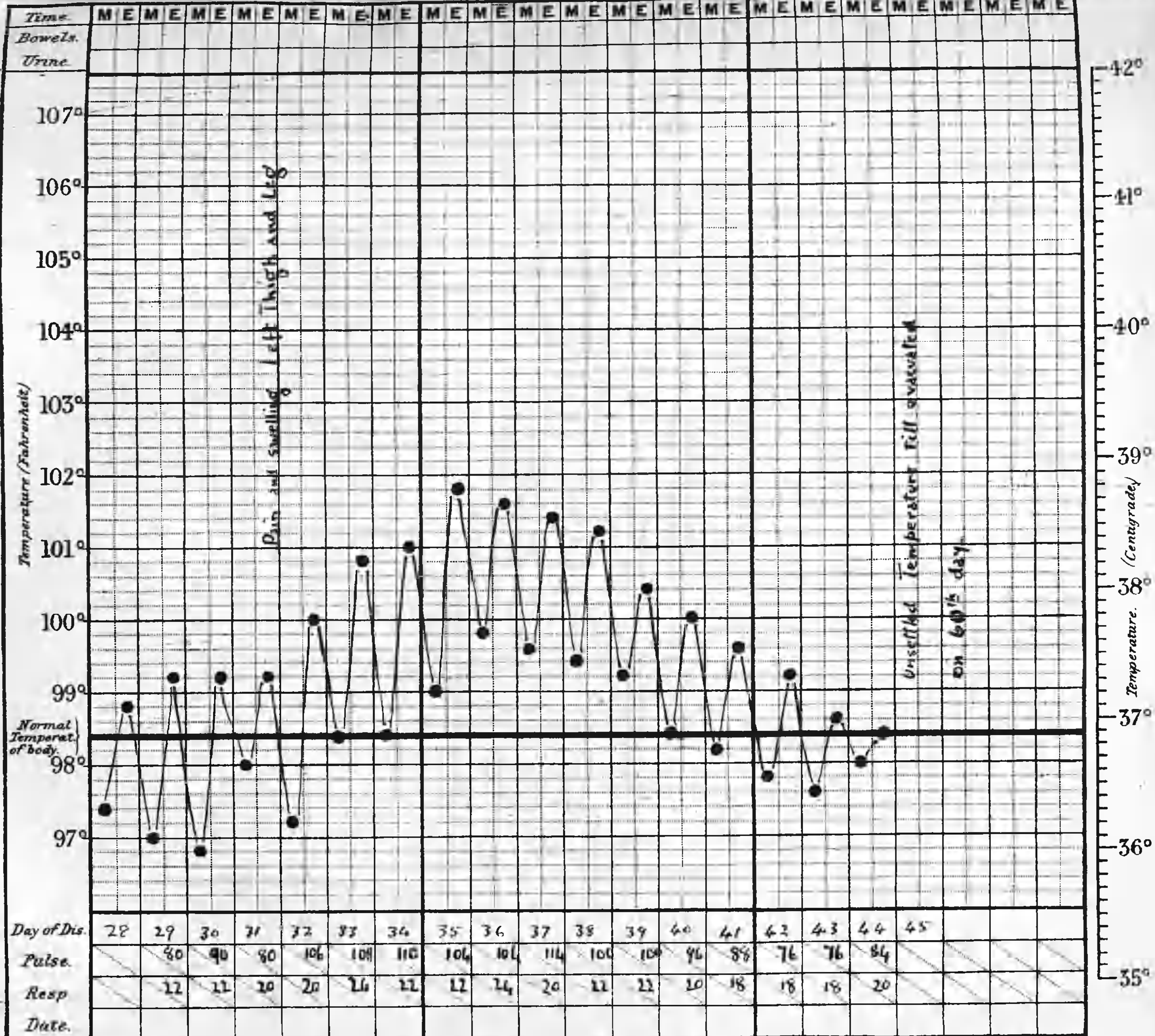
Notes of Case.

Name

Age

Diet

Case Book No.



Date of admission.

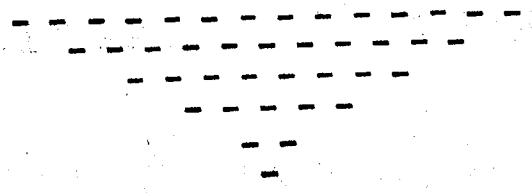
Result

Entered at Stationers Hall. Printed and Published by Wodderspoon & Co. 6 Gate Street Kingsway W.C.2 Gould's Clinical Chart

Notes and References: Section III.

(1) Warthin and Weller "Medical Aspects of Mustard Gas".

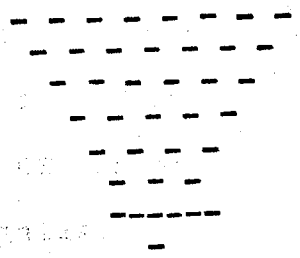
(2) American University Experimental Station. Monograph No. 1.



SECTION IV.

LESIONS OF THE

RESPIRATORY TRACT.



LESIONS OF THE RESPIRATORY TRACT.

A. THE UPPER AIR PASSAGES.

Incidence: With the exception of some fifty cases of uncomplicated skin lesions, inflammatory changes in the nasopharynx were always observed. In most instances the reaction is severe during the first two days, but the condition yields readily to treatment. In only 156 cases was there severe pharyngitis with ulceration or membrane formation, and of these 120 developed acute Bronchitis or Bronchopneumonia. I have not seen any cases of sloughing of the nasal mucous membrane, but obstruction by dense purulent material is common. A definite laryngitis occurred in 218 cases; the following table shows their distribution.

Total number of cases:	1500	Per cent.
Slight Laryngitis.	115	7.6
Severe laryngitis with complete or partial aphonia.	81	5.4
Ulceration of the vocal cords (from laryngologist's report).	22	1.4

Morbid Anatomy: At autopsy the principal lesions are found around the tonsils, the epiglottis, and the larynx. These consist of ulceration and pseudo-membrane formation. The tonsils are often deeply necrosed and one finds not infrequently/

infrequently small shallow oval ulcers behind the posterior pillars and in the pyriform sinuses. Sloughs are found also on the back of the tongue and the epiglottis, but rarely on the posterior pharyngeal wall. The appearances of the larynx and trachea are more characteristic of Mustard Gas. Of 26 necropsies, in 10 a false membrane was found lining the whole length of larynx and trachea; in 4 it extended little beyond the larynx; and in 2 it was confined to the neighbourhood of the cords. In 10 cases no membrane was found. It was present in all acute cases dying between the 3rd and 10th days, and in 4 others (12th day - 2; 15th day - 1, and 17th day - 1). In view of these facts it is a matter for surprise that urgent obstruction of the larynx did not occur. In several cases large flakes of membrane were expectorated (e.g. Case 3). On removal of this false membrane the underlying tracheal surface was found to be dark purple in colour, intensely congested, and denuded of its epithelium.

Symptoms: Much of the discomfort from which gassed patients suffer is due to the early nasal obstruction and the aggravation of the lesions of mouth and throat by the enforced oral breathing. The mucous membrane becomes dry and cracked; the tongue is heavily coated; and sordes collect around the teeth. With the onset of laryngitis the symptoms

are/

are aggravated: there is a short, husky, painful cough, and sputum is brought up with difficulty.

Formerly we attributed many symptoms, such as restlessness, dyspnoea, and cyanosis, to laryngeal obstruction and Dr. Wilson supports this view. (1) A review of the evidence, however, suggests several objections to its acceptance:

(I) These symptoms were well-marked in several cases in which no membrane was found, and they are common in severe Bronchopneumonias of civil life (e.g. The "Suffocative Catarrh" of the older writers).

(II) The dyspnoea is seldom paroxysmal, and in only one case (non-fatal)strider was observed. The dyspnoea continued for many days without any acute crises.

(III) No death occurred from asphyxia of the laryngeal type.

I conclude therefore that, while pseudo-membrane may have aggravated the condition, the real cause of the symptoms lay in the deeper involvement of the lung - con-:solidation-plugging of the smaller bronchi, areas of collapse, and consequent anoxaemia.

B. THE BRONCHI AND LUNGS.

Incidence: In 363 (24.2 per cent.) of my cases there was clinical evidence of involvement of the lower respiratory tract/

tract, the lesions varying from a mild Bronchitis to an intense, rapidly fatal Bronchopneumonia. In 203 of these cases there was pyrexia of more than a week's duration. Respiratory lesions were the principal cause of death in 30 of my 40 fatal cases.

Morbid Anatomy: Summary of the records of 26 Autopsies: The lesions were not confined to one lung in any case, but for the most part were more advanced on the one side than the other. Necrosis, when present, was widely distributed.

Pleurisy was present in all but the fulminating cases. It was generally represented by a covering of thick recent lymph, most frequently over the posterior and diaphragmatic surfaces. No fluid was found in the pleural cavity except in one case (pneumothorax) where an ounce of foul-smelling fluid was observed. In late cases the pleural adhesions were well-organized (cases 29 and 30). The Bronchi contain a yellow or brownish purulent fluid, often in large quantity. The surface of the larger tubes is deep red or purple in colour, and scattered areas of necrosis are clearly visible to the naked eye.

As regards the lung itself, four fairly well-defined types of lesion are found. (1) In the fulminating case, already described, the lungs are bulky, very heavy, and pit on pressure. The surface is dark blue in colour, and fairly uniform/

uniform. On section the cut portions bulge outwards and drip blood and frothy serum. The sizeable bronchi exude on pressure clear or sanious serous fluid, but little, if any, pus. Macroscopically there is no evidence of consolidation but only congestion and oedema. Microscopic examination of the sections shows (2) "Acute Capillary Bronchitis of irregular distribution; in some cases much alveolar oedema relating to severe damage to the bronchioles. The alveolar capillaries are for the most part congested, and there are some small haemorrhages into the alveoli. The exudate is hardly purulent, and few organisms are seen."

(ii) In the Acute Case, in which death took place early in the course of Bronchopneumonia, the appearances closely resemble the secondary Bronchopneumonias of civil life. The lesions found approximate to one or other of two main groups, the disseminated form and the pseudo-lobar. This division is of course artificial, but for the purposes of description may be retained.

The disseminated type is usually recorded clinically as Capillary Bronchitis. The lung is not so bulky as in the fulminating case, and does not pit on pressure. It is dark in colour and shows scattered, depressed, slaty-blue areas of collapse between which are more purple tracts of congestion. Small emphysematous bullae are seen along the/

the free margins. The substance is firmer than normal and largely crepitant; but gentle pressure between the fingers may reveal points more solid than the general substance. On section the cut surface does not bulge as a whole but small prominent areas are seen on close examination. There is a moderate amount of oozing of dark blood, and the smaller bronchi show pin-head projections of yellow muco-pus. The fresh section presents two varieties of colour: around the small bronchi for varying distances are seen circular or wedge-shaped areas of pale red or greyish colour, and finely granular surface. These bronchopneumonia islands are separated from one another by tracts of dark red congested lung. In some cases the solid areas are so minute that the sectional surface appears as though it had been lightly peppered with fine pinkish-red grains. In others the areas are large and many show grouping in clusters around the distribution of a bronchus.

Microscopically the appearances are those of discrete bronchopneumonia radiating from capillary bronchi which are filled with fibrino-purulent plugs. The lesions are most intense close to the bronchiolar walls - from which they originate - and fade gradually towards the periphery, where there is congestion and alveolar catarrh. The exudate shows remarkable variations even in one section: close to the bronchi it is frankly purulent and often haemorrhagic;

farther/

farther from the central focus it becomes fibrinous and oedematous.

The confluent or pseudo-lobar form is recognisable as such by physical signs during life. Post-mortem the lung is fuller and firmer than the above, and large masses are solid to the feel. On section the extensive areas of consolidation stand out prominently. The bronchi exude purulent material on pressure. Microscopically the appearances are those of confluent bronchopneumonia with oedematous, haemorrhagic, and purulent exudate. Very rarely is there difficulty in differentiation from true lobar consolidation, as the exudate is almost always more purulent in the neighbourhood of the bronchioles from which the infection is derived.

(iii) The third group may be roughly termed the suppurative type. It includes a great variety of lesions which have their origin in septic Bronchopneumonia. The acute stage has progressed farther, however, and actual necrosis of lung tissue has taken place. Clinically this group is recognised by the development of profound toxaemia, ill-smelling sputum with characteristic microscopic appearances, and a pyrexia of the swinging septic type.

The post-mortem appearances vary with the advancement of the lesion. In early cases the necrotic changes are not very evident macroscopically, but the solid areas are more friable than/

than usual and the smaller bronchi exude a thin, non-tenacious purulent fluid. In more advanced cases the necrotic areas are plainly visible, grey, breaking, and infiltrated with pus. Microscopically there are widespread acute inflammatory changes of the bronchiolar walls, with much tissue necrosis in and around these, and copious microbic infection.

"Infection" writes Dr. Shaw Dunn, "seems to follow very frequently on the destruction of epithelium in the trachea and bronchi, and it spreads by the bronchi causing the suppurative changes . . .In the worst type the infection acquires a high local virulence, probably because the organisms obtain unimpeded growth inside the fibrin plugs which protect them (as does a sequestrum of bone, for example) from the action of leucocytes. The result is focal necrosis of the bronchiolar walls and of surrounding areas of pulmonary tissue; this may spread to form larger lesions."

(iv) A chronic type of lesion is found in a few cases, but is no doubt representative of a larger number who survive. The appearances are those of a chronic interstitial pneumonia with general fibrous thickening of the alveolar walls. Some alveolar spaces, contain plugs of organizing fibrous tissue, and in one case the bronchioles showed a condition of 'obliterative bronchiolitis.'

A note on Bacteriology:

In the acute Bronchopneumonias direct examination of the sputum/

sputum showed a mixed infection in which either pneumococcus or streptococcus was the predominant organism. My experience of direct and cultural examination of the sputum is not sufficient to justify me in drawing general conclusions. But I may at least say this, that in most of the outspoken Broncho-pneumonias (with clinical signs of consolidation) I found the pneumococcus predominant, while in the more acute and fatal "capillary Bronchitis" the streptococcus held the field. Furthermore, cases of the former type showed a tendency to self limitation, the temperature often falling by crisis or lysis about the 11th to the 14th day. In two cases of confluent Broncho-pneumonia the sputum showed a predominant streptococcus; post mortem the lung tissues gave an abundant culture of a short streptococcus. In the case of empyema (24) and that of pneumothorax (21) the infecting organism, indicated by sputum examination and proved by culture of the pleural fluid, was a haemolytic streptococcus. In two late cases, in which a slow necrosis of pulmonary tissue led to the fatal result, staphylococcus was found in the tissues.

In the case of recrudescences I suggest the following sequence of events as a possibility: a primary mixed infection with a predominant pneumococcus: resolution between the 11th and 14th days so far as the pneumococcus was concerned: and continued invasion of the lung tissue by one of the other pyogenic organisms resulting either in a chronic process or in a renewed access of fever/

fever.

Dr. Wilson,[3] however, repudiated this suggestion, and considered it more probable that the re-crudescence was the result of a ward infection, such as McCallum and Cole[4] describe in relation to their cases of pneumonia following measles. The whole subject of mixed infection and re-crudescence, however, demands further investigation.

SYMPTOMS: The course of a typical case.

It is not easy to tell the exact point of time at which secondary infection of the lung commences. In some cases there is, during the second day, quite a definite abatement of the initial symptoms of gas poisoning, and the pneumonia sets in abruptly with a sharp rise of temperature and marked respirations on the third day. In other cases this lull is not marked, and initial symptoms pass imperceptibly into those characteristic of Bronchopneumonia. By the end of the third day the typical appearances are fully developed. The patient lies on his back with his mouth open; the face is not flushed as a rule, and almost invariably there is a slight cyanotic tinge about the lips and the malar prominences. The mental condition as a rule is clear and vigorous. The breathing is hurried and noisy; very rarely is it either obstructed or voluntarily restricted. Cough is frequent, harsh and painful: the pain, however, is due to the

inflammatory/

inflammatory condition of the larynx: it is not of pleural origin. The pulse is invariably rapid during the acute stage; it is strong and of good volume. The temperature tends to remain high, above 101°, with small daily remissions. In favourable cases the fever persists for from 8 to 14 days (usually about 11 days) and then falls by rapid lysis. In a few cases true crisis occurs. After this period convalescence generally proceeds slowly without serious interruption. A smouldering temperature for a week or ten days is a common feature, and is perhaps due to absorption of toxic products from damaged bronchial walls, necrotic areas of skin, etc. Convalescence is completely established in from three to four weeks. In grave cases the symptoms are more marked. Cyanosis begins early and steadily deepens; pulse and respirations gradually rise, and the temperature has a tendency to fall. Toxaemic signs begin to appear; at first slight evening delirium, and later intense restlessness, muttering delirium, picking at the bedclothes, etc. In many patients, however, the mind remains clear to the end, and death appears to be due to failure of the right heart.

Special Features: Pain: There is frequently complaint of chest pain, but this is not of the pleuritic type. It is more accurately described as a sensation of rawness localized beneath the sternum. It is due to acute inflammation of the trachea and bronchi. In graver cases true pleuritic pain/

pain appears, usually in the region of the nipple on either side. It appears relatively late in the disease and is due to the spread of infection to a localized area of pleura.

Dysphoea is a constant symptom; in the early stages it is accompanied by little real distress, but the struggle for breath is one of the most terrible features of sthenic cases during the last few days. It is apparently due to the filling up of the smaller bronchi with tenacious mucopus; in part also perhaps to the diminished range of expansion of a lung whose elasticity is greatly impaired; and in part possibly to a condition of bronchial spasm. This oppressive type of dyspnoea is not a characteristic of toxaemic cases.

Cough: The peculiar nature of the cough in Mustard Gas bronchopneumonia is mainly accounted for by the laryngo-tracheal involvement. In the early stages the cough is intensely hoarse, and the impaired movements of the vocal cords and the pharyngeal muscles do not give it the "grip" necessary for the ejection of sputum. Consequently, although the sputum has not the tenacious character found in lobar pneumonia, the patient has the greatest difficulty in expectoration. In fatal cases the expulsive power of the cough is abolished altogether, and the mucopurulent material lies undisturbed in the pharynx or larynx. It is this which gives rise to the well known laryngeal gurgle which appears in these cases as a terminal phenomenon.

The Sputum is of considerable importance. In all severe cases

I/

I estimated the daily total, and examined specimens both in bulk and microscopically, as a routine. The chart of the daily total was often of value in following the progress of the case. A gradual fall in a total originally high was of good prognosis, while a sputum total persistently high after the fall of temperature and respirations was a warning of further mischief. By examination in bulk the development of necrosis, bronchiectasis and gangrene were indicated by the characteristic appearance of the lowest layer on settling; the smell varied from a faint post mortem room odour in necrosis to an intense foetor in gangrene.

In microscopic examination, after many failures, I found the technique recommended by Besançon, and de Jong (5) simple in execution and of very considerable clinical value. A portion of sputum was received directly into a sterile Petri dish. In the laboratory it was washed with normal saline to remove gross contamination and then spread on slides with a platinum loop. The routine stains used were Unna's Methylene Blue and Gram's stain. In each case a rough count of organisms was made, disregarding the buccal and pharyngeal cells, and the predominant organism noted. The diagnostic accuracy of this method was strikingly demonstrated at several autopsies and in the case of empyema. The nature of the matrix was then ascertained, the reddish hyalin mucus being differentiated from the violet-blue sero-albuminous exudate. By this means the existence/

existence and degree of oedema could be gauged. Finally, the cellular appearances were inspected, and the existence of extreme degeneration, excess of blood corpuscles, eosinophilia, etc., specially noted. Examination of smears revealed in cases of extensive necrosis and gangrene the presence of elastic tissue.

Physical signs:

Inspection: Orthopnoea is the rule during the acute stage. There is rarely any difference to be noted between the two sides. The whole chest is increased in volume, and the range of respiratory movement is diminished. Powell and Hartley (6) point out that in health the resiliency of the chest wall is in favour of inspiration. Now in the oedema and broncho-pneumonia of gas poisoning the lungs are increased in bulk, so that this reserve power of resilience is more or less neutralized. The reserve capacity of the thorax being thus taken up, inspiration becomes an act of voluntary force, and the accessory muscles of inspiration are brought into play. In grave cases, sucking in of the inter costal spaces and of the lower ribs signifies a profound interference with the respiratory function.

Palpation is of little value, and vocal fremitus is not obtainable as a sign.

Percussion: In the early stages of an acute case there is always some impairment at the bases. Later, when the oedema has diminished/

diminished, and the bronchopneumonia is fully developed, this impairment often clears, so that the whole chest is resonant. Bronchopneumonic consolidation was indicated by definite dulness in less than half the cases, throughout the course of the illness. During the first five days the presence of large areas of dulness was exceptional. Alteration of the percussion note is much more common in late cases, in which a pleural involvement has appeared.

Auscultation: During the first few days there is little alteration of the breath sounds. Two varieties of adventitious sounds are heard in typical cases -

(1) Sonorous and large bubbling râles obviously derived from the larger tubes.

(2) Small bubbling râles, somewhat distant and muffled, due to the presence of oedema of the lung.

During the succeeding days there is an extraordinary variety of auscultatory sounds, very difficult to interpret. Three types may be distinguished.

(a) The outspoken bronchopneumonia: prolonged expiration and bronchial breathing are heard over large areas of the chest wall. Adventitious sounds are few or absent. Bronchophony is well-marked.

(b) The discrete type: Small patches of bronchial or broncho-vesicular breathing are discovered. Elsewhere are heard sibilant and small bubbling râles. In some cases bronchial breathing is not so evident, but small or medium crackling

râles/

râles are heard, coming right up into the stethoscope. This, in my experience, is a valuable and important sign of consolidation. The voice sounds are not markedly altered.

(c) The clinical "Capillary bronchitis": In this type the voice and breath sounds are feeble, especially at the bases. Expiration is hardly audible. These sounds, however, are largely masked by innumerable small bubbling râles which are heard throughout the chest.

In the stage of resolution the signs of consolidation disappear as a rule very slowly. Well-marked crepitus redux is present in about half the cases.

COMPLICATIONS AND TERMINATIONS.

Compared with Influenzal Pneumonia the complications of Mustard Gas Pneumonia are few.

Pleurisy, as demonstrated by accumulations of lymph on the lung surfaces, and the presence of recent adhesions, is found in most of the fatal cases dying after the second day. It is seldom made obvious during life wither subjectively or by examination. A typical acute pleurisy clinically demonstrable by friction sounds, occurs not infrequently after the acute stage has passed. The temperature chart, after settling for a few days, shows a secondary rise, as shown in cases 13-16.

Empyema/

Empyema occurred in only one case in our series. It developed on the left side after a temporary abatement of the acute bronchopneumonia between the 11th and 14th days. The causative organism was a streptococcus. A fuller description of this case is given in the appendix (case 24)

Pneumothorax occurred in one case. It developed on the 17th day, and was due to the rupture of a small abscess near the apex of the right lower lobe (case 21)

No Cardiac, cerebral, or other complications were observed.

Pulmonary apoplexy was noted in one case. The patient was admitted on the 3rd day suffering from extensive burns: the progress of these was unfavourable and on the 9th day the pulse began to fail and the patient became delirious and incontinent. Up to the 8th day the chest signs gave no special cause for anxiety. On the 9th day the breathing was very hurried and the sputum tinged with blood; there was extreme thirst and a running pulse. (Case 36)

Post Mortem a fresh and extensive haemorrhage, the size of an apple, was discovered at the base of the left lung. There were no signs of organization in the red clot.

Termination in Bronchiectasis.

Of 26 autopsies a well-marked bronchiectasis was present in 3 cases. In one of these (death on 13th day) the right lower lobe showed advanced cylindrical bronchiectasis. Another was associated with gangrene - the case quoted ~~above~~ below. In the third

case/

case (death on 56th day) the left lower lobe showed little else than dilated cylindrical and saccular bronchi (cases 22, 23, and 29.)

It is difficult to estimate the frequency of Bronchie-:ctasis in patients who survived. In the three fatal cases the physical signs were masked by concurrent Bronchitis and Bronchopneumonia. The diagnosis was made from the special characters of the sputum, and the type of the fever. Similarly in several non-fatal cases Bronchiectasis was diagnosed by this means. The characteristic type of fever is shown in the charts. (Cases 26, 27, 28.) Wasting is a very prominent manifestation of this condition.

Termination in Pulmonary necrosis and abscess formation.

Necrotic changes were found in the broncho-pneumonic patches in 10 cases, and in four actual abscess formation occurred. In three of these the abscess was in the left upper lobe. No typical signs were present during life; the temperature charts were in no way characteristic - The only indications, in fact were those presented by the character of the sputum. (Case 20 appendix)

Termination in Gangrene.

In this series one case occurred. The clinical signs were those of intense toxaemia and a horrible foetor of the sputum. The patient died on the 15th day. Post mortem examination showed/

showed in addition to the characteristic appearances of disseminated gangrene of the lung, almost complete destruction of the vocal cords (case 23.)

Termination in Chronic Pneumonia.

Delayed resolution was of common occurrence, even in relatively mild cases. In most cases it amounted to persistent cough with mucopurulent sputum, and a smoulder of temperature lasting for several weeks. In a few, a definite recrudescence occurred, ushered in by acute pleuritic pain. In two cases only chronic pneumonia was demonstrated post mortem.

It is not easy to determine to what extent chronic Pneumonia developed in cases transferred to England. That it did occur in a small proportion is clear from the after-histories which we have received through the courtesy of the medical research Committee. In two cases fibroid changes were demonstrated by X-ray as well as clinical examination. Examination for the Tubercle Bacillus was always negative. The period of invalidism in cases transferred to England varied between three and six months.

Prognosis.

In this series, the mortality from Bronchopneumonia was 20 per cent., most of the deaths occurring in the acute stage between the 3rd and 11th days. Grave indications are the early onset of deep cyanosis and toxaemia. A steady decline

in/

in the sputum curve is a good sign, but a rapid fall with abolition of the cough reflex is a fatal indication. The evidence from pulse and respirations is of doubtful value and even the degree of pyrexia may be very deceptive. Death appears to be due quite as frequently to interference with the function of respiration by oedema and consolidation, as it is to poisoning of the vaso-motor centres. In this respect, the condition differs from the pneumonias of civil life.(6)

Diagnosis.

A definite diagnosis of Bronchopneumonia can seldom be made on physical signs alone, but from post mortem evidence one can confidently assert that in severe cases of gas poisoning after the third day, where the main trouble is in the chest, consolidation is always present.

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Notes and References.

(1) Quarterly Journal of Medicine, January 1920.

(2) Dr. J. Shaw Dunn - private report on sections. The great part of the patholical histology referred to in this section is founded on Dr. Shaw Dunn's reports on sections submitted to him by Dr. Ingram.

(3) Quarterly Journal of Medicine, January 1920.

(4) McCallum and Cole, Journ. Amer. Med. Assoc. April 1918, lXX, No. 16,1147.

(5) Besançon and De Jong, "L'Examen des Crachats".

(6) Osler, Principles and Practice of Medicine page 97.

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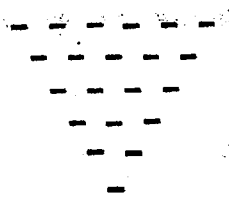
S E C T I O N V.

SYSTEMIC EFFECTS.



SYSTEMIC EFFECTS.

The official French accounts of Mustard Gas Poisoning,[1] issued during 1918, expressed strongly the view that Mustard Gas, as such, always exercises a poisonous influence on the general system. The German reports[2] (published July 1918) were more guarded, but hinted at the possibility of absorption of the gas by the skin and consequent systemic lesions. The general trend of more recent studies has been to emphasize the local lesions and to consider such general symptoms as occur to be largely due to secondary infection. Warthin[3] for example, concludes that "all the changes seen can be explained as due to the direct local action of Mustard Gas Vapour, or as secondary to shock or secondary infection of the lesions."

It is now necessary to examine in detail the evidence for and against the view that Mustard gas has a specific general action:

1. Meyer,[4] the discoverer of Dichlorethylsulphide, stated that the local application of the liquid or its vapour to the skin was able to give rise to metastatic inflammation of the eye and to pneumonia. This statement received consider-:able support but was finally disproved, so far as animal experiments were concerned, by Warthin. It has been dis-:proved again and again in my own experience. I have collected six cases of very severe skin lesions which developed no conjunctivitis/

conjunctivitis and no respiratory symptoms. One of these (case 31) came to autopsy, and no damage whatever was found in eyes, bronchi, or lungs.

2. Gastro-intestinal system: The French accounts state that where there is a general affection of the skin produced by Mustard Gas, the digestive apparatus is always affected. "The gastric mucosa has lost its sheen and is of a darkish-red colour. One finds on the greater curvature punctate ecchymoses and sometimes true ulceration . . . . The intestine is often affected. Towards the end of the jejunum and in the ileum one finds oedematous segments of bowel, infiltrated and congested throughout their whole thickness. The large intestine is more rarely affected: the lesions are usually situated in the rectum, and false membrane and ulcer-:ation may be seen." The German war accounts note "Malaise and frequent vomiting at the commencement of the illness . . . frequent digestive troubles such as pain in the stomach, diarrhoea and vomiting, and obstinate constipation. Haemorrhages in the intestinal mucous membrane, as well as inflammatory exudates . . .have been found at autopsy."

Warthin, on the other hand, after reviewing the evidence brought forward by French and Italian authors, summarizes the results of his own experimental work as follows:

"The gastro-intestinal symptoms seen in gassed human beings are probably chiefly reflex, associated either with shock or

with/

with respiratory irritation. As in other forms of gassing, it is probable that the erosions or ulcers of the stomach and intestine may be embolic in character, the emboli arising in the primary or infected mustard gas lesions of the skin or elsewhere. It is also possible that in man as in animals, localised eschars of the gastro-intestinal mucosa may be produced by the direct action of mustard gas swallowed in contaminated food or saliva (5)".

From the cases of my series I have obtained clinical and pathological evidence.

(a) Clinical evidence: The symptoms which lead one to suspect gastro-intestinal lesions are nausea, vomiting, abdominal pain, and diarrhoea or constipation. Now, while a history of nausea and vomiting is given by the majority of severely gassed patients, the persistence of these symptoms beyond the second day is very rare indeed. In this series only 13 cases were collected: 8 of these suffer-:ed from extensive Burns: in these the vomiting was associated with other manif-estations of severe shock. Two of them, in fact, died in a syncopal attack. The remaining 5 cases gave a history of having drunk contaminated shell-hole water: on admission they suffered from nausea, salivation, and occasional vomiting after food. There were no lesions of the eyes or the respiratory tract, but three showed/

showed pharyngeal congestion and excoriations round the lips. The symptoms persisted in a mild degree, without pyrexia, for 3 - 5 days, but all 5 were completely convalescent within 10 days of onset.

Reference is made later to a small group of cases in which delayed vomiting, undoubtedly functional in origin, occurred. (Section VI.)

Diarrhoea was noted in 48 cases during their stay in hospital. It was no more frequent in gassed men than in the ordinary medical cases of the wards. In the more serious gas cases diarrhoea, or indeed any sign of gastro-intestinal disturbance, was extremely rare after the first 48 hours. Constipation, of course, was common - but no more severe than one usually finds in a strong man who is suddenly confined to bed and a milk diet. Abdominal pain was a much more frequent complaint, and often accompanied by epigastric tenderness. It was, in most cases, the result of muscular strain produced by coughing.

(b) Pathological evidence: 10 cases in which vomiting was a prominent symptom during life, came eventually to autopsy. In 3 of these vomiting had continued up to the point of death. In these three cases the gastric vessels were deeply engorged, and petechial haemorrhages were seen around the lesser curvature and in the duodenum. One case showed/

showed a patch, the size of a five-shilling piece in which the gastric mucosa had definitely lost its sheen. Sections were transmitted to Dr. Shaw Dunn who reported (6) that there were certainly no gross lesions to be found. In the remain-:ing 7 cases no change greater than congestion was found. I examined the entire intestine in all 10 cases and found no signs of any changes deeper than a simple engorgement of vessels.

In consideration of this evidence, then, I conclude that gastro-intestinal symptoms are a rare pheuomenon in Mustard Gas Poisoning, but that when present, they are due to the operation of one of the following causes:

(1) Shock: This, as already mentioned, is a common manifestation of the onset of extensive skin involvement, and is relatively slight in cases free of serious cutaneous injury. Similarly, vomiting is well-marked and prolonged only in cases where the skin is seriously affected. I believe that shock is the most common exciting cause of vomiting.

(2) The swallowing of contaminated material, with saliva etc., is a possible cause; it is not improbable that small quantities of mustard gas are sufficient to act as an emetic, although not in such strength as to cause gross lesions.

(3) Reflex irritation from cough, the tickling of an oedematous uvula etc., do give rise to vomiting in certain cases; but this type of vomiting is readily distinguished

by/

by its onset only after a prolonged paroxysm of coughing.

3. The Urinary System: The French authorities lay great stress on the urinary changes as indicative of a general poisoning of the system: "In serious cases it is almost constantly present; if it reaches a marked degree and persists, it is a grave sign. It has a true prognostic value."

Dr. G.R. Herriman,(7) after a careful study of 30 cases of Mustard Gas Poisoning of all degrees of severity, comes to the following conclusions: "Mild cases of skin burns show no changes in the urine. Moderately severe and severe cases show after the first week definite changes consisting of a diminution of the urinary output, increased concentration and acidity, albuminuria, and diminished urea and chloride output; in the sediment may be found casts, renal epithelium, red blood cells and an increased number of leucocytes.

We believe that the changes in the urine may be interpreted as dependent upon the secondary infection and, in part possibly, due to the absorption of toxic products from the necrotic skin rather than to any direct toxic action of Mustard Gas."

Unfortunately Herriman's cases were not seen until after the 10th day of illness, so he was unable to determine the time of onset of these changes. I have been more fortunate in securing earlier cases - some within 24 hours of gassing, -

and/

and so am able to arrive at definite conclusions, on this point. On the other hand I had neither the apparatus nor the opportunity to make such complete analyses as Dr. Herriman has done.

In my series the urine of nearly a thousand patients was examined - a number which included all the severe cases. The occurrence of albuminuria is strikingly rare; I was able to collect only 30 cases in which more than a faint trace was present. These cases were all serious, and in every one secondary infection had taken place before the appearance of the albumin.

Sixteen of these cases were associated with very severe cutaneous lesions; in these albuminuria did not appear until after the 4th day, when necrosis was at its height and separat-:ion of the sloughs had begun. It was not an isolated phenomenon but was always accompanied by other signs of septic absorption. In 4 of the worst cases an acute nephritis supervened.

14 cases of albuminuria remain to be considered: in 3 of these the onset was definitely related to the development of a complication - empyema, pulmonary abscess, and femoral thrombosis. In the remaining 11 cases it was associated with severe bronchopneumonia; in these it was moderate in amount, and the urine presented the usual febrile characters of high colour, high specific gravity and increased acidity. Such appearances/

appearances are, of course, not peculiar to Mustard Gas, but may be found in many cases of pneumonia in civil life. A number of my bronchopneumonic cases were free from albumin throughout the illness.

Microscopically the usual appearances were noted, the only remarkable features being the large number of blood-casts in three cases of nephritis.

Other Urinary Characters. In cases of extensive burns one constantly found a very high degree of acidity, but neither acetone nor diacetic acid were present, even in the terminal stages. No sugar was found in any case.

Findings at Autopsy: In 15 out of the 18 cases in which Kidney sections were examined Dr. Shaw Dunn found "Marked congestion, especially of the glomeruli. This" he adds, "has no specific significance, as it is almost always present with other gases."

As a prognostic feature albuminuria is in itself of little significance. In many fatal cases it appeared only as a terminal phenomenon, while not a few, in which it appeared early and in considerable amount, ultimately made a perfect recovery.

The significance of renal changes may therefore be summed up as follows:

A marked degree of albuminuria is a rare event in Mustard Gas Poisoning. When present, it is always associated with

secondary/

secondary bacterial infection. The severest degrees of albuminuria, and actual nephritis, have been found only in cases of extensive septic burns.

4. The Blood: The interpretation of the Blood changes following upon Mustard Gas Poisoning has been the subject of much controversy: Dr. Matthew J. Stewart (8) examined the blood in a number of cases and found in most a leucocytosis with a relatively high polymorph count. He concluded that, so long as the leucocytosis was maintained, the case generally fared well; but a progressive fall in the count in the course of the infection was of grave omen.

Dr. G.R. Herriman (7) examined the blood in 30 cases, and found - in severe infections - a well-marked polymorph leucocytosis. He observed no cases of leucopenia. It is to be noted, however, that none of his cases were examined before the 10th day of illness; it is probable therefore that by that time regenerative changes were in the ascendant. As one would expect, Dr. Herriman also finds a slight secondary anaemia in severe cases; eosinophilia, and the appearance of myelocytes and young forms of leucocytes.

The most extensive blood examinations in this condition were those undertaken by Dr. Krumbhaar (9). This observer found a leucopenia in 23 out of 108 cases examined, but "most of these could be examined only once, shortly after being gassed." This examination does not appear to be of much value, as many of the/

the counts may have been made, before the development of blood changes had begun. In 31 cases, however, Dr. Krumbhaar made more than one examination, and discovered a leucopenia in 19, while 6 of the remaining 12 showed a falling count. "When the leucocyte count fell below 5000," he adds, "recovery followed in only three cases; so that a severe leucopenia came to indicate a very bad prognosis, and a persisting leucocytosis a good prognosis." In all these cases Dr. Krumbhaar finds, but does not emphasize, an initial leucocytosis.

The findings of these observers, it seems to me, are essentially similar: the two workers on early cases find an initial leucocytosis corresponding with the onset of secondary infection; except in a few grave cases in which either a leucopenia or a falling count was observed. In favourable cases a leucocytosis persisted. The observer of late cases finds a leucocytosis in combination with regenerative blood changes. These changes are in no way specific, but are found in many other states, notably severe pneumonias of civil life. I consider, therefore, that Dr. Krumbhaar has no justification for his conclusion that "mustard gas exercises on the blood a direct toxic action."

My own results are in agreement with the above findings: Dr. G.H.H. Almond and I were able to study only 10 cases, but these were fairly representative of the severe types with which we had to deal.

The/

The red corpuscles numbered over five million in all but two prolonged septic cases, in which the counts were respectively 4,300,000 and 4,100,000. The haemoglobin content was never below 75%. The leucocytes showed no change until the 3rd morning, (morning examinations only were made) when a sharp rise in numbers signalized the onset of secondary infection. The average count in the first week was between 15,000 and 25,000. One case only - one of profound toxaemia with death on the 6th day - showed a leucopenia (7,300 on the 4th morning). One fatal case showed a progressive fall in the count from 14,000 to 8,000 on the day before death. In the remainder the leucocytosis was maintained, although two deaths occurred. One case of severe burns reached the figure of 44,000 in the second week. In all cases the polymorphs were relatively high - 75 - 85 per cent. We did not find eosinophilia in any cases, but we did not pursue our investigation beyond the third week. In three late cases (3rd month) the leucocytosis had disappeared.

Blood-Pressure: Dr. Frazer undertook a series of observations on the blood-pressure in a considerable number of our cases. No changes were observed in the lighter cases and even in the more severe the results were largely negative. Two points of interest, however, were noted:- a fall of pressure accompanying the initial shock (two fulminating cases) and in three "sthenic" cases in which death took place from "suffocation" a rapid rise

in/

in the systolic readings to 160 mm. (two cases) and 170 m.m. (one case).

5. The Heart: There are few references to the condition of the heart in the literature of Mustard Gas Poisoning. In this series two types of case occurred in which symptoms referable to the heart were noted. The first of these, which may be described under the reading of "Effort Syndrome" I shall refer to later. In the second type, which affected a small number of severe cases, the maximum impulse of the heart was situated from 4½ to 5½ inches to the left of the midsternal line, and symptoms of cardiac insufficiency were present. In one early case of severe burns the patient died of syncope. Eleven cases, in all, were collected, and all but two were severely burnt. No appreciable degree of dilatation was seen in cases of uncomplicated pneumonia. In one case only were the classical signs of cardiac failure noted. I cannot, with any confidence, suggest an interpretation of these cardiac signs. In some probably it was a manifestation of shock, while in others the struggle of the right ventricle against pulmonary obstruction may have led to an acute dilatation. The diagnosis of enlargement of the heart was by no means always confirmed at autopsy; probably the condition was overcome by the onset of rigor mortis.

6. TOXAEMIA:

A comparison of the respiratory complications of Influenza with/

with those of Mustard Gas Poisoning throws a good deal of light on the true nature of the general symptoms in the latter condition. In this respect the two diseases present a close superficial resemblance and a striking fundamental difference.

In both conditions there is a primary injury to the bodily tissues, in particular those of the respiratory tract, which opens the door to secondary infection by pyogenic organisms. In Influenza the causal agent - whatever be its actual nature - is essentially a toxic one, and prepares the way for secondary infection by a general lowering of the patient's vitality and resistance. Mustard Gas, on the other hand, is primarily mechanical in its action, giving rise to local tissue damage - of the bronchial walls, for example - and secondary infection develops in, and spreads from, these lesions. For this reason the gassed patient (save in the case of shock from extensive burns) is sthenic at the time of onset of secondary infection, while the Influenza patient already suffers from toxaemia. In both conditions the presence and degree of 'secondary' toxaemia depend upon the nature and the virulence of the organism causing secondary infection, and the extent to which the patient's vitality has been lowered by the causal agent.

Thus it comes about that no case in this series developed toxaemic symptoms within 72 hours of gassing (while in 40 fatal cases of Influenza under my care toxaemia was frequently present within 24 hours).

Secondary/

Secondary toxaemia (If I may use this term in the sense described above) was present in about 50 per cent of my fatal cases, and in 3 per cent of the whole series. As a prognostic sign it is of no great value, for several fatal cases were rational, vigorous and clear-headed to the last (see case 4). In many cases recovery followed quite severe degrees of toxaemia.

Two clinical types of toxaemia were recognised:

(i) A state of apathy or somnolence, very characteristic of the severe Mustard Gas case. "He often falls asleep with the drinking-cup at his lips" was the note on a severe case of burns. This type was strangely persistent: as late as the 19th day some patients complained of an uncontrollable tendency to sleep. Apathy, rather than somnolence, was the mark of the severe pneumonia: "He lies on his back taking no notice of anything. When he coughs, he rolls the sputum languidly round his mouth and makes no attempt to expectorate." Yet all these patients are quite rational, when roused.

(ii) The Terminal Toxaemia: This differs little from the ordinary type seen in any fatal case of acute infectious disease - Wandering, delirium, mental confusion, trying to get out of bed; and in overwhelming cases, low incoherent muttering, picking at the bedclothes etc., and incontinence. In this series the onset of toxaemia of this type was always associated/

associated with pulmonary necrosis.

7. Liability to Intercurrent Disease:

In severe cases of Gas Poisoning there is always some degree of lowered resistance to subsequent infection. This is not more evident after Gassing than after other septic diseases, nor is there any specific liability to infection. Two examples may be quoted : -

(i) The incidence of Influenza was no greater, and its mortality no higher, in gassed cases than in the ordinary hospital patient. Three patients, who were just convalescent from severe Gas Poisoning, developed Influenzal Pneumonia. One died on the 8th day (96 days after gassing); the other two (cases 15 and 24) made perfect recoveries. No other severe cases, so far as I can ascertain, succumbed to Influenza, although 6 were acutely ill during their stay in hospital.

(ii) A small outbreak of Diphtheria occurred in hospital at a time when more than 150 Mustard Gas Casualties were under treatment. Dr. Ingram and I took cultures from the throats of 140 patients, and found positive results definitely more frequent among the Convalescents from Gas Poisoning. Only two gassed cases, however, were numbered among the 20 who developed the disease. The one, a convalescent, died; the other, a serious case of Bronchopneumonia, recovered.

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Notes and References.

(1) Notice clinique et thérapeutique de l'intoxication par les gaz, 1918.

(2) German Instructions regarding the diagnosis and treatment of Gas Poisoning, Jan. 1918.

(3) Warthin. "The Medical Aspects of Mustard Gas Poisoning".

(4) Quoted from Warthin as above.

(5) Ibid.

(6) Dr. Shaw Dunn - report on Dr. Ingram's sections.

(7) Dr. G.R. Herriman - Clinical Pathology of Mustard Gas Poisoning.

(8) Matthew J. Stewart. Reports of Chemical Warfare Medical Committee, No. 17.

(9) E.B. Krumbhaar: "Blood and Bone Marrow in Yellow Cross Gas (Mustard Gas) Poisoning."
Journ. Amer. Res. Vol XL No. 3. Sept. 1919.

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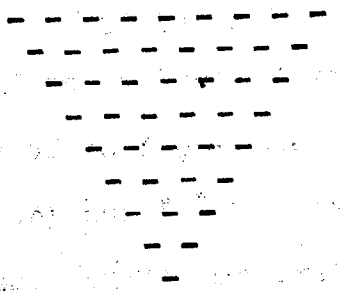
FUNCTIONAL CONDITIONS

ASSOCIATED WITH

MUSTARD GAS POISONING.



FUNCTIONAL CONDITIONS ASSOCIATED WITH MUSTARD GAS POISONING.

It has been shown in the foregoing sections that the organic lesions resulting from Mustard Gas Poisoning arise from

(a) direct local action of the escharotic substance;

(b) the infection of a local lesion by pyogenic bacteria, and the spread of organismal processes from these foci, and

(c) the absorption of bacterial toxins from an infected lesion into the general circulation.

There is no evidence, clinical or pathological, that dichlorethylsulphide, per se, gives rise to lesions of the general system. Nevertheless, Mustard Gas Poisoning is the exciting cause of an important group of disorders to which the term "functional" * has been applied. The principal functional conditions are closely related to the most common organic lesions produced by the Gas,

thus/: -

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* I use the term "functional" in the wide sense of "opposed to organic" without implying any narrower significance in which the word has been employed.

thus : -

Site of lesion	Organic effect	Functional effect
The eyes.	Conjunctivitis, Blepharitis and Keratitis.	Persistent photo-:phobia and lacrimation.
Respiratory Tract.	Laryngitis, Bronchitis, and Bronchopneumonia.	Aphonia, night cough, chest pain.
Gastro-intestinal system.	Acute vomiting.	Persistent vomit-:ing, diarrhoea, and abdominal pain.
Nervous system.	Shock, collapse.	Anxiety-neurosis, Effort-syndrome.

Incidence: Functional manifestations played a part of no little importance in this form of poisoning. Their significance may be gauged from the fact that 332 (22%) of our cases were delayed in hospital for several weeks beyond the normal period on this account alone. The following table shows the "bodily distribution" of the functional conditions in this series:-

Total number of cases	1500	Per cent.
Functional phenomena.	332	22
Photophobia.	189	12.6
Aphonia.	108	7.2
Vomiting.	16	1.0
"Effort syndrome".	19	1.2
	332	22.0

Aetilogy/

Aetiology and Pathology: At the risk of covering much trodden ground, I shall attempt to trace the history of these cases from their real origin - apart from the immed-:iate or exciting cause - and so establish a connecting link between them and the more generally known neurosis of War.

War neurosis, in general, arises out of the conflict between instinctive tendencies and certain controlling forces the use of which the individual acquires in the course of experience. Instinct as defined by McDougall,(1) is "an innate psycho-physical disposition which determines its possessor to perceive, and to pay attention to, objects of a certain class; to experience an emotional excitement of a particular quality upon perceiving such an object; and to act in regard to it in a particular manner, or at least to experience an impulse to such action." The instinct with which we are concerned is that of self preservation; the object perceived is the hostile menace, whether attack, or shell-fire, or poison gas; the emotional excitement is fear or a modification thereof, and the unmodified reaction to that instinct is flight.

We have now to consider the nature of the controlling forces, the development of which prepares the individual to withstand the stress and shock of war. These forces are of two kinds, general and specific:

The general forces: "Every normal human being grows up under the/

the constant influence of the society into which he is born, and his mental development is moulded by it at every point. He becomes the heir to an intellectual and moral tradition which has slowly been built up, bit by bit, through the efforts of thousands of generations." (2) I need not linger over these points. For the soldier the moral tradition is of vital importance, by which propositions such as that fear is reprehensible, that loyalty to national ideals is the highest virtue, are accepted without question. The social training of the individual is no less important, for thereby he is raised from the low level of purely selfish behaviour to the plane of conduct where he thinks and acts in the interests of the mass.

This brief outline will serve to indicate the nature of the general forces of control, a heritage of every civilized man. The application of specific forces is reserved for the training of the soldier.

In preparing the soldier for war, special attention is paid to the instinct of self-preservation, and all military training is directed, explicitly or implicitly, towards the strengthening of its control. By means of drill and military discipline, the vague ill-directed forces of the gregarious instinct are stimulated and organized for the combined action so necessary in war. By bayonet-drill, instructional attacks and the like, the instinct of pugnacity is aroused, and the unconscious'

unconscious mind of the soldier still further strengthened against fear. Moreover, military training makes a strong appeal to the higher mental qualities, by encouraging self-control, esprit de corps, and endurance.

Thus it is that the soldier sets out for the front with a strong balance on the side of control. The normal well-trained man succeeds in preserving this balance - without conscious effort - through his whole military career. Many others, however, whether through innate weakness, or faulty upbringing, or insufficient training, begin sooner or later to feel the strain of mental conflict. The first weakening usually occurs on the emotional side; it is not the shell-fire, the monotony, nor the cold and misery of the trenches, it is an intense emotional experience, such as the sight of a comrade killed, which first casts a shadow on the mind that no effort of will can remove. From this point the strain and horror of it all begin to tell; the healthy process of suppression,(3) by which distressing sights and sounds are unwittingly blotted out from the consciousness, begins to fail, and a system of voluntary repression has to be employed in its stead. Repression, being voluntary, involves a mental strain; furthermore, conscious control may be successful by day, but at night repressed thoughts and memories crowd into the mind, and their baneful influence disturbs sleep, gives rise to war-dreams, and produces ever-increasing anxiety/

anxiety and depression. In spite of this vicious circle of mental processes, repression may hold sway successfully for a long period . . . . until one day an unusually severe shock overweighs the balance beyond redress.

Such is the history which, over and over again, I have obtained from men who were suffering from functional conditions associated with gas poisoning - an initial lowering of resistance produced by the influence of shock, illness or fatigue; and then the long wearing process of mental strain and conflict.

But, the question arises, why should mild gas poisoning be considered a form of shock, peculiarly liable to overthrow the control of the self-preservation instinct? In my view, the answer is this, that gas poisoning calls into being a complex emotion, fear blended with a sense of mystery, a feeling of the unknown, which makes a profound impression on the patient's mind. During the recent war, in spite of the ever-increasing destructive power of rifle and shell-fire, the soldier became more or less accustomed to their effects, the nature of which he understood. But poison gas has not yet been released from the shadow of mystery; the presence and the strange workings of this viewless courier of death fill the man's mind with a peculiar lingering dread. If his mental balance is already at the turning point, neurosis is certain to develop, unless he can find a means of escape from the conflict.

His/

His lowered mental vigour, due to the dissipation of energy which has been necessary to maintain repression, puts him in a position particularly favourable to the influence of suggestion. His mind, groping for a way of escape, seizes greedily upon a symptom, and by a process of suggestion accepts it as a solution. - Thus an initial irritation of the eyes leads to the development of persistent photophobia; the man becomes the possessor of a "substitution-symptom", and the mental strain, for the time being, is relieved. Similarly, aphonia, vomiting, and the various symptoms founded on the organic lesions of Mustard Gas, form the nucleus of the symptom-complex known as Substitution - or conversion - neurosis.

Symptoms.

The great majority of functional conditions develop, as one would expect, in mild cases. The severe cases do not require a substitution-symptom. Nevertheless the after histories which I have obtained of cases evacuated to England indicate that a goodly proportion of severe cases develop symptoms of functional origin during convalescence.

In the description of functional symptoms abstract general accounts are not very satisfactory. I think I can best illustrate the condition by detailing the history of an actual case.

Functional Photophobia.

Rrivate H. was admitted on the third day after gassing.

The/

The symptoms and signs were those of a mild case - slight erythema of the back and chest, and superficial burns on the genitals. The throat was red and congested, and the patient complained of cough. The eyes showed considerable hyperaemia in the palpebral fissure but no visible lesion of the conjunctiva. Examination produced a good deal of lacrimation - There were no other manifestations. The patient was instructed, inter alia, to give up the use of the eyeshade (which had been provided at the clearing station). He was transferred to the convalescent ward.

Three days later all physical signs of gassing had disappeared, with the exception of a superficial scab on the scrotum. The patient declared himself to be well, except for watering of the eyes, and evening headache which he attributed to the effects of sunlight. At the end of a week - the normal period of a mild case - he was called up for discharge to convalescent camp. He paraded, an object of utter misery, wearing an eyeshade of his own manufacture. Examination showed intense blepharospasm, and forcible separation of the lids caused profuse lacrimation and complaint of shooting pains in the head. Careful inspection of the conjunctiva revealed no abnormality. The pupil reactions were normal; and vision was $^{6}/_{9}$ both eyes, the slight defect being due to lacrimation.

This condition responded readily to vigorous treatment - untreated cases may persist indefinitely, and indeed the irritation/

irritation produced by rubbing etc., may ultimately give rise to a true chronic conjunctivitis. The subsequent history of this case will be discussed later.

Rixon and Matthew[4] remark that a patient who has "discovered" a substitution symptom is generally cheerful, as he has found a means of escape from the mental conflict. This is only partly true, however, for, as in the case quoted, the discovered symptom may itself give rise to a great deal of distress. This point is helpful in differentiating neurosis from malingering. Similarly, the "Effort Syndrome" which not uncommonly accompanies anxiety neurosis may on its own account cause real suffering and apprehension.

Second in order of frequency comes the condition of aphonia. This gives rise to little subjective distress when uncomplicated, but in most cases it is associated with severe night-cough. The patient's chief complaint is loss of sleep. Laryngoscopic examination reveals no lesion of the vocal cords, but congestion of the pharynx persists in consequence of the cough. Functional vomiting is a sequel of the severer type of case: all the members of this group gave a history of initial vomiting, and 10 of the 16 cases showed pyrexia of more than a week's duration, due no doubt to a mild degree of Bronchitis. An important characteristic of this condition, from the diagnostic point of view, is that the vomiting does not proceed uninterr-:uptedly from the onset of gassing. The acute vomiting, as

in/

in the cases of great severity, dies down after the first few days, and then, after a more or less prolonged interval, the functional condition gradually develops.

Case 86, Cpl.G., was admitted on the 4th day of disease. A note on the Field Medical card stated "Vomiting; eyes; some throat." Examination on admission showed a case of moderate severity with conjunctivitis, skin lesions, and Bronchitis. Two days later the patient began to complain of gastric symptoms - slight nausea and discomfort after food. On the eighth day there was some retching, and the following day he vomited his whole meal after dinner. On the tenth day all food was at once rejected. He was then placed upon fluid diet, which served only to aggravate his condition. This state of affairs continued intermittently, on every variety of diet, until the 18th day.

In another case the vomiting was not fully developed until the 22nd. day: it persisted until the 36th day. In both these cases a rapid cure was effected by means of suggestion, after all dietary measures had failed.

Other less common functional manifestations were observed in gassed cases, but their actual relation to gas poisoning was not fully demonstrated. One interesting case, however, occurred in which severe burns of the shins and the dorsum of both feet were associated with hysterical paralysis of the legs. (Case 33 appendix).

"Effort/

"Effort Syndrome".

Incidence: Dr. Wilson observed in the course of his work as officer in charge of the Medical Division, that a number of Mustard Gas convalescents were being detained in hospital beyond the normal period on account of the symptoms of fatigue, breathlessness on exertion, and palpitation amounting in some cases to a definite "Effort Syndrome". On this account we decided to determine the exercise tolerance of a series of 100 consecutive convalescents. In the first week of convalescence we found that as many as 30 per cent gave an exaggerated response to effort, but this number was rapidly reduced by the institution of graduated exercises. In only 19 cases the symptoms of "effort syndrome" persisted in spite of treatment. Of these, 9 dated their symptoms from a previous infection (Trench Fever - 7, Rheumatic Fever 1, and pre-war infection 1), 4 cases had been previously in hospital, labelled"Neurasthenia", and only 5 attributed their symptoms directly to Mustard Gas Poisoning.

Much has been written, on the one side and the other, concerning the frequency of the "Effort Syndrome" after Mustard Gas. Dr. Wilson, after a full examination of the evidence, comes to this conclusion: "the "Effort-Syndrome" - so common after phosgene poisoning and in other war diseases - is very rare in Mustard Gas Poisoning; when it does occur, a previous infection is much more likely to be the causal factor than any effect due

to/

to Mustard Gas" - (5)

I should like to add this qualification, that in men already predisposed by fatigue, infection, or strain, Mustard Gas may act as the exciting cause of this neurosis; although more frequently it brings about the onset of functional conditions more intimately related to its characteristic organic effects.

Sequelae: The end-results of these cases were on the whole encouraging. Only 1% were returned from convalescent camps on account of relapse, but I have little doubt that in a much larger proportion the old mental conflict was renewed when the soldier returned to the front again; that his resistance was definitely lowered by the temporary breakdown for which Mustard Gas was responsible; and that sooner or later he became unfit for service with the expeditionary force. I have complete records of one case only, the case of photophobia already quoted. His symptoms I had cured in hospital, and after some weeks at a Convalescent Camp he was sent up the line once more. Three months after gassing I received a note from him : " . . . .you will be surprised (sic!) to learn that I have been sent down to the base again. I had been losing a lot of sleep at nights, and during the marches I was so short of breath that I could not keep up with the platoon . . . . This camp is quite pleasant, but I sleep no better. Sometimes

there/

there are air-raids, and every night I lie awake thinking I hear them coming . . . " I was certainly not surprised to find that his next letter, a month later, was dated from England. One might call this patient a pre-disposed neuropath, yet he served at the Front for two years prior to gassing, without one admission to hospital.

----------oOo----------

Notes and References.

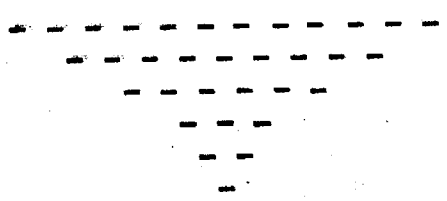
(1) McDougall, "Social Psychology".

(2) McDougall, "Psychology" (Home University Library).

(3) I use these terms in the sense defined by Rivers - "Instinct and the Unconscious".

(4) Rixon and Matthew, "Anxiety Neurosis".

(5) Quarterly Journal of Medicine, Jan. 1920.



T R E A T M E N T.

TREATMENT.

(I) Preventive Measures:-

When a Mustard Gas casualty occurs, much can be done by early prophylaxis to mitigate the severity of the case. The patient should be given a complete change of clothing, and the moister parts of his body, particularly the genital region, the axillae and the flexures, should be carefully rubbed down with cotton wool or lint damped with 5% Dakin's solution. The nose should be irrigated, the mouth cleansed, and the throat gargled with a mild solution of hypochlorite. All measures of protection from air - oils, ointments and close-fitting dressings are absolutely contra-indicated. The eyes should be irrigated gently with either a solution of equal parts of Boric acid and soda Bi-carbonate, or preferably, as Warthin recommends, (1) by a 5% Solution of dichloramine -T.

(II) Therapeutic Measures:

(i) Ocular lesions: in mild cases the treatment indicated above should be continued. In cases of purulent conjunctivitis without corneal injury the eyelids should be carefully drawn apart, and the conjunctival sac thoroughly cleansed every four hours with the dichloramine -T. solution. I do not recommend the use of paraffin, or castor oil, and I have found no good results from silver preparations. Where the cornea is damaged the pupil should be kept moderately dilated by atropin in/

in the form of an ointment, and the four-hourly cleansing continued. In chronic Blepharitis I have found yellow oxide of Mercury ointment useful, and in persistent conjunctival irritability cold bathing and cold compresses have been of value. The eyes should never be bandaged; a simple brown paper shade suspended from a forehead tape is sufficient for all purposes. In milder cases, the use of any form of eyeshade should be discontinued at the earliest possible moment, in view of the tendency of many patients to develop functional conditions.

(ii) The Nasopharynx: I have found in weak Dakin's solution the most reliable remedy. It should be used freely as mouth wash and gargle. If used early and in a thorough manner, it is, I believe, very effective in minimising the chiances of late infection of the bronchi and lungs.

I know of no specific treatment for laryngeal involvement, but inhalations through steam of menthol and compound tincture of Behzoin were of value in relieving pain.

(iii) Cutaneous Lesions: Uninjured skin, and parts where the injury is no more severe than erythema, should be kept thoroughly dry and covered with a bland dusting powder.

For all degrees of skin involvement more severe than this, early or late, sterile and infected, Dakin's solution may be regarded as an almost specific remedy. The affected surfaces should be bathed every four hours for a period of 15-20 minutes with/

with the solution; in the intervals they lightly covered with lint soaked in weak boric acid or normal saline. Where possible a bath should be employed (e.g. a sitz-bath for the genitals); otherwise gentle irrigation through a fine nozzle will serve the purpose fairly well. The genitals should be supported, not by a sling, but on a broad bandage which passes over the thighs and is secured behind. Surgical cleanliness is necessary in the dressing of all skin lesions. Vesicles should be punctured and evacuated before coagulation takes place. A bedcradle and a water-mattress are necessary in all severe cases. Great care must be taken to avoid any rough handling of the skin, and the epithelium should be jealously preserved except when it serves to prevent the escape of septic discharges.

The only general measures of importance are the vigorous treatment of shock, and the prevention of undue acidity and concentration of the urine. For the latter condition fluids should be forced, and sodium bicarbonate given internally, in quantities which depend upon the reaction of the urine.

(iv) Lesions of the Respiratory Tract.

It is unnecessary to deal with this subject in detail. No specific treatment is known. If the nature of the infecting organism can be discovered special measures may be directed against it. Otherwise the general therapeutic measures employed/

employed in secondary broncho-pneumonia are indicated.

(v) Functional Conditions:

The successful treatment of the substitution-symptom depends on the recognition of the fact that it is only an outward symbol of the disease. In many cases the removal of the photophobia, or the aphonia, as the case may be, serves only to render manifest a latent repression-neurosis. Rivers (2) rightly insists that the cure of the neurosis is of primary importance; for in many cases the symptom disappears with the improvement in the mental condition. Unfortunately this method of treatment was, in France at least, impracticable. The environment essential for the successful healing of neurosis is to be found, not in hospitals, but in convalescent camps. There the patients mingle with healthy men, join in games, follow agreeable occupations which divert the mind from brooding memories; there too, a cheerful spirit prevails, and the free un-fearful talk of warfare draws from the patient's mind its most guarded secrets. The danger of hospitalization is a very real one, and nothing can be worse for the neurotic convalescent, than constant communication with the seriously sick and wounded, and - no less important - those whose destination is England. In spite of this, however, men could not be discharged from hospital so long as photophobia, aphonia, vomiting, etc., was likely to interfere with their ordinary duties at convalescent camps, - so the order of treatment/

treatment had to be reversed.

The following methods are recommended :-

(i) Photophobia: Prophylaxis is most important. Unless a man shows a definite purulent conjunctivitis, the eyeshade should be at once discarded. The patient is encouraged in every way to use his eyes, principally by means of organised games in the open air. In fully developed cases cold face plunges, cold packs to the eyes, and sterner disciplinary measures were of great value.

(ii) Aphonia yielded readily to treatment, and relapses were rare. The routine method was a process of re-education and suggestion emphasized by the application of the faradic current. 95% of the patients were cured in three applications.

(iii) Vomiting was the most intractable of all the functional conditions, and required the close personal attention of the physician. The influence of suggestion plays a large part in the treatment, and each case must be studied individually. The strong suggestion that a certain carefully selected diet is bound to cure is always helpful. The actual nature of the diet is not very important, but it should be peculiar, dry, and not too pleasant. The real success of the cure depends on the skill and tact of the attendant nurse.

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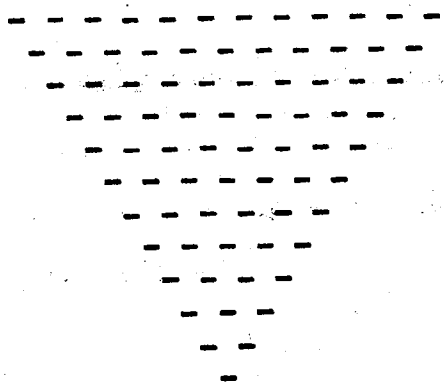
Notes and References.

(1) Warthin: The Medical Aspects of Mustard Gas Poisoning.

(2) Rivers: "Instinct and the Unconscious."



APPENDIX.

The following abstract of clinical notes has been compiled for the purpose of illustrating the principal types of lesion described in the text. Space does not permit of more than the barest outline of a small number of cases; but similar types have been correlated as far as possible, and additional charts introduced for comparison.

For the sake of brevity severe cutaneous lesions have been represented diagrammatically, the hatched areas indicating severe burns with necrosis of the epidermis; the dotted areas, simple erythema.

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THE MEDICAL SUPPLY ASSOCIATION.

DISEASE.

Notes of Case.

Name

Age

Diet

Case Book Nos. 1, 2, and 3.

Subcutaneous

CASE 1. Burns and shock.

CASE 2. Oedema and acute inflammatory reaction in lungs. Burns not severe.

CASE 3. Severe Burns and shock.

Date of admission.

Result

Time. 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

Bowels.

Urine.

Temperature (Fahrenheit)

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

Normal Temperat. of body.

I II III

42° 41° 40° 39° 38° 37° 36° 55°

Temperature (Centigrade)

Day of Dis	1		2		3			1		2		3			1	2		3	
Pulse	68	104	80	108	132			76	90	140		150				138	156	136	
Resp.		24	10	28	40	52			24	50		50				44	52	60	
Date.																			

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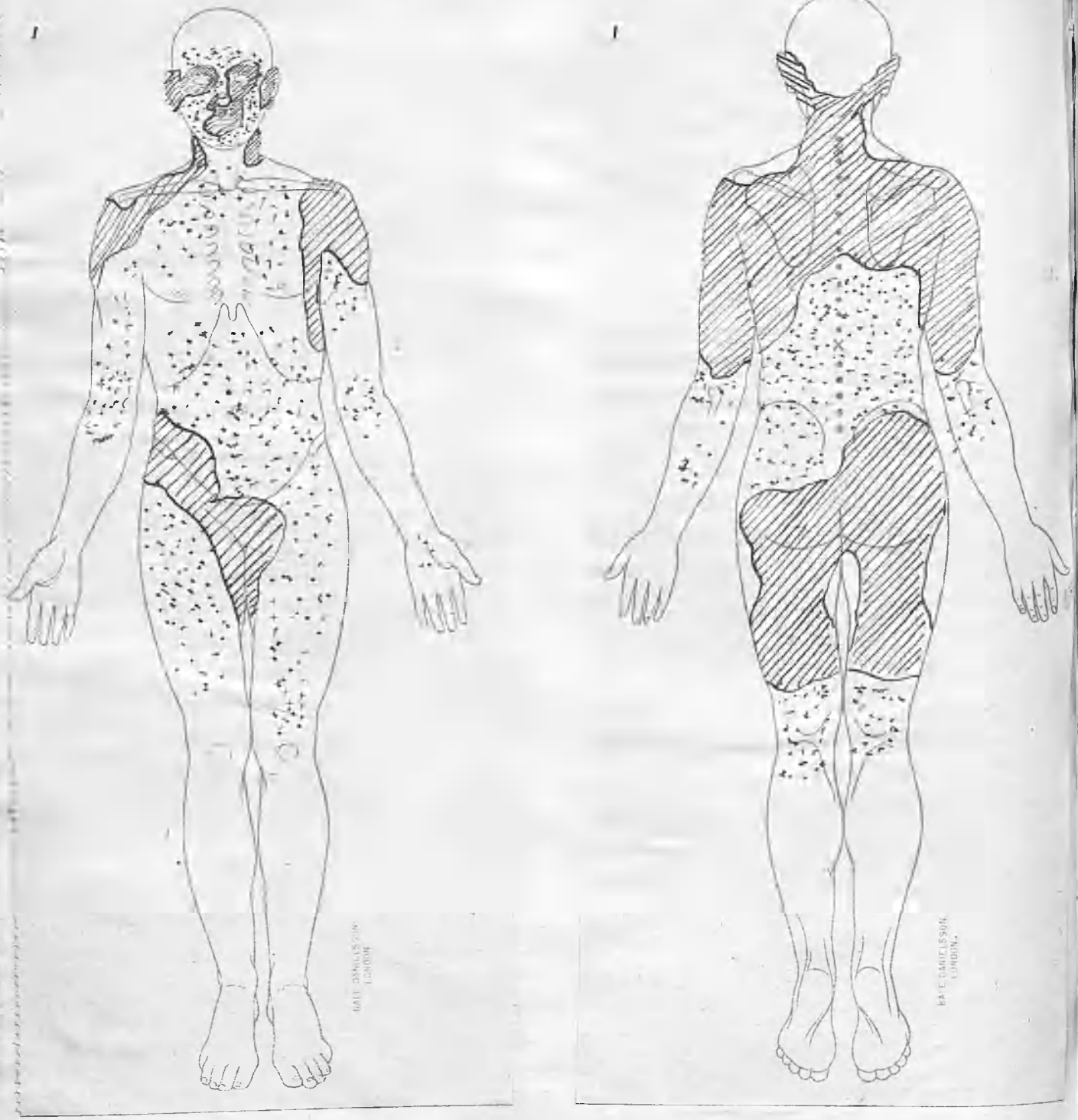


Diagram of Skin Lesions — CASE: 1.

Note: Hatched areas represent second degree burns.
Dotted " " simple erythema.

GROUP A : FULMINATING TYPE.

Case 1: Gnr. D. Gassed 9th June at 3 a.m.

1st. day Wounded and partly buried by a shell. Three hours later sudden onset of pain in the eyes and intense heat of the skin, followed almost immediately by severe and prolonged vomiting. Clothing changed at Dressing Station. Admitted at 10 p.m. Vomiting and attacks of faintness and giddiness continued at short intervals. General erythema. Short husky cough. Mental condition normal.

2nd. day: Vomiting, at first food, and later bile-stained fluid. Cyanosis, slight in the morning, deepened during the day, and towards evening respirations began to rise. Chest signs: poor expansion, especially of right side. Impaired note, weak breath sounds and small moist râles at bases. Small patch of dulness and bronchial breathing in R. upper axilla. Heart max. impulse 5½ inches to left of midsternum. No bruits. Action weak and irregular. Mental condition normal at night. Blisters began to appear in the morning, and successive crops came out during the day. Large areas of skin cyanotic. Urine normal.

3rd. day: Conscious and rational till 11 a.m. Vomiting continued. No urgent dyspnoea, but failing pulse. Died at 1.30 p.m. (58½ hours).

Post-Mortem: Skin lesions as in diagram. Pharynx, larynx and trachea deeply congested without ulceration or membrane. Old dense adhesions R. apex, but pleurae otherwise negative. Lungs bulky and oedematous. On section exude frothy, blood-stained fluid but no pus. No signs of consolidation but lower third of R. upper lobe dark, airless and collapsed. Stomach deeply congested with ecchymoses along lesser curvature. No signs of erosion. No marked changes in other organs.

Case 2. Pte. H. Gassed 9th August.

1st. day: Early history not known. C.C.S. note says "Colour good: much mucus from nose."

2nd. day: On admission deeply cyanosed. Jaw dropped and breathing stertorous. Answers rationally when roused but too ill to give history. General erythema but severe burns only on genitals. Chest: Resonance impaired at bases, where there are diminished breath sounds and moist râles. Lower intercostal spaces sucked in with inspiration. Heart normal in size and position but sounds weak. Urine normal.

3rd. day Black cyanosis; rapid shallow respirations without obstructive signs. Unconscious from 10 a.m. Died at 3 p.m.

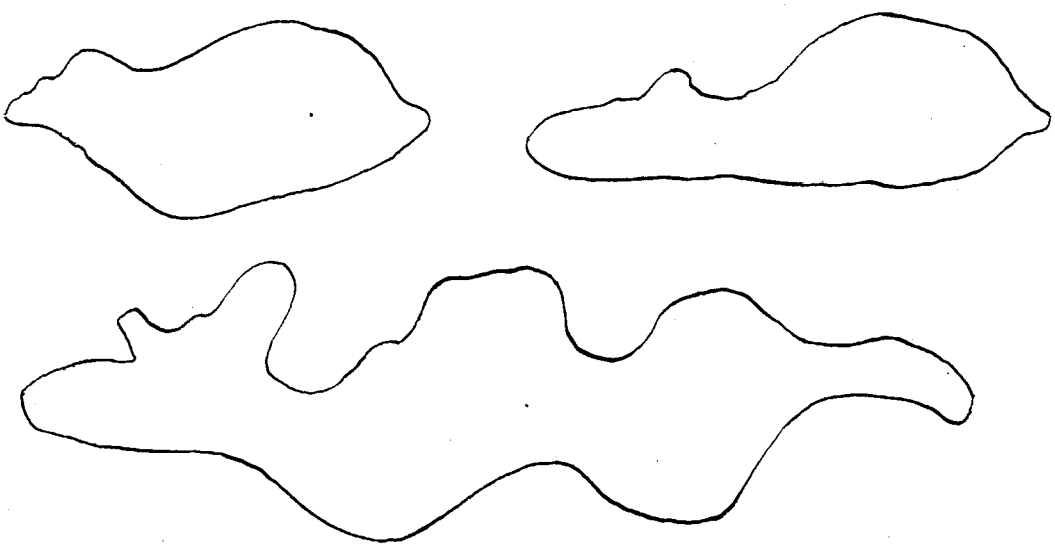
Post/

Post-mortem: Eyes show brawny white oedema in palpebral fissure; severe burns on genitals but nil elsewhere. Pharynx moderately injected, larynx and trachea deep purple in colour, but no ulceration or membrane. Lungs moderately oedematous with marginal emphysema. On section ooze dark blood and serous fluid - no pus. No signs of consolidation.

Microscopically "acute capillary bronchitis of irregular distribution, with, in a few areas, early consolidation around the smaller bronchi. Very few organisms seen."

Case 3: Pte. T. Gassed 9th August at 4 a.m.

1st day: Gassed while asleep and well-covered with blankets - consequently no burns except of face and eyes. Awoke with acute pain in eyes. Very slight nausea and vomiting.
2nd day: On admission deeply cyanosed; shallow heaving respirations, but no signs of throat obstruction. Signs of oedema at bases, but otherwise normal chest. Mentality normal. No albumin in urine.
3rd day: Spasmodic dyspnoea in the morning relieved by inhalations. Coughed up three large tracheal casts - actual size.



Chest signs: Sucking in of lower ribs. Breath sounds heard quite clearly through gurgling in trachea and bronchi. No dulness or bronchial breathing anywhere. Died at 11 p.m. (66 hours).

Post-mortem: Thick white membrane covering larynx and trachea throughout their length. (earliest case of pseudo-membrane formation) the underlying surface showing scattered areas of excoriation. Pleurae negative. Lungs bulky and oedematous. Microscopically: Acute widespread capillary bronchitis. In some areas much alveolar oedema. Exudate hardly purulent. Some fibrin and haemorrhage.

DISEASE.

Notes of Case.

Name

Age

Diet

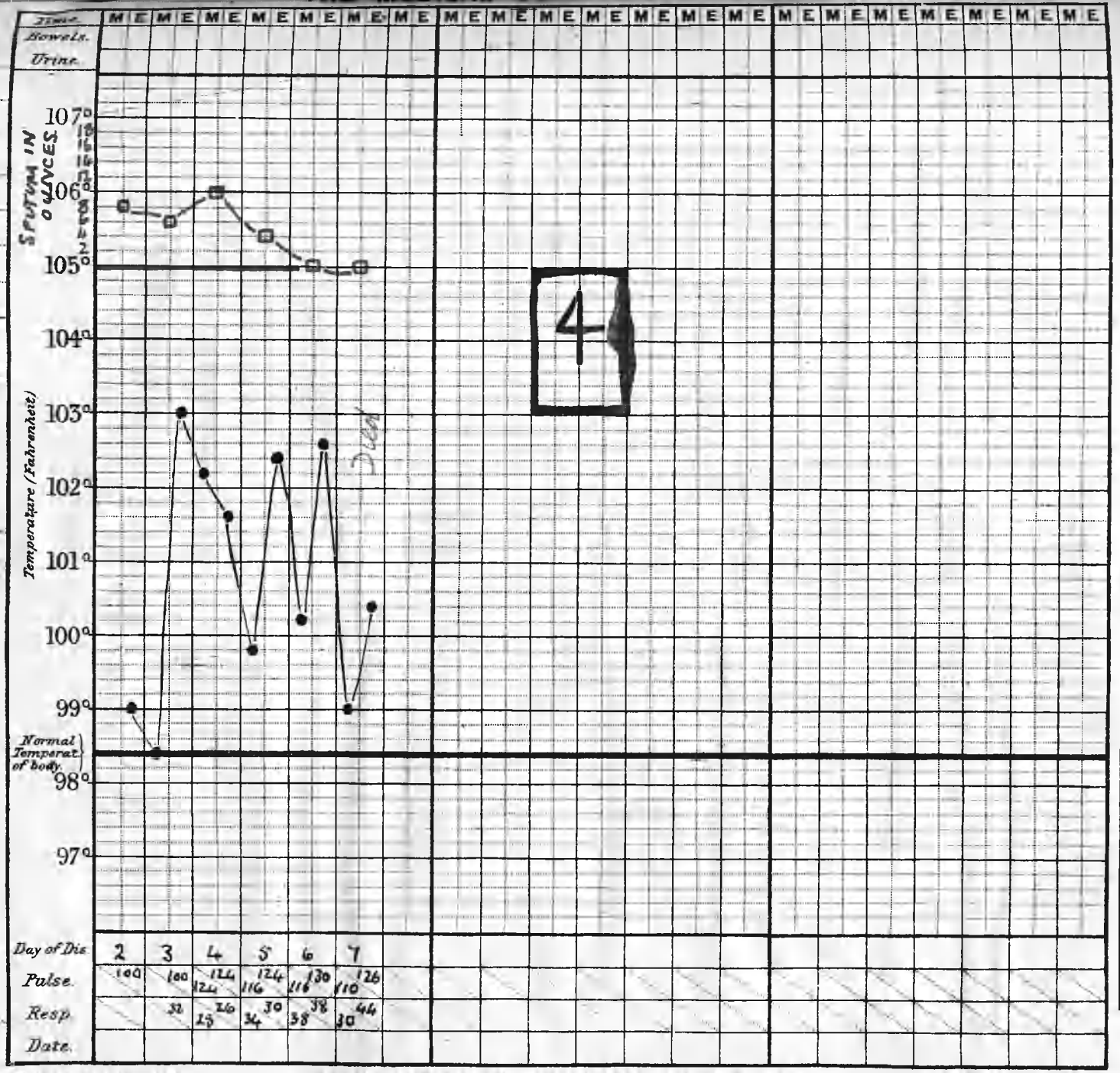
Case Book N° 4

Bronchi Pneumonia

NOTE: Progressive fall in sputum daily total in spite of good general condition of [illegible]

Date of admission.

Result



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Case 4: Pte G.

Gassed 2nd August, at 8 a.m.
Latent period 3 hours.
Died 9th August at 7.30 a.m. (8th day)

History: Sudden onset with pain in eyes, burning of skin and severe vomiting. Stimulation required at Field Ambulance.

2nd day: Very severe conjunctivitis and extensive burns - No chest trouble.

3rd day: Very flushed: breathing deeply and easily. Chest signs of extensive bronchitis - no dulness. A little thick yellow sputum.

4th day: Improving: acute purulent bronchitis: no dulness or alteration of breath sounds. Urine normal. Bringing up more sputum.

5th day: General condition good in spite of drowning with fluid. Front and back of chest harsh noisy bronchitic sounds: bases resonant.

6th day: Burns reacting well: very clean. Seems to be gradually filling up with fluid. Urine - a trace of albumin.

7th day: Attacks of severe tightness across chest and more urgent dyspnoea. Pulse weakening. Mental condition unclouded. At 8 p.m. wrote a clear, well-constructed letter home - within 12 hours of death. No signs of consolidation were observed at any time.

Post-mortem: Skin lesions as indicated in diagram. Pharynx, larynx, and trachea intensely congested with patches of ulceration. Fairly extensive false membrane extending from the vocal cords as far as the thyroid.

Lungs: Right covered with recent soft adhesions, only the diaphragmatic surface being free. On section upper and lower lobes dark and engorged with blood: middle lobe practically normal. Left: No pleural adhesions: changes not so marked. no macroscopic evidence of consolidation, and all sizeable portions of lungs floated in water. No marked changes in other organs.

Microscopically: "Widespread capillary Bronchitis with fibrino-purulent plugs, and commencing discrete bronchopneumonia. In some areas slight oedema: in others (right) semi confluent bronchopneumonia with much haemorrhagic and fibrinous exudate; probable commencing necrosis around some bronchioles."

Case 5: Pte L. is similar to the above, and showed a very remarkable absence of toxaemia. Within two hours of death he asked me to tell him why his eyelids and feet were twitching. Albumin appeared in the urine on the 5th day. In this case severe skin lesions were confined to the genitals. There were no/

DISEASE.

Notes of Case.

Name

Age

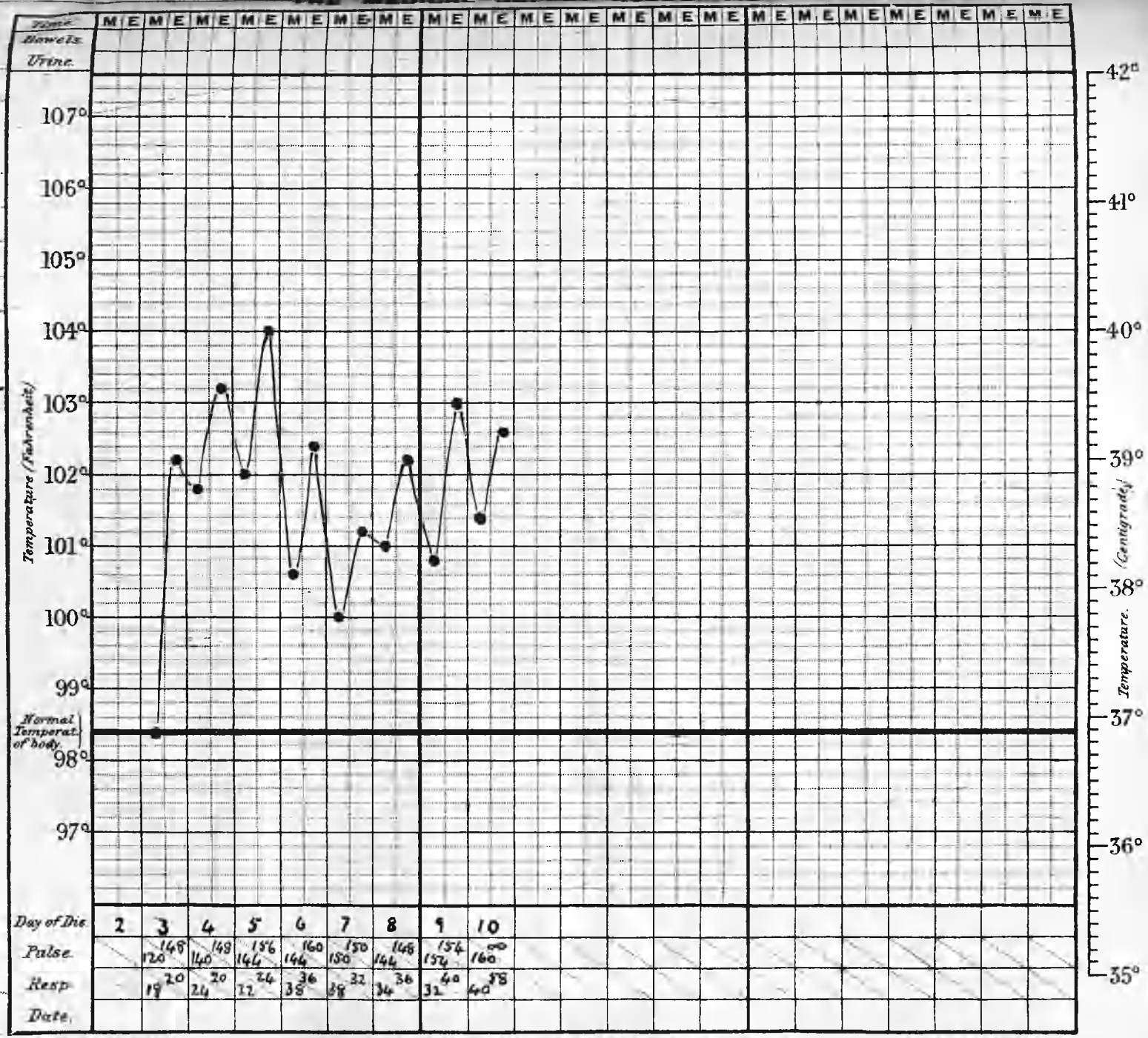
Diet

Case Book N° 5

Clinical Capillary Bronchitis. Striking rapidity of pulse in spite of mild degree of burns and absence of toxaemia.

Date of admission.

Result Death 10th day.



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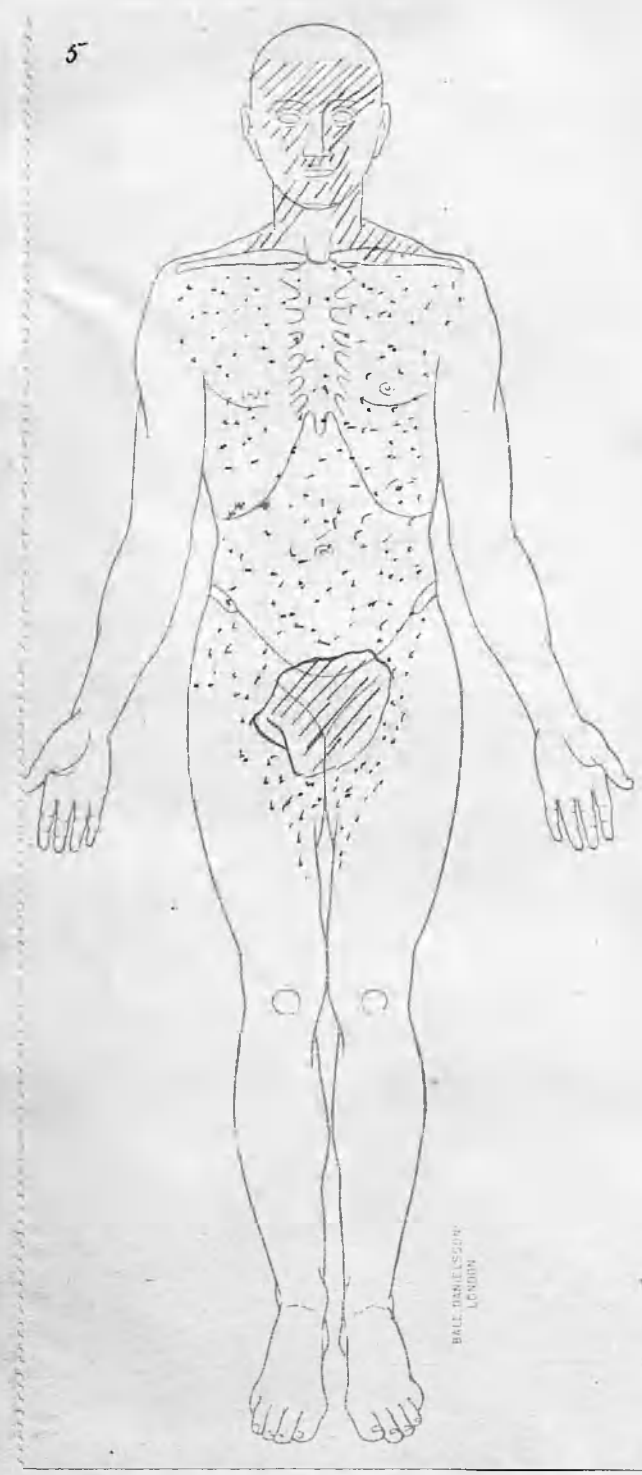
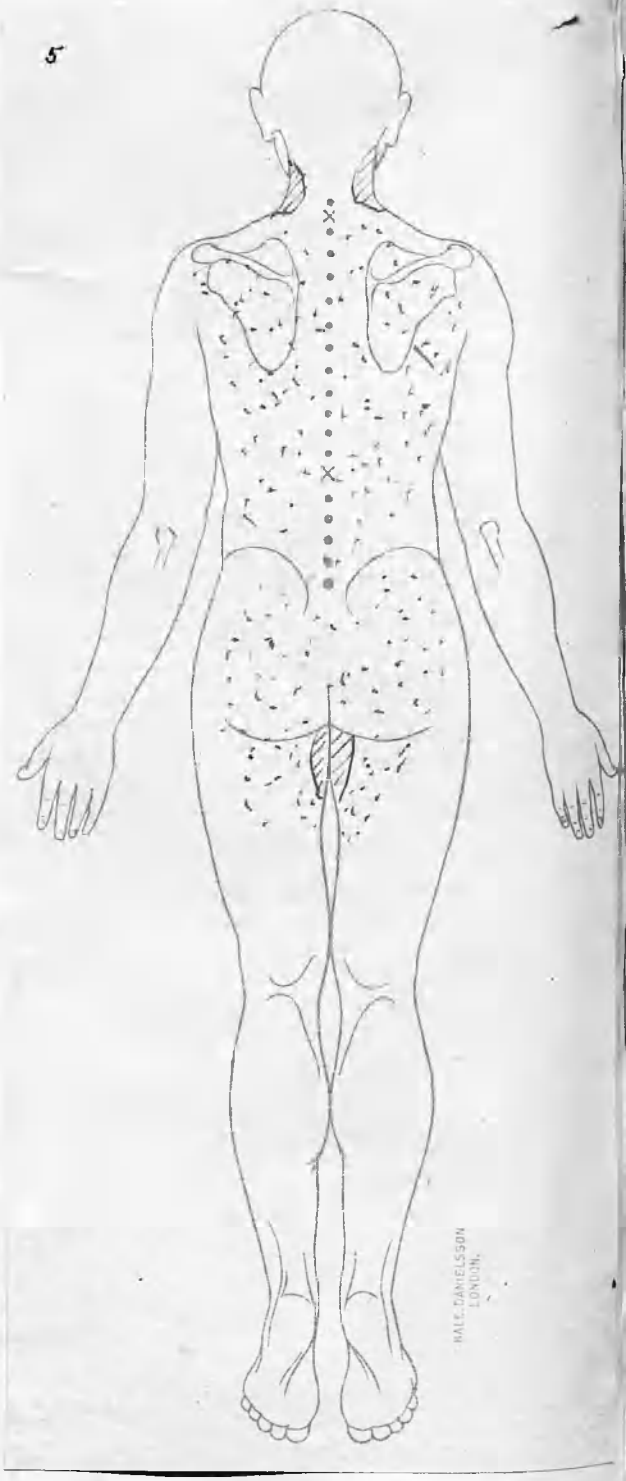




Burns Chart - Case 5.

no signs of colsolidation during life, but widespread lobular bronchopneumonia was proved at post mortem. The pulse was remarkably rapid throughout, but there were no signs of cardiac dilatation, or failure until shortly before death.

Case 6 Cpl. C. is an even more acute case of this type. This man was very severely burnt. Our clinical note (on the 5th day) will serve to illustrate his condition.
5th day: Typical mustard case. Moist bubbling sounds (tubes full of fluid) all over back, most marked up to level of 3 inches up the vertebral border of scapula, when breath sounds become suddenly clearer and purer. One inch below left nipple is a patch of crepitations coming up under the stetho-scope. There (he volunteered) he had pain on breathing.

Post-mortem: Sizeable portions of both lungs floated in water, but numerous tiny nodules could be seen arranged around the smaller bronchi . . . . .
Microscopically "Acute Bronchopneumonia with large areas consolidated. Exudate largely haemorrhagic and purulent."

From this intermediate type we pass on to the "outspoken bronchopneumonia". Only two fatal cases of this type were recorded, but this was by far the commonest variety in the milder cases.

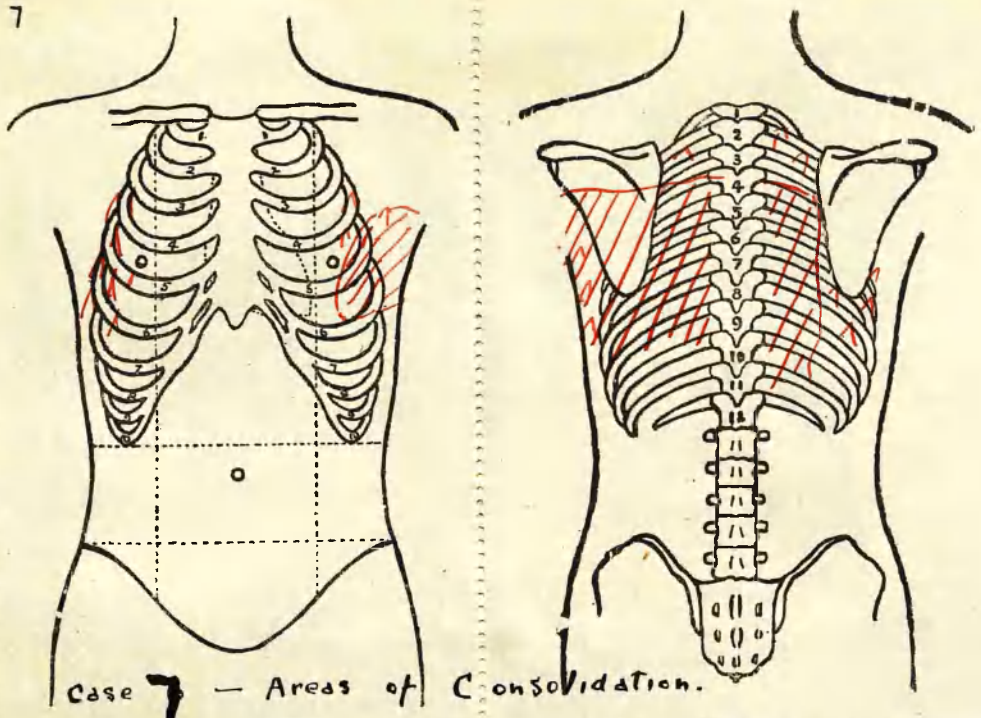
Case 7. Cpl. S.

Gassed 6th September at 3.0 a.m.
Latent period 4 hours.
Died 12th September at 9.0 p.m.

History: etc. Sudden onset with nausea and vomiting from which he recovered during the first day. Improved greatly until admission on the third day when he began to feel short of breath and tight across the chest.
4th day: Mental condition normal. Short of breath but no actual dyspnoea. Chest: no adventitious sounds over shaded areas but only dulness and loud bronchial breathing. Both axillae full of squeaking ronchi. Burns not severe.
5th day: much improved; no longer breathless on sitting up. Signs as above.
6th and 7th days: Deep cyanosis: engorgement of right heart. Respirations shallow and heaving . . . . .

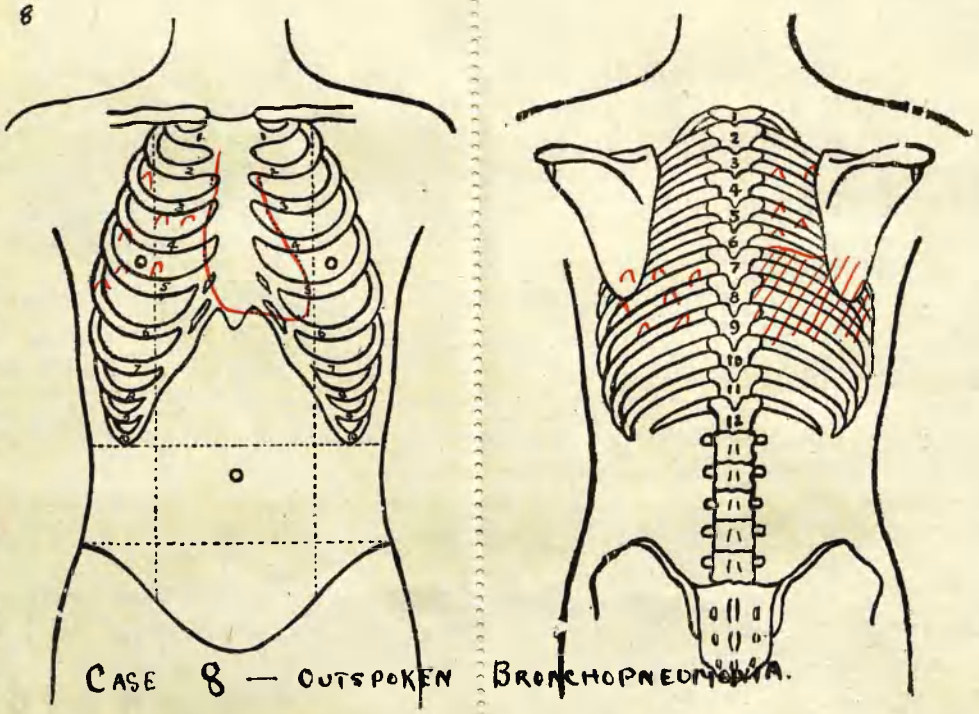
Post-mortem: Massive consolidation of left lower lobe; elsewhere scattered patches of bronchopneumonia. R. middle lobe congested only.
Microscopically "advanced confluent bronchopneumonia. Exudate chiefly purulent, especially around bronchioles - from which the/

7



Case 7 — Areas of Consolidation.

8



Case 8 — Outspoken Bronchopneumonia.

THE MEDICAL SUPPLY ASSOCIATION

DISEASE.

Notes of Case.

Name

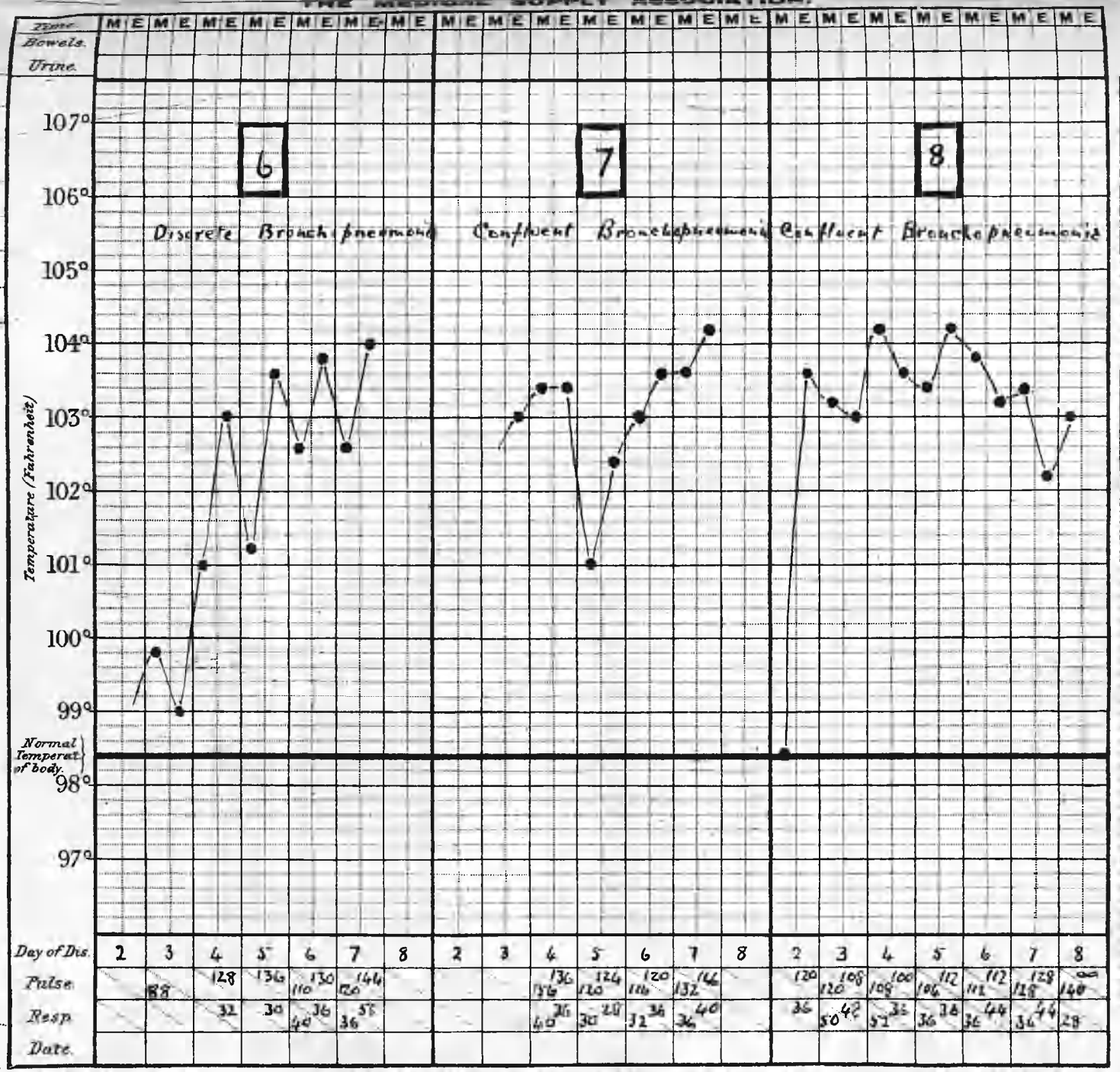
Age

Diet

Case Book N° 6, 7, 8

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

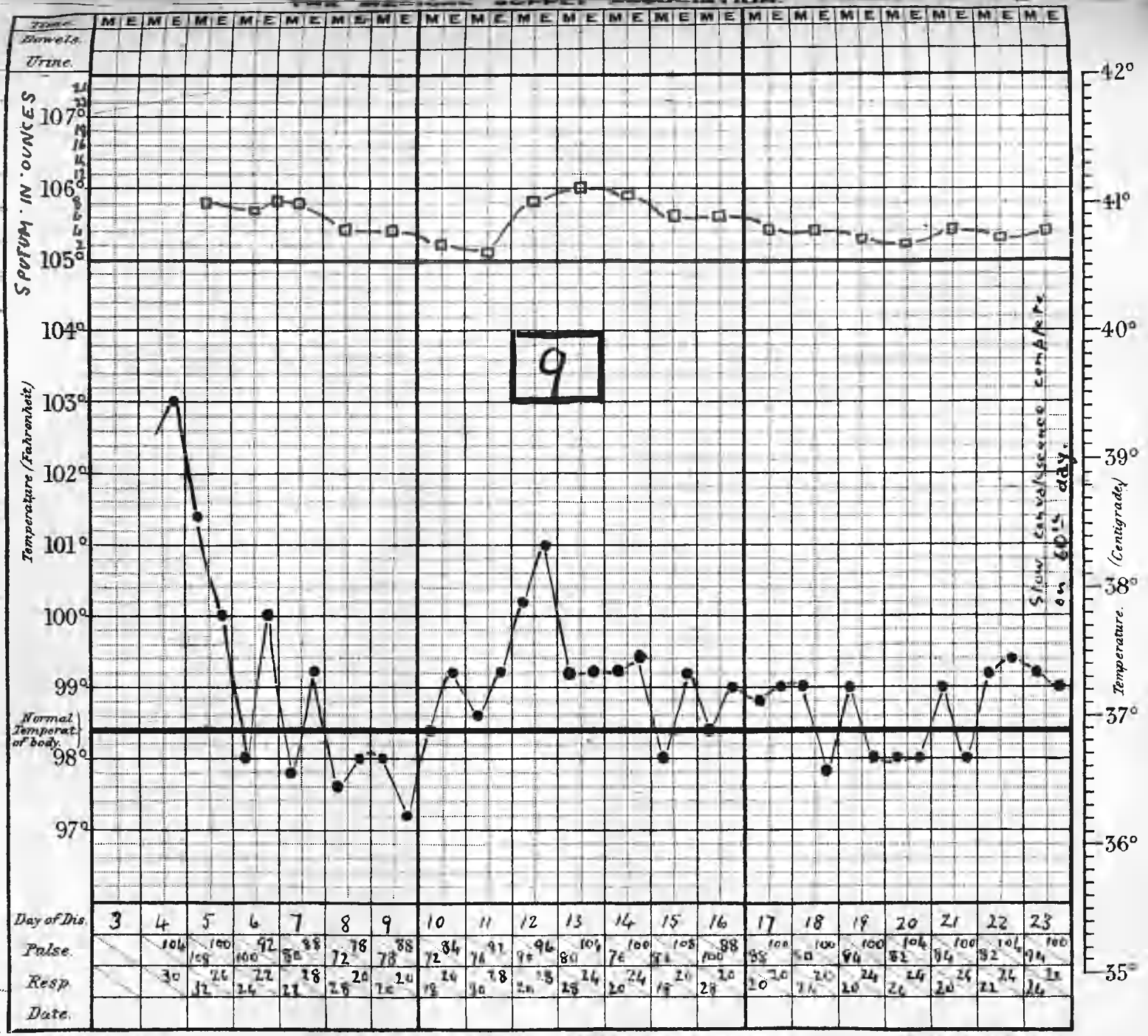
Diet

Case Book No. 9

Sputum curve rises with secondary rise in temperature. c/f cases 16 + 24.

Date of admission.

Result



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the process originates."

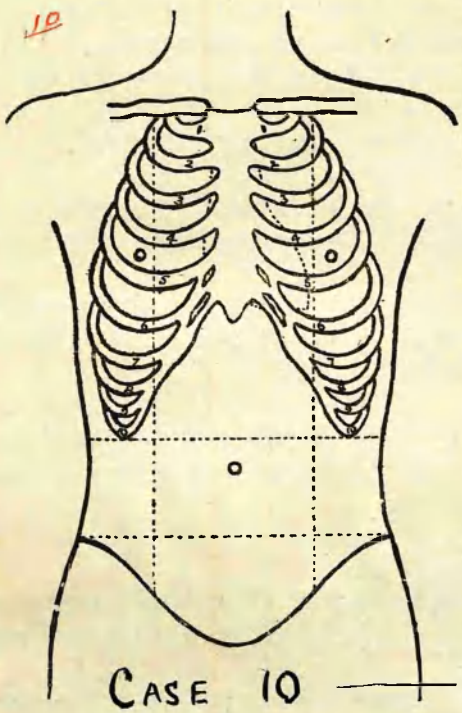
Case 8: Pte. T. is a more acute example of the same type.
2nd. day: Complains of stifling in the chest and headache. Slight cyanosis but very marked dyspnoea dyspnoea. Signs of generalized acute Bronchitis with early consolidation R. base.
3rd. day: Sudden diminution in the amount of sputum. Areas of bronchial breathing at both bases.
4th and 5th days: Rapidly extending consolidation especially of right base. Restless and talking incessantly, but answers rationally. No albuminuria.
6th to 8th days: Continuous noisy delirium; refuses all food. Increasing cyanosis and dyspnoea. Physical signs indistinguishable from double lobar pneumonia.

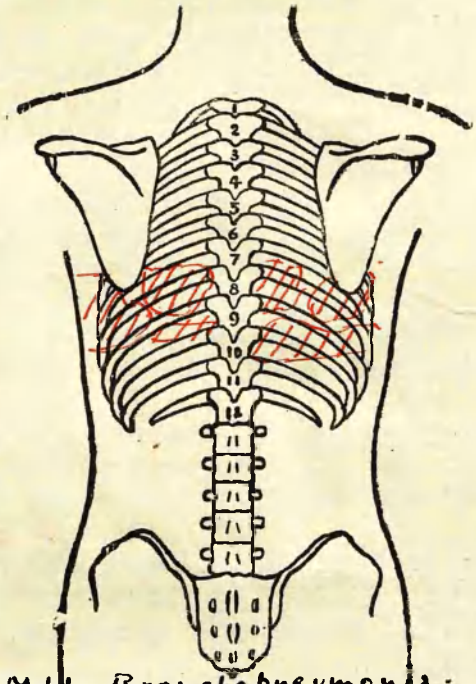
Post-mortem: Burns not severe. Massive consolidation both lower lobes. Microscopically confluent Bronchopneumonia with early necrosis of bronchial walls.

Case 9: L/C. M. Gassed 31st July.
4th day: On admission slight cyanosis; pulse and respirations raised. Moderate dyspnoea. No complaint of pain except in throat.
Chest signs. Mild generalized bronchitis; chest resonant throughout except at right base where there is dulness, with diminished breath sounds and fine inspiratory crepitations.
5th and 6th days: General condition, pulse and temperature improving but no change in signs. Dulness as above but no bronchial breathing.
7th day: Band of definite dulness and bronchial breathing (as shown in diagram). All adventitious sounds in chest have disappeared.
8th day: Slight upward extension of dull area, but otherwise no change. Complaint of pain on breathing - in right side.
9th - 13th day: No change in signs and no improvement in patient's general condition. Slight evening delirium, insomnia and restlessness.
13th to 15th day: Signs unchanged but gradual general improvement. Less restless and takes food better.
17th day: Band of dulness as before, but air is beginning to re-enter alveoli. Close to spine coarse "redux" crepitations have appeared, but towards axilla the bronchial breathing and bronchophony is as well-marked as ever.
20th day: Redux crepitations have extended all over dull area. Patient is very thin and wasted, but is now beginning to take food normally.
26th day: Chest gradually clearing, but still cough and several ounces of sputum daily.

Note: I have followed the progress of the physical signs of this/

10





CASE 10 — Mild Bronchopneumonia.

THE MEDICAL SUPPLY ASSOCIATION.

DISEASE.

Notes of Case.

Name

Age

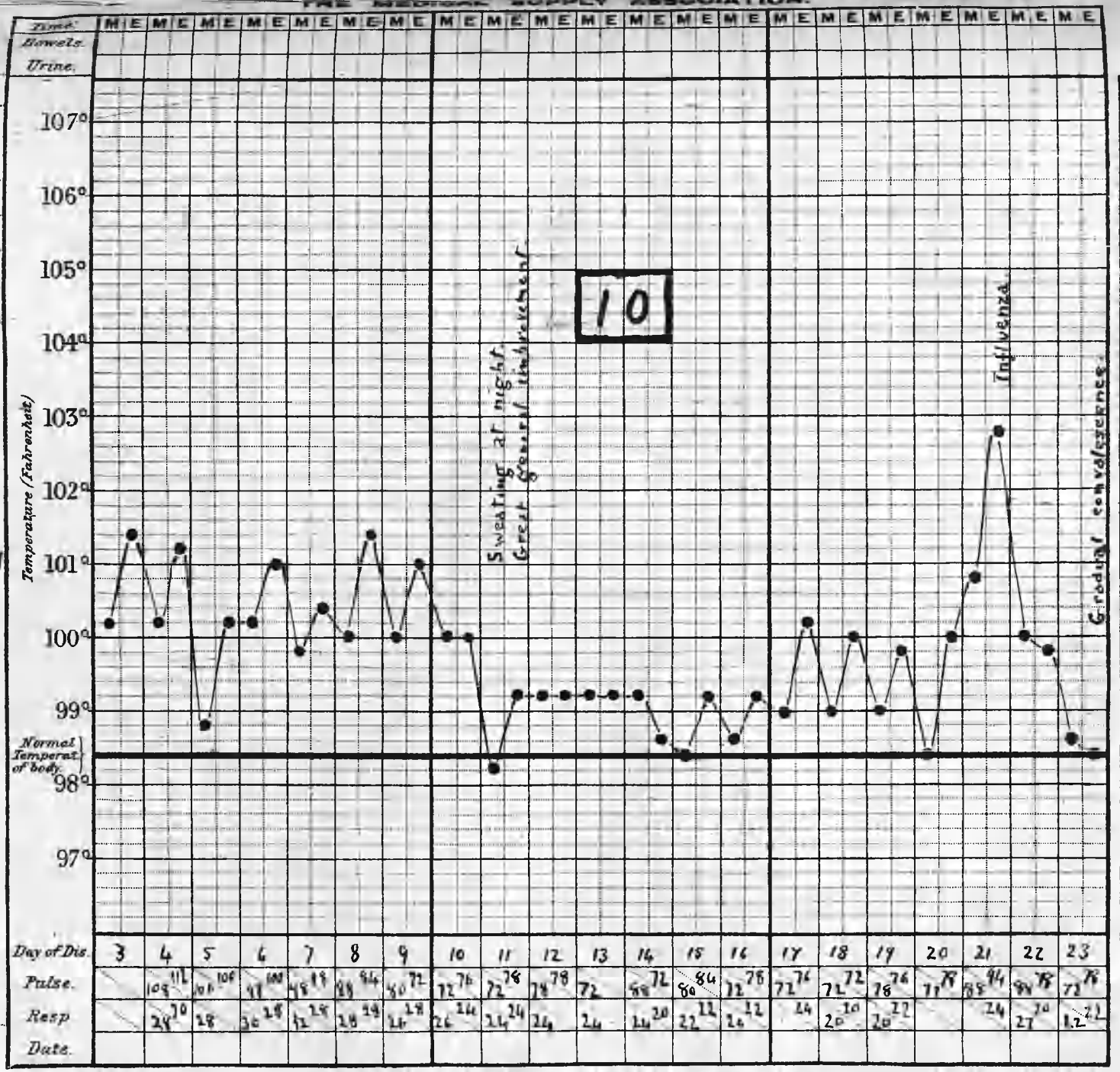
Diet

Case Book No. 10

Fairly typical mild Mustard Gas Pneumonia with completion of acute stage on 11th day. Persistence of dulness and bronchial breathing at both bases (unaffected by attack of Influenza) until 30th day.

Date of admission.

Result



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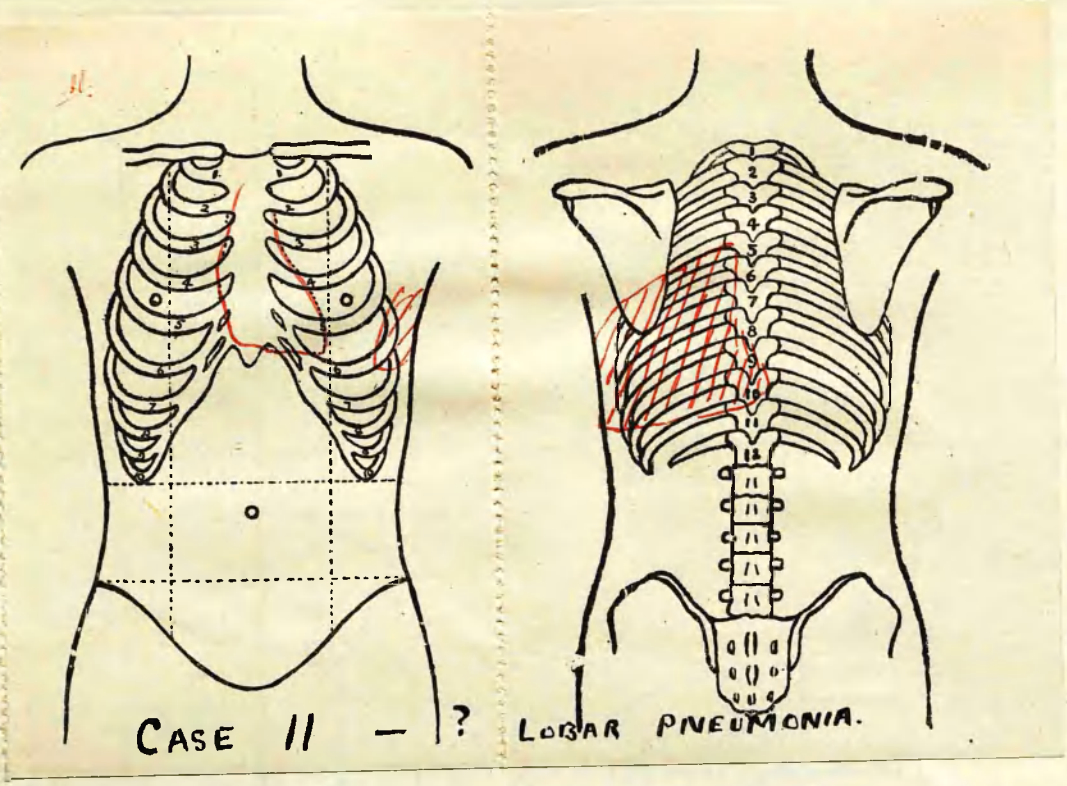

CASE II — ? LOBAR PNEUMONIA.

THE MEDICAL SUPPLY ASSOCIATION.

DISEASE.

Notes of Case.

Name

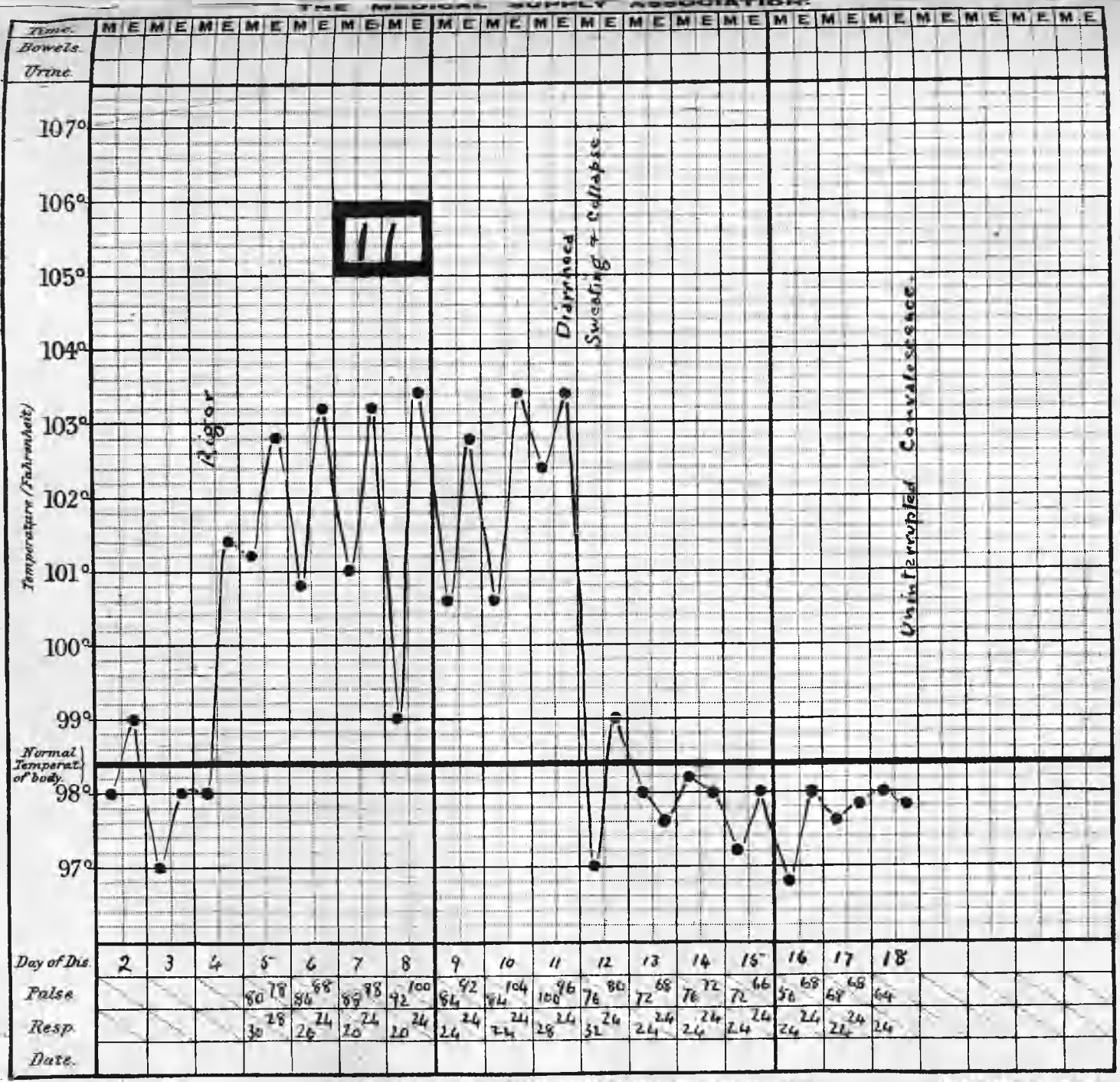
Age

Diet

Case Book N° 11

Date of admission.

Result



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this case somewhat fully, because this is a typical example of moderately severe Mustard Gas Poisoning. The special features are (a) the slow evolution and persistence of the chest signs; (b) wasting and loss of strength out of all proportion to the "mild" picture presented by the temperature chart (c) Persistent cough with sputum after all physical signs have disappeared. This type of case is so common in Gas Poisoning, that I have four more examples to represent the condition. Two of these (cases 10, and 12) are almost identical With the one already quoted, and details of their history are unnecessary. I have attached their charts for comparison. The other two (cases26 and 27) show a similar condition in a much more severe degree, while the fatal cases 29 and 30 probably give the clue to the real nature of the condition - a chronic bronchopneumonia caused by an organism of low virulence, foci of infection being provided in the damaged bronchioles.

Case 11, however is atypical, and very rare in Mustard Gas Poisoning. It resembles closely a mild lobar pneumonia of civil life:

3rd. day: Slight cyanosis: torpid condition. No signs in chest except diminished breath sounds at left base near spine. Definite rigor at night with rise of respiration. full, bounding pulse.

4th day: Slight diarrhoea and foul tongue. Leucocytosis 30,000. Note over left base impaired, and area of bronchial breathing close to spine.

6th day: Dulness and intense bronchial breathing over whole left base. Patient cyanosed and looks acutely ill. Yet respirations not greatly increased.

11th day: (i.e. 8th day of pneumonia) Dulness and bronchial breathing over whole left back except extreme apex. Typical pneumonic crisis at night, with severe diarrhoea and collapse.

13th day: Characteristic crepitus redux left back. No further rise of temperature; uninterrupted convalescence.

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DISEASE.

Notes of Case.

Name

Age

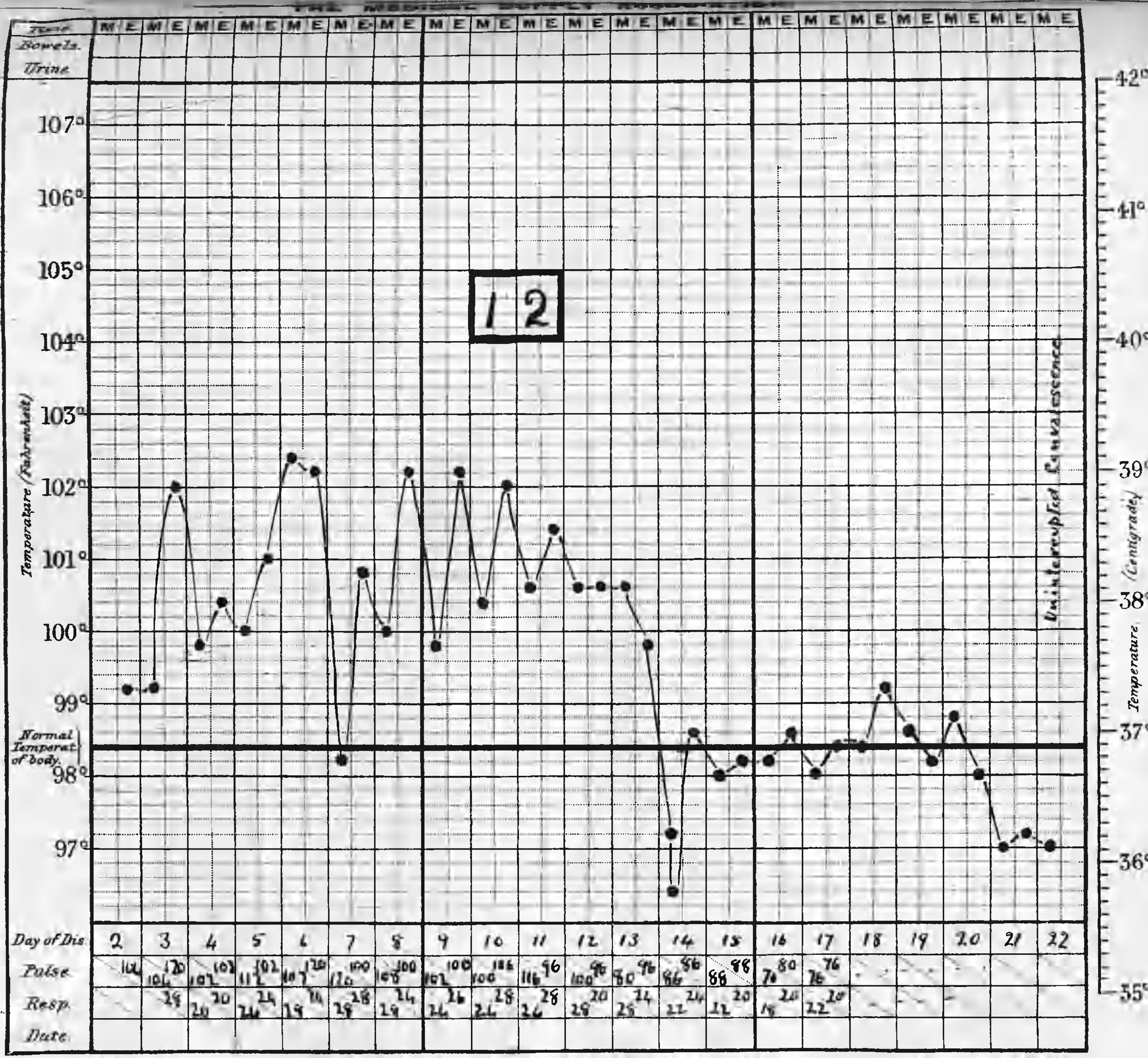
Diet

Case Book No 12

Case of pseudo-lobar broncho-pneumonia with well-marked crisis on 13/14th day.
No skin lesions
Physical signs in chest (R. base) persisted till evacuation on 30th day.

Date of admission.

Result



GROUP C: RECRUDESCENCE:

This large group is represented here by four cases: in all the initial bronchopneumonia was of the type already described. About the 11th day or so the temperature fell and pulse and respirations showed a corresponding improvement. The daily sputum total, however, remained high. After an apyrexial period of varying length new respiratory symptoms suddenly developed - always ushered in by pleural pain, and never by general catarrhal signs. The most typical example is case 16, in which the recrudescence was marked by the onset of a definite, localized pleurisy which gradually subsided without extensive pneumonic involvement. In the more severe (and rarer) cases (13 & 14) the initial pleurisy was followed by an acute pseudo-lobar pneumonia, ending by crisis. The differential diagnosis from Influenza is not difficult, and indeed several of these cases subsequently developed a typical Influenzal attack (Case 15).

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THE MEDICAL SUPPLY ASSOCIATION.

DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No. 13(i)

Onset with pain in eyes and vomiting 6 hrs. after exposure. On admission sev. conjunctivitis and burns on back.

Chest: 4th to 15th days = patchy consolidation Right Base. Left base clear.

15th to 21st day = great improvement in general condition and clearing of Right Base.

21st day: Sudden onset of fever + pain L. chest.

Date of admission.

Result

13(i)

Time: 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

Bowels.

Urine.

Temperature (Fahrenheit): 107° 106° 105° 104° 103° 102° 101° 100° 99° Normal Temperat. of body. 98° 97°

Temperature (Centigrade): 42° 41° 40° 39° 38° 37° 36° 35°

Day of Dis.	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
Pulse.	60			96				88 84	88 98	72 84	86 84	104	88 96	80 90	88	80 86	80	88	88 88	86	120 116
Resp.	20			30				24 32	24 28	24	18	28	28 32	20 20	24	20 24	24	24	24 24	24	32 32
Date.																					

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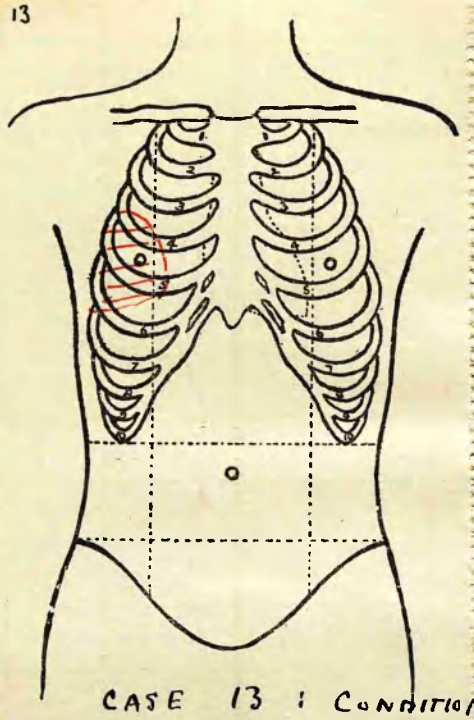
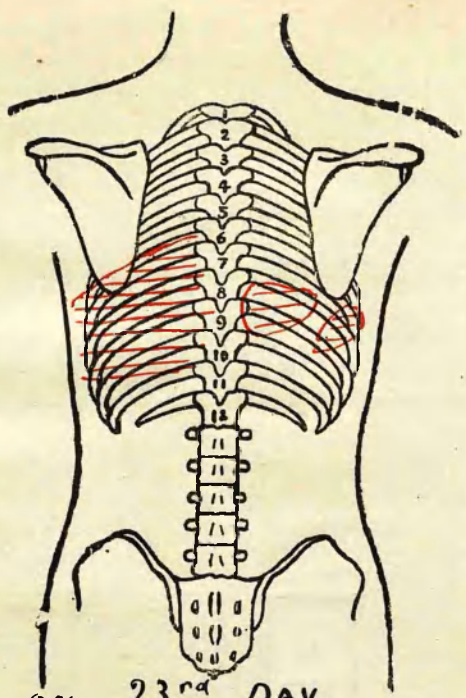
13





CASE 13 : CONDITION ON 23rd DAY.

THE MEDICAL SUPPLY ASSOCIATION.

DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No. 13 (ii)

22nd – 31st day

Massive consolidation
Left Base with
Pleuritic friction
L. Axilla.

31st day —
Crisis and rapid
convalescence.

Date of admission

Result

13 (ii)

Time.	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
Bowels.																																
Urine.																																

Temperature (Fahrenheit)

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

Normal Temperat. of body.

42° 41° 40° 39° 38° 37° 36° 35°

Temperature. (Centigrade)

Day of Dis.	23	24	25	26	27	28	29	30	31	32	33	34	35	36
Pulse.	96 100	84 92	96 104	104 108	104 108	118 120	120 120	112 104	88 90	90 92	80 90	100 94	84 70	64 82
Resp	32 28	22 24	24 26	24 28	24 26	44 30	40 30	36 34	30 32	28 34	40 24	24 28	30 20	20
Date.														

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DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No. 14

Broncho-pneumonia

3rd — 10th day.

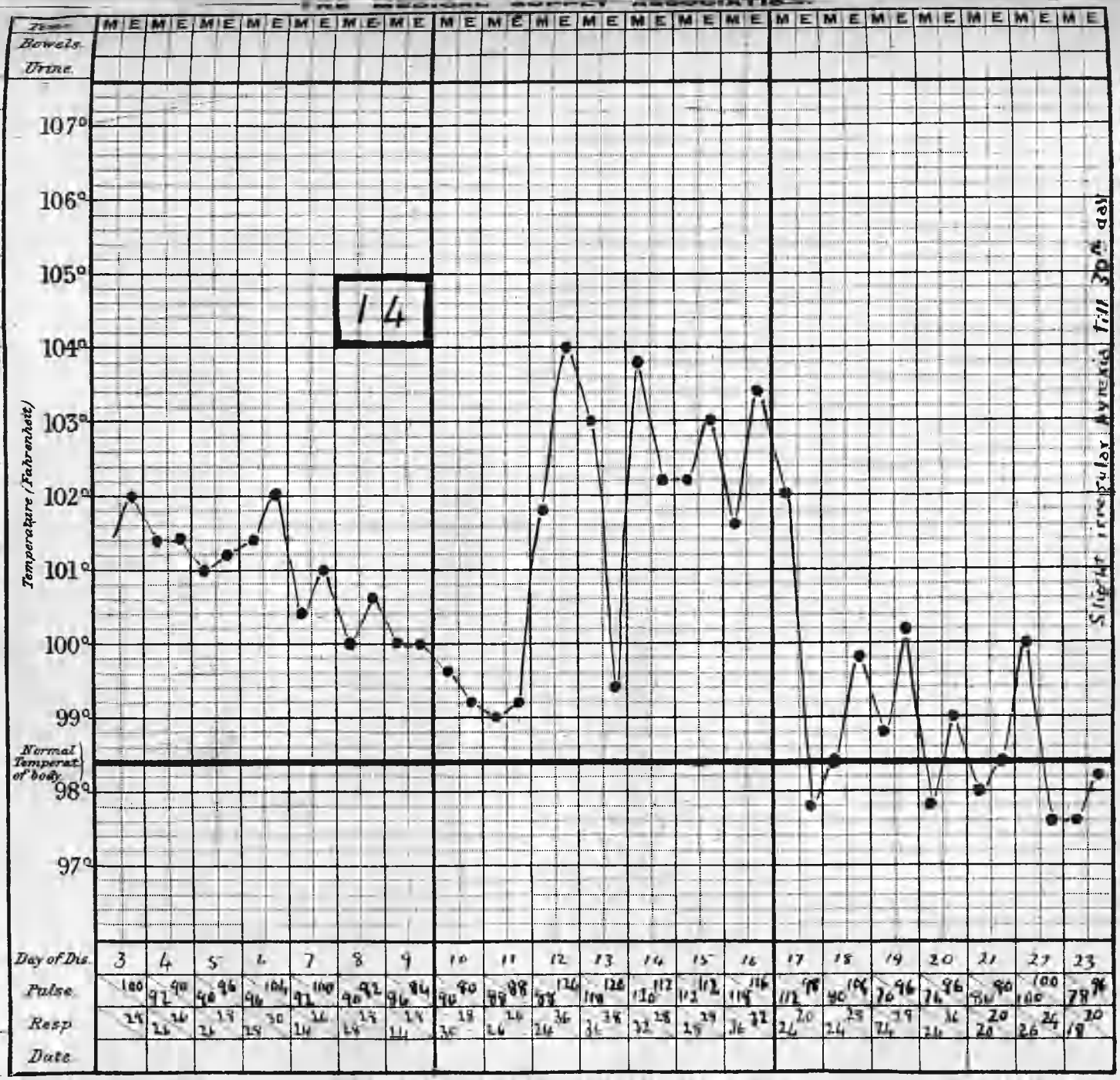
Signs of consolidation Left Base.

11th — 17th day.

Renewed access of fever with signs of massive consolidation Right Base.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No 15

4th – 10th day –
Initial mild broncho pneumonia (discrete).

10th – 27th day.
Apyrexial period.
Still cough and much Bronchitic sputum.

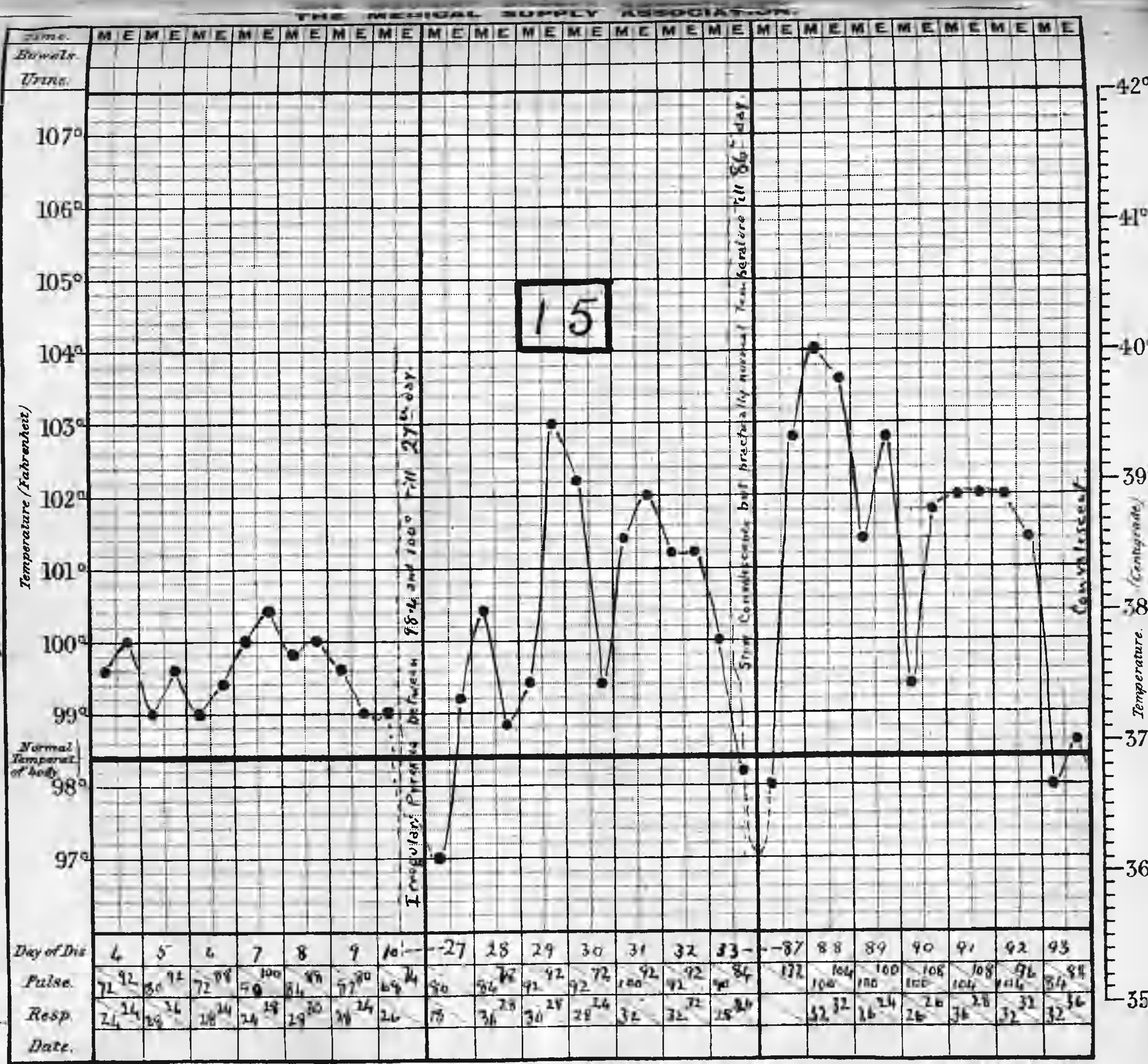
27th – 33rd day.
Recrudescence with fresh Pleurisy left side. No massive consolidation.

33rd – 85th day.
Slow but complete convalescence.

87th day
Readmitted with acute Influenzal Bronchopneumonia (Left base)

Date of admission.

Result



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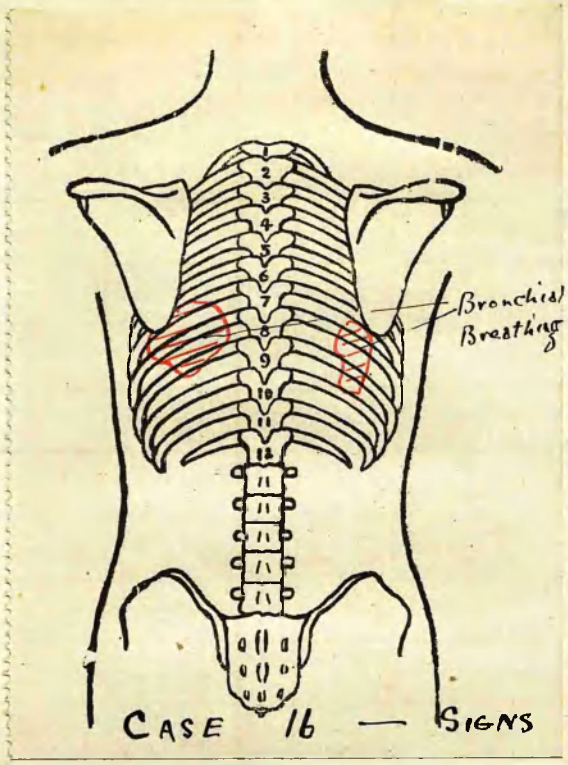
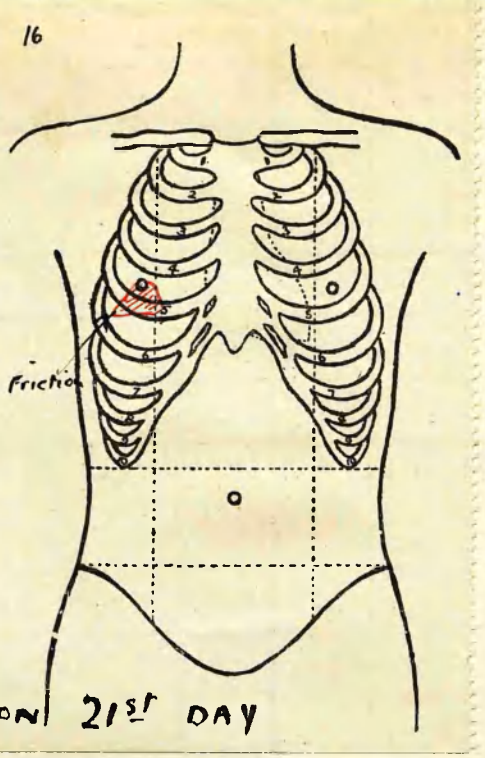


CASE 16 — SIGNS

16



ON 21st DAY

THE MEDICAL SUPPLY ASSOCIATION.

DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No. 16

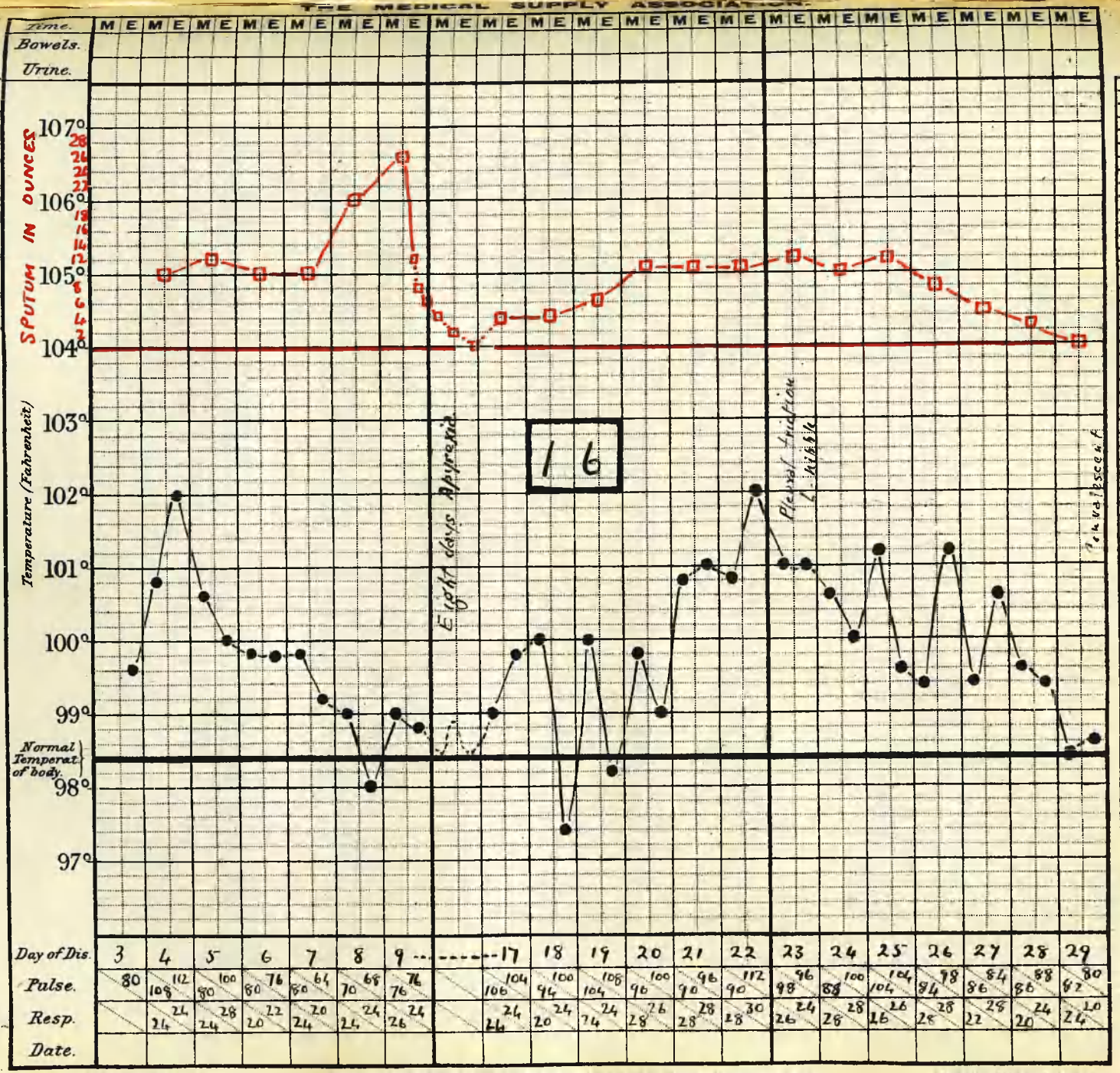
Bronchopneumonia with

Note rise in sputum total corresponding with development of fresh signs in chest.

Recrudescence

Date of admission.

Result



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GROUP D: TOXAEMIA.

Cases 17 and 18 represent very acute types of Broncho-pneumonia in which the Respiratory symptoms were early overshadowed by the development of a profound toxaemia. Sputum examination pointed to infection by a haemolytic streptococcus.

Case 17: Spl D. Gassed 25th June.

4th day: Severe conjunctivitis and 'steamy' corneae. Skin not severely burnt. Breathing not laboured. Lungs clear in front but fine crepitant râles at left base. Urine clear.

5th day: Increasing cyanosis. Breathing comfortable. Answers questions rationally, but has attacks of curious quiet 'wandering'.

6th day) Signs of consolidation gradually developing.

7th day) Sharp dry râles coming right up into stethoscope. seems to be deeply poisoned. Wandering; difficult to raise.

8th day: Deep cyanosis: Quick jerky movements of fingers and picking at bedclothes. Not bringing up any sputum . . . . .

In Case 18 the toxaemia is even more intense. As early as the 4th day our clinical note was "Torpid: hardly coughs at all: resents any movement; will not knock a fly off his face - yet gets out of bed when not closely watched."

Case 19 Pte.L. is one of many examples of the mild, apathetic toxaemia referred to in the text. On the 8th day "he has gradually sunk into a condition of extreme apathy; he retains sputum in his mouth; allows food to dribble away from the side of his mouth and will not swallow - yet he is perfectly rational when roused, and complains of no pain in his throat." It was not until the 28th day that this man rose to a state of normal interest in life.

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DISEASE.

Notes of Case

Name

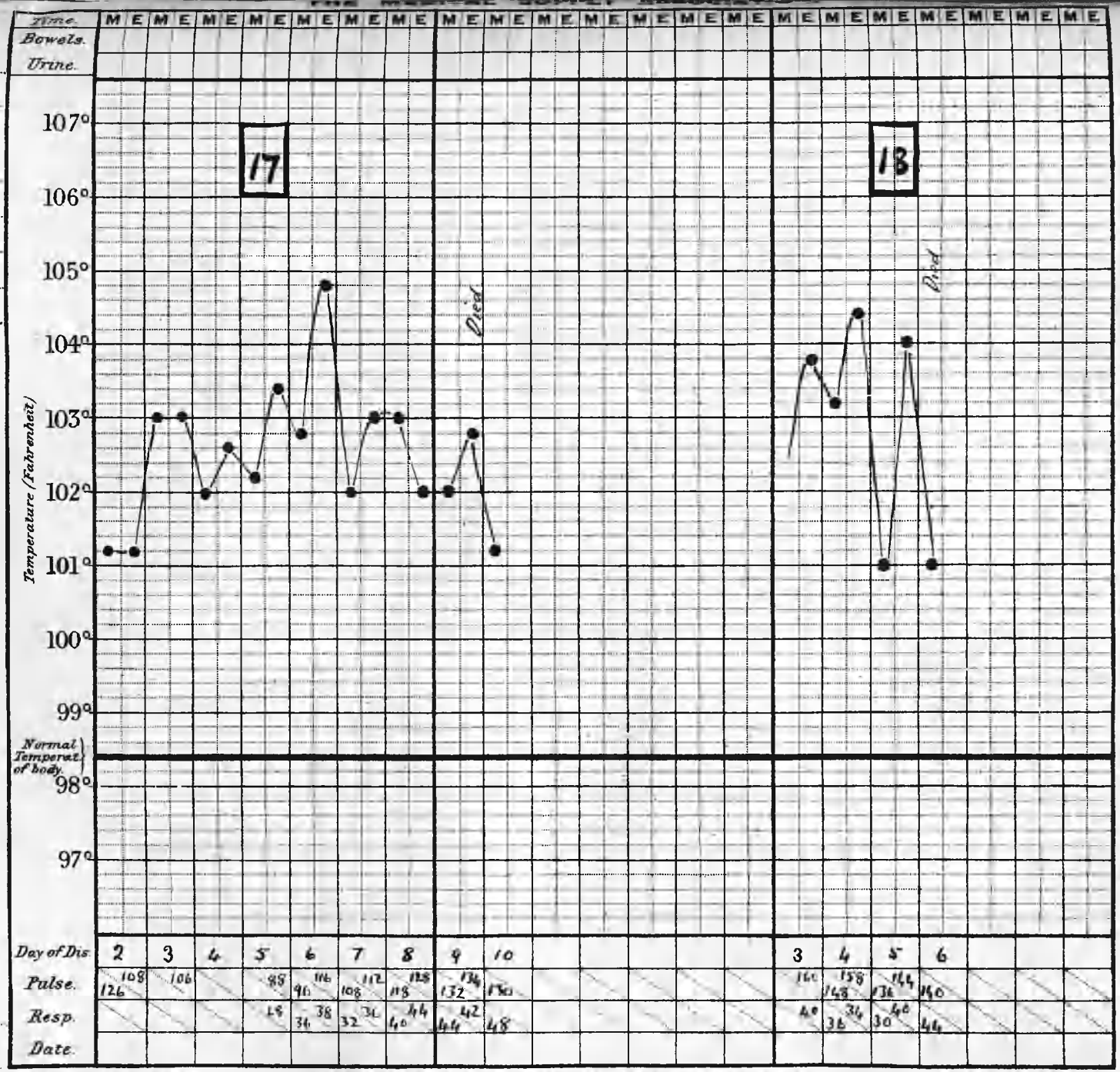
Age

Diet

Case Book N° 17
18

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

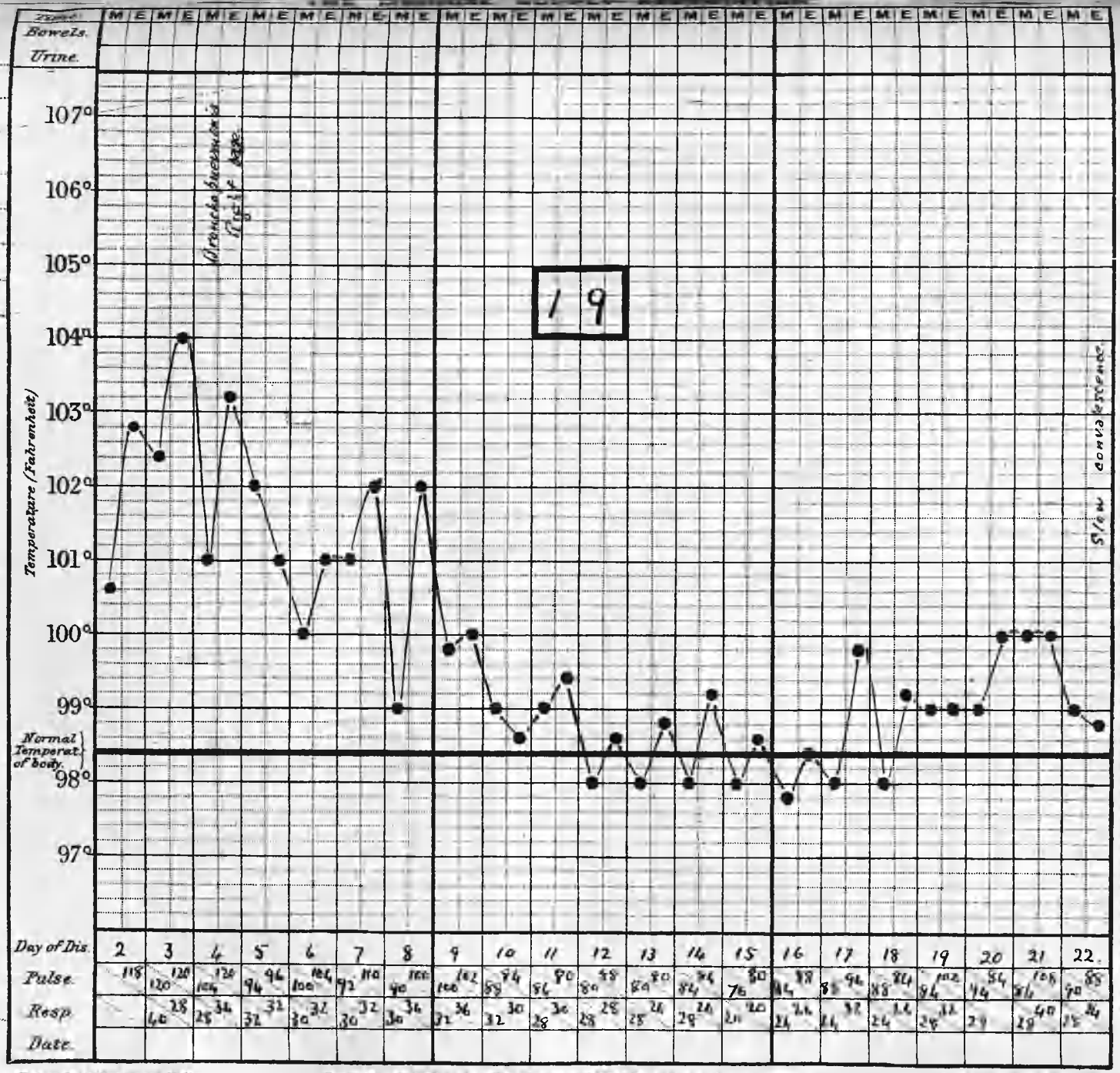
Age

Diet

Case Book N° 19

Date of admission.

Result



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GROUP E: COMPLICATIONS ETC.

Abscess of Lung.

Case 20. Pte. C: Very severe burns and eye lesions. Initial temperature crest apparently due to skin condition, as no respiratory symptoms developed until the 7th day. On the 8th day the defensive processes seemed to break down for spreading necrosis and gangrene of the skin took place. Respirations and temperature rose rapidly, and areas of dul-:ness and bronchial breathing appeared in the chest with remarkable suddenness. On the 9th day the sputum was foetid. Albumin, blood, and pus appeared in the urine.
On the tenth day there appeared to be an intense septicaemia, restlessness, great thirst, delirium and incontinence. The patient died on the 12th day.

Post-mortem: False membrane along whole larynx and trachea: Lungs: Both lower lobes bound down by recent adhesions. Left upper lobe solid and contained a single large abscess cavity (the size of a plum) filled with sanious pus. The remainder of the lungs showed extensive areas of broncho-pneumonia, several of which were grey in colour, friable and infiltrated with pus. The kidneys showed intense congestion and tubular haemorrhages.

Pneumothorax.

Case 21 L/C F.

Until the 10th day this case appeared to be a perfectly satisfactory Bronchopneumonia. In the evening (10th day) however, there was slight delirium, and during the following days the daily sputum total fell ominously. On the 15th day there was little change in the general condition but - not an uncommon event - a patch of pleural friction appeared on the left side. On the 16th dulness was noted over the right back between the 5th and 8th dorsal vertebrae, and X-ray examination demonstrated a definite shadow in this region. No symptoms appeared, however, until the morning of the 17th day when at 7.30 a.m. the patient was seized with acute pain in the right side. At 10 a.m. the classical signs of pneumothorax were present. The patient died on the 18th day.

Post-mortem: a small abscess cavity, the size of a 5/- piece was found near the apex of the right lower lobe. A similar, but less advanced necrotic patch was seen in the left upper lobe. The former abscess had suptured into the right pleura, giving rise to pneumothorax.

Empyema.

Case/

Case 24: Pte.D. In this case the usual type of broncho-pneumonia (massive consolidation left base) appeared to have come to an end by the 13th day. On the 14th day the chest was almost clear except for an area of moderate dulness and bronchial breathing at the left base. This dull area ex-:tended gradually, and signs of a fresh acute infection appeared. On the 22nd. day signs of fluid appeared at the left base; on the following day 26 ounces of slightly turbid yellow fluid were withdrawn. This fluid grew a pure culture of a haemolytic streptococcus.

Bronchiectasis:

Case 22 shows this condition in an acute form, yet during life there was little indication either from the temperature chart or from the physical signs. The latter showed a massive bronchopneumonia at the right base and scattered areas of consolidation on the left side.

Post-mortem: an abscess was found in the left upper lobe, and numerous necrotic areas were scattered throughout both lungs. The right lower lobe, however, was almost entirely replaced by dilated, ulcerated bronchi. The pus from the abscess and the fluid in the bronchiectatic area gave pure cultures of staphylococcus aureus.

Case 29 is an example of a much more chronic type - also staphylococcal. This case shows a severe initial broncho-pneumonia with a definite remission between the 14th and 24th days. From the 25th day chest symptoms and signs at the left base began to develop again. On the 30th day there was acute pain in the left side and a few days later a rub near left nipple was detected. On the 36th day the left base was very dull, with diminished breath sounds and vocal resonance. The left chest was explored but only an ounce of blood-stained fluid withdrawn. This fluid grew a pure culture of staphylococcus aureus. From the 42nd. day the sputum was offensive, and elastic tissue was present.

Post-mortem: a very extensive saccular bronchiectasis was found throughout the left lower lobe. In both lungs elsewhere a good deal of necrosis around the bronchi.

Cases 26, 27 and 28 are severe examples of the same type. In all these cases there was great wasting and loss of strength. The sputum at one time was very purulent and had a character-:istic "post-mortem room" odour which we learned to associate with pulmonary necrosis. After-histories obtained from these cases seem to indicate that all three were suffering from chronic bronchiectasis.

Case/

Case 25 is a good illustration of the abrupt transition from a bronchopneumonia of the ordinary type to an acute process of pulmonary necrosis. Until the 9th day the patient's general condition was good, and his appetite normal. From that day onwards loss of weight and strength developed with extraordinary rapidity. At death on the 15th day the body was wasted to a skeleton.

Post-mortem extensive areas of necrosis were found throughout both lungs. Pure cultures of a haemolytic streptococcus were obtained from these necrotic areas.

Case 30 is a striking example of the acute self-limited pneumonia followed by a late secondary infection - an initial bronchopneumonia lasting twelve days followed by eight days of general improvement. On the 23rd. day a secondary rise of temperature occurs with signs of pleurisy and a fresh area of consolidation. There are no catarrhal or general symptoms associated with the recrudescence.

Inoculation experiments and cultures taken from the lung post mortem give a growth of streptococcus haemolyticus. At autopsy the lungs show evidence of two distinct processes of different dates - an acute bronchopneumonia (generalized) and a chronic fibrous pneumonia confined to the left lower lobe.

Gangrene of the lung.

Case 23: Gassed 15th May.

2nd. day: Severe burns: solid white oedema in palpebral fissure with steamy corneae. Patient lies perfectly still in bed, disliking movement and refusing food. No chest signs except diminished air entry at bases.

5th day. Torpid: Spits only when raised suddenly to the sitting position: otherwise the sputum lies in the mouth. Chest signs of a purulent bronchitis - no obvious consolidation.

13th day: Chest: coarse râles and rhonchi in front. Posteriorly fairly good air entry at bases, but a patch of loud bubbling sounds in right axilla. Cough reflex apparently lost, but when patient is laid on his side, he emits rather than expectorates large quantities of intensely foul yellowish brown sputum.

Post-mortem: Severe burns of face, chest, back, and genitals. Extensive ulceration of tonsils, pharynx, epiglottis, vocal cords and larynx. Surfaces of larger bronchi gangrenous. Both lungs entirely bound by recent adhesions. Both lungs on section show little else than a network of dilated and gangrenous bronchi.

DISEASE.

Notes of Case.

Name

Age

Diet

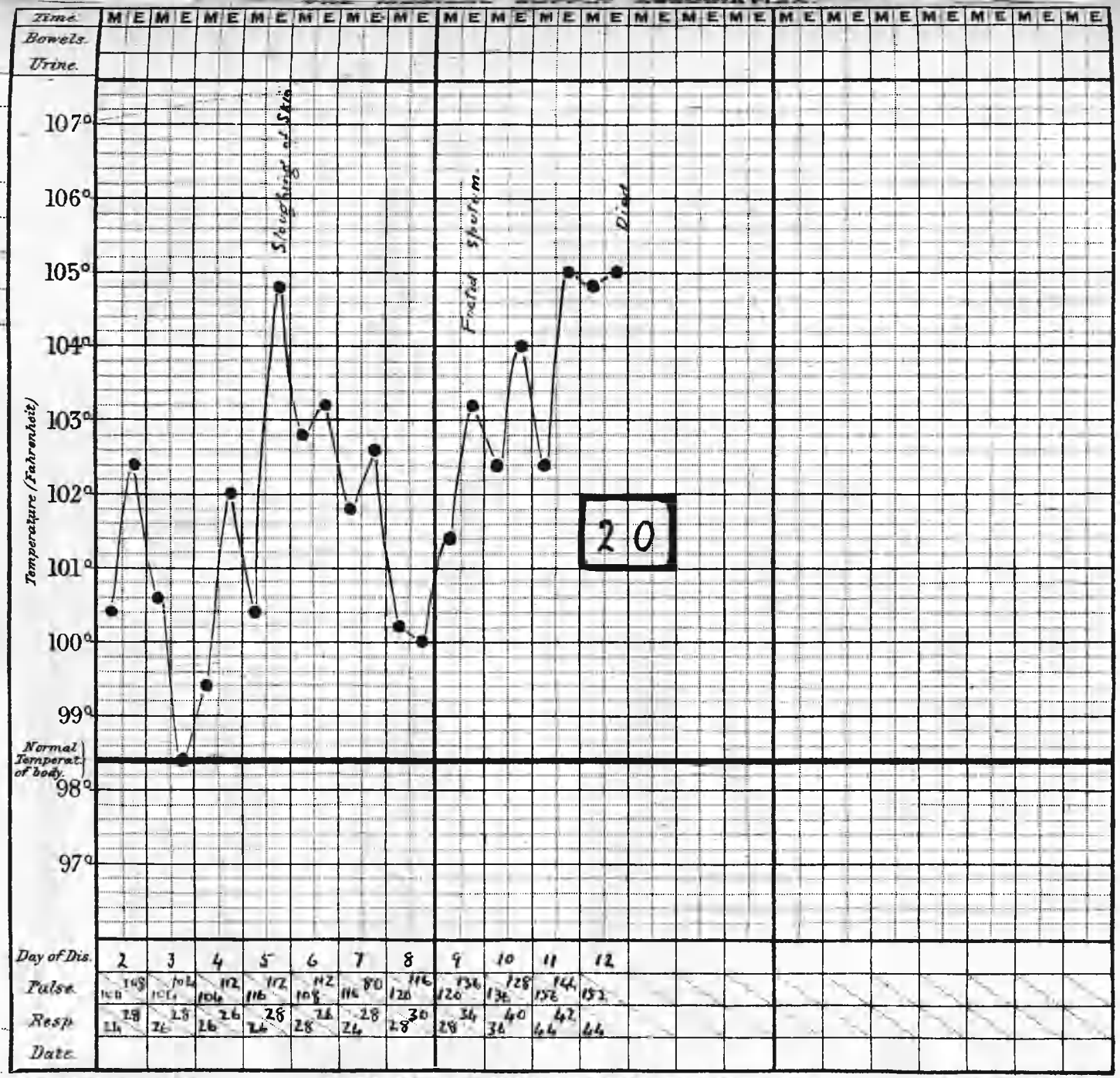
Case Book No. 20

Severe burns (vide chart on next page).

ABSCESS OF LUNG.

Date of admission.

Result



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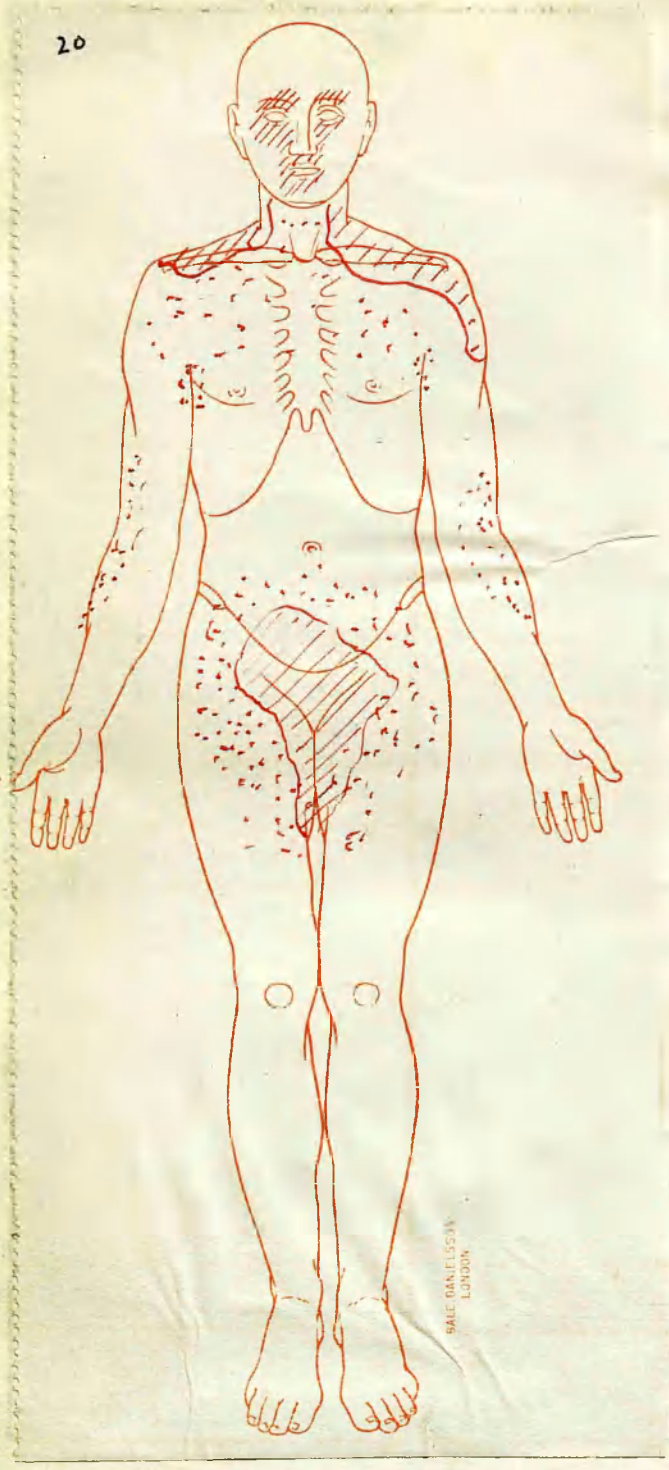
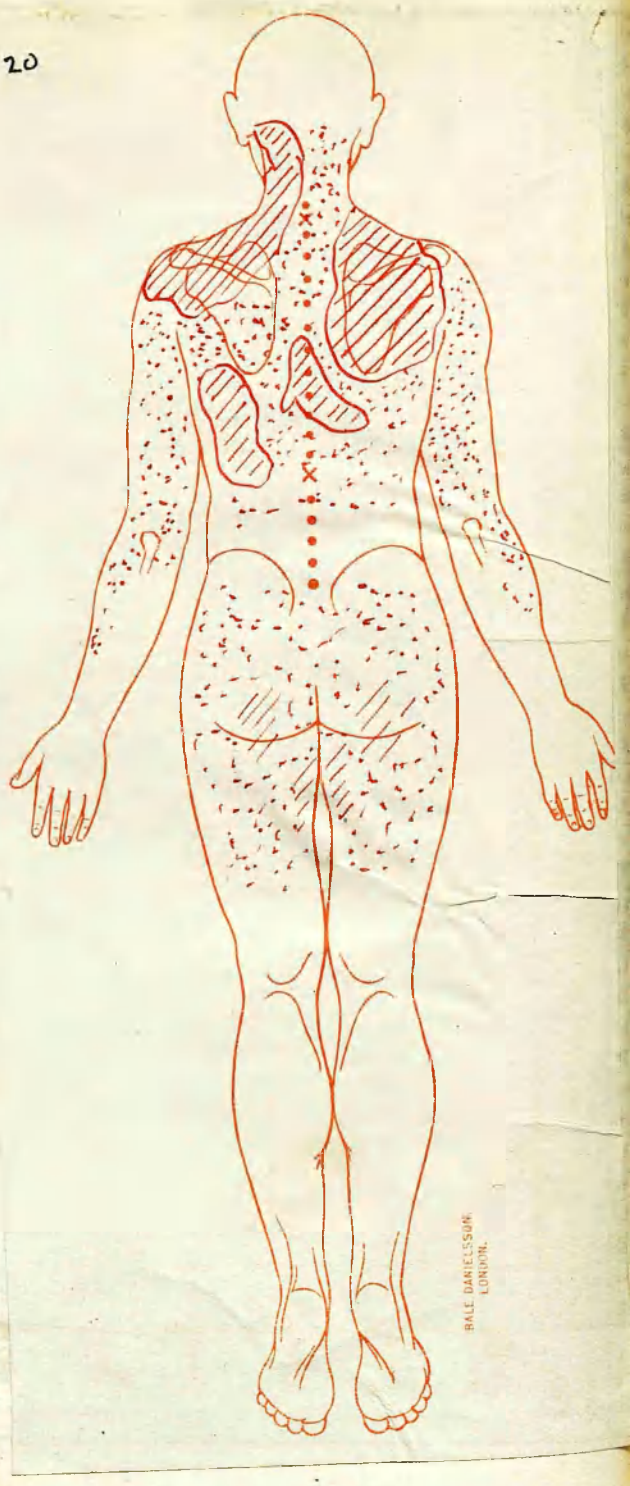




CASE 20 — SKIN LESIONS

DISEASE.

Notes of Case.

Name

Age

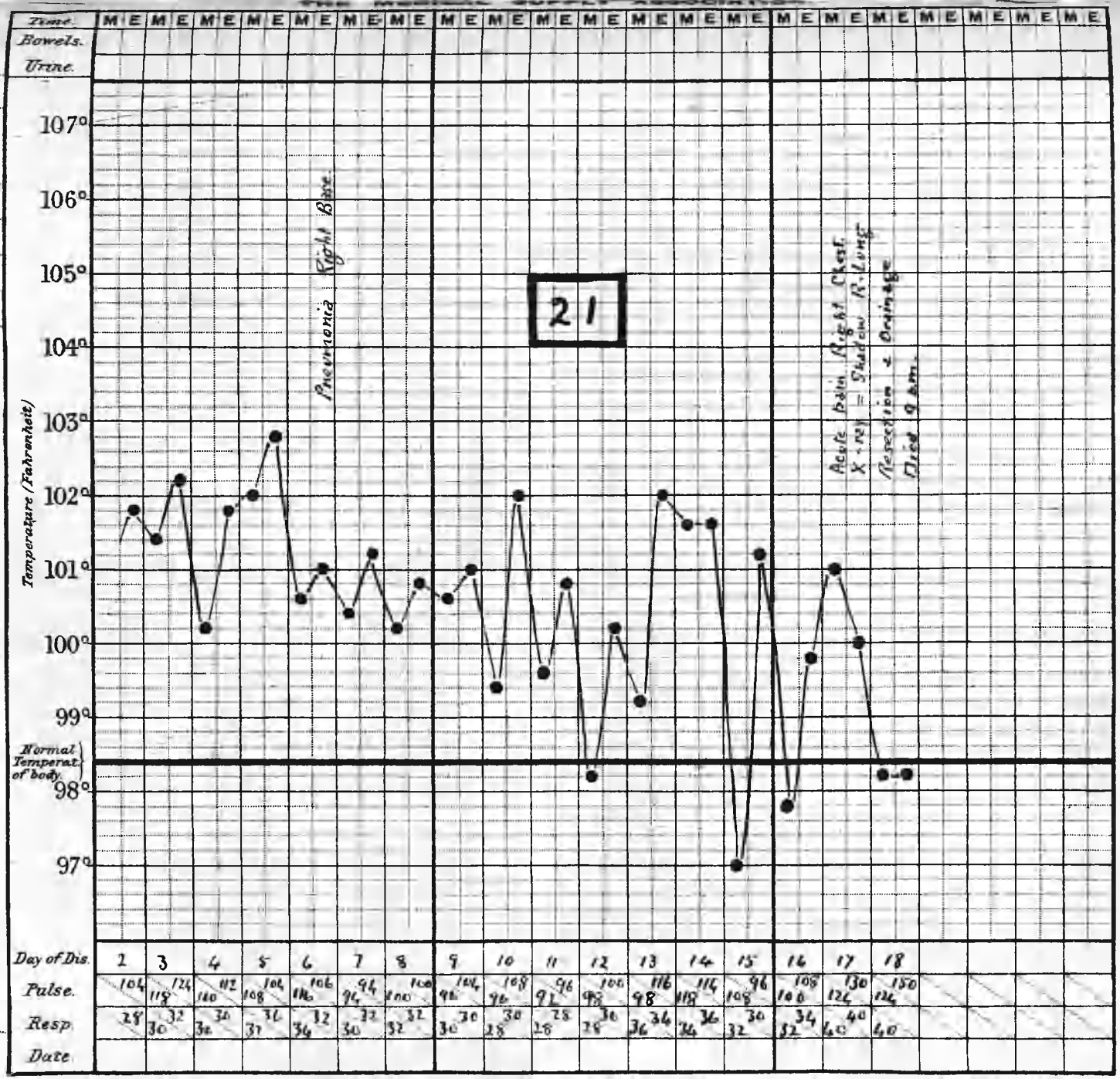
Diet

Case Book No. 21

PNEUMOTHORAX.

Date of admission.

Result



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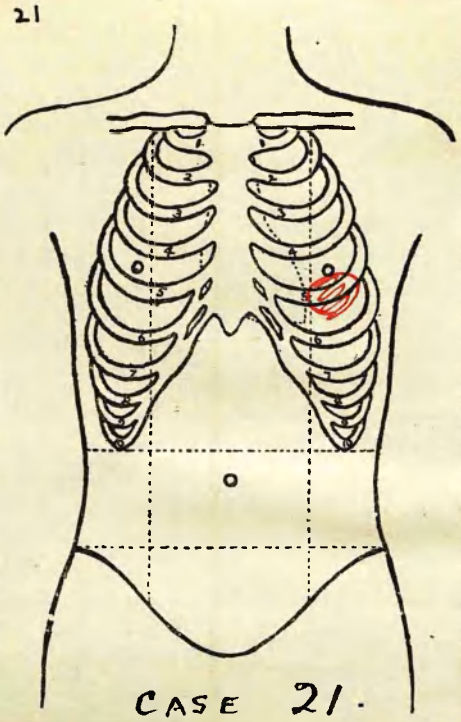
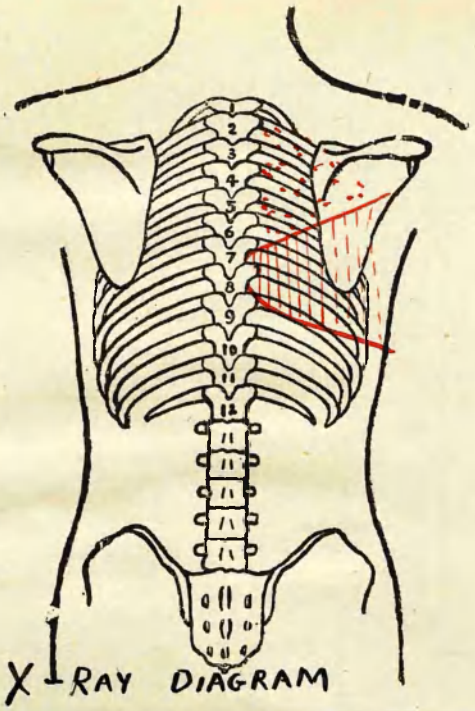


CASE 21.



X-RAY DIAGRAM

DISEASE.

Notes of Case.

Name

Age

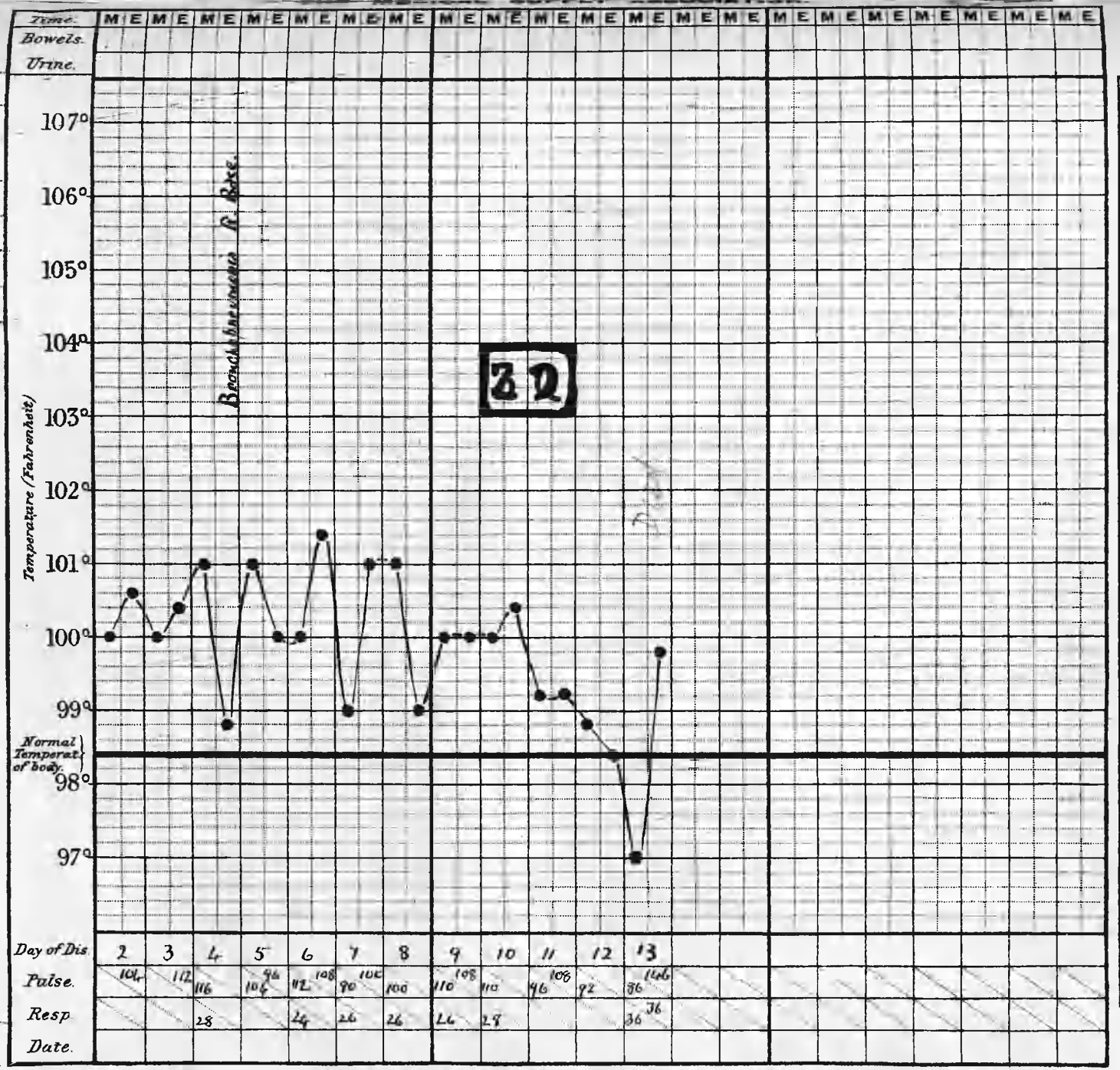
Diet

Case Book N° 22

ACUTE BRONCHIECTASIS.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

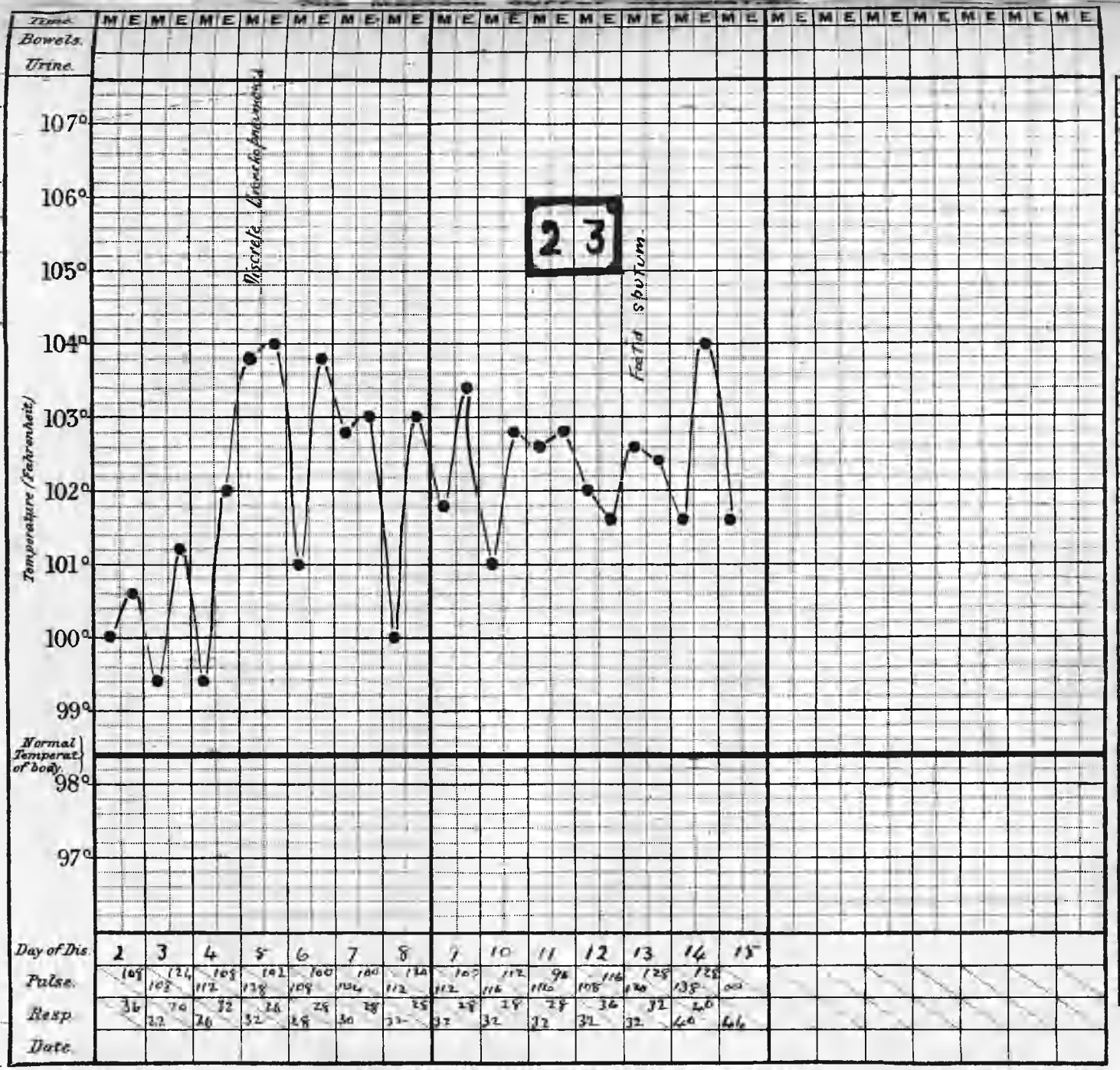
Diet

Case Book N° 23

GANGRENE OF LUNGS.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

Diet

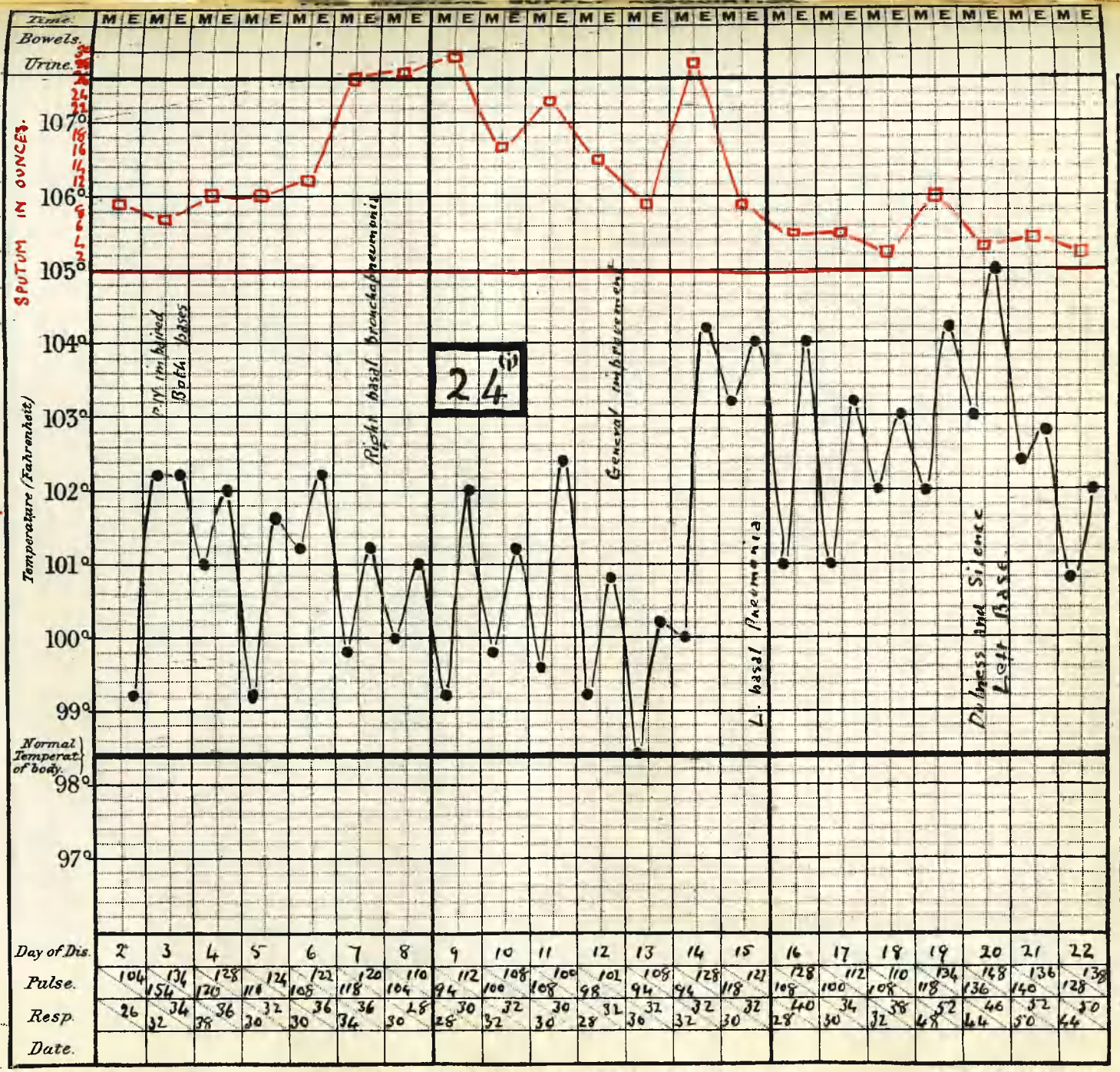
Case Book No. 24

EMPYEMA

Note fall of Sputum count after initial Broncho pneumonia without subsequent rise on development of Empyema

Date of admission.

Result



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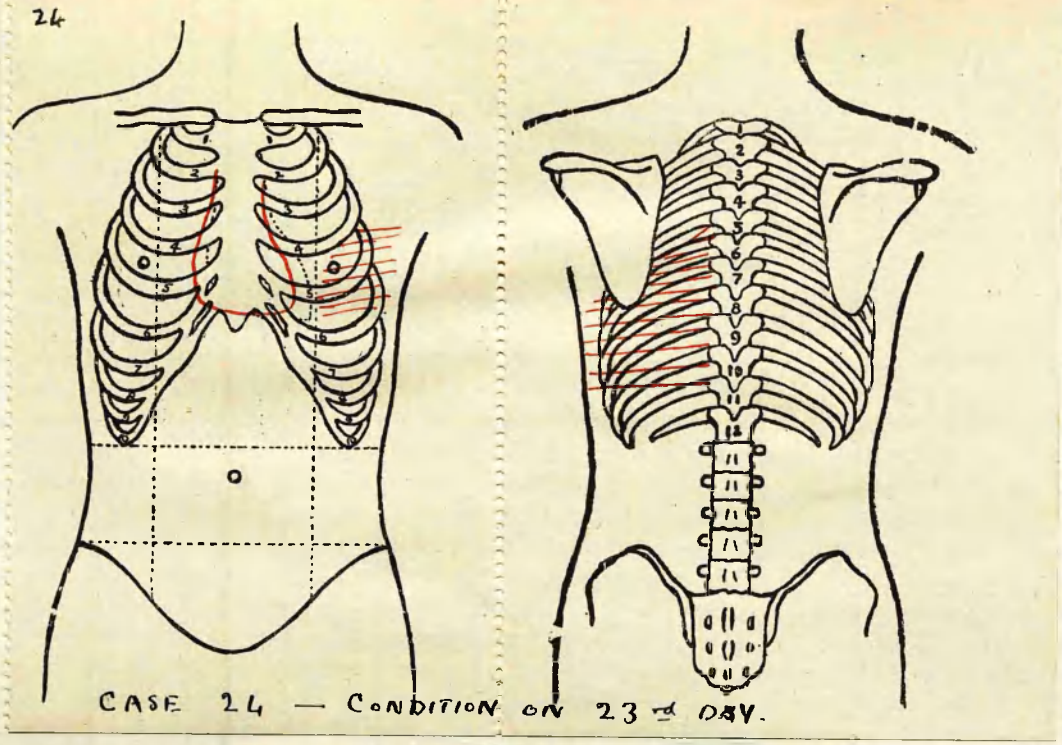
Gould's Clinical Chart

24



CASE 24 — CONDITION ON 23rd DAY.

DISEASE.

Notes of Case.

Name

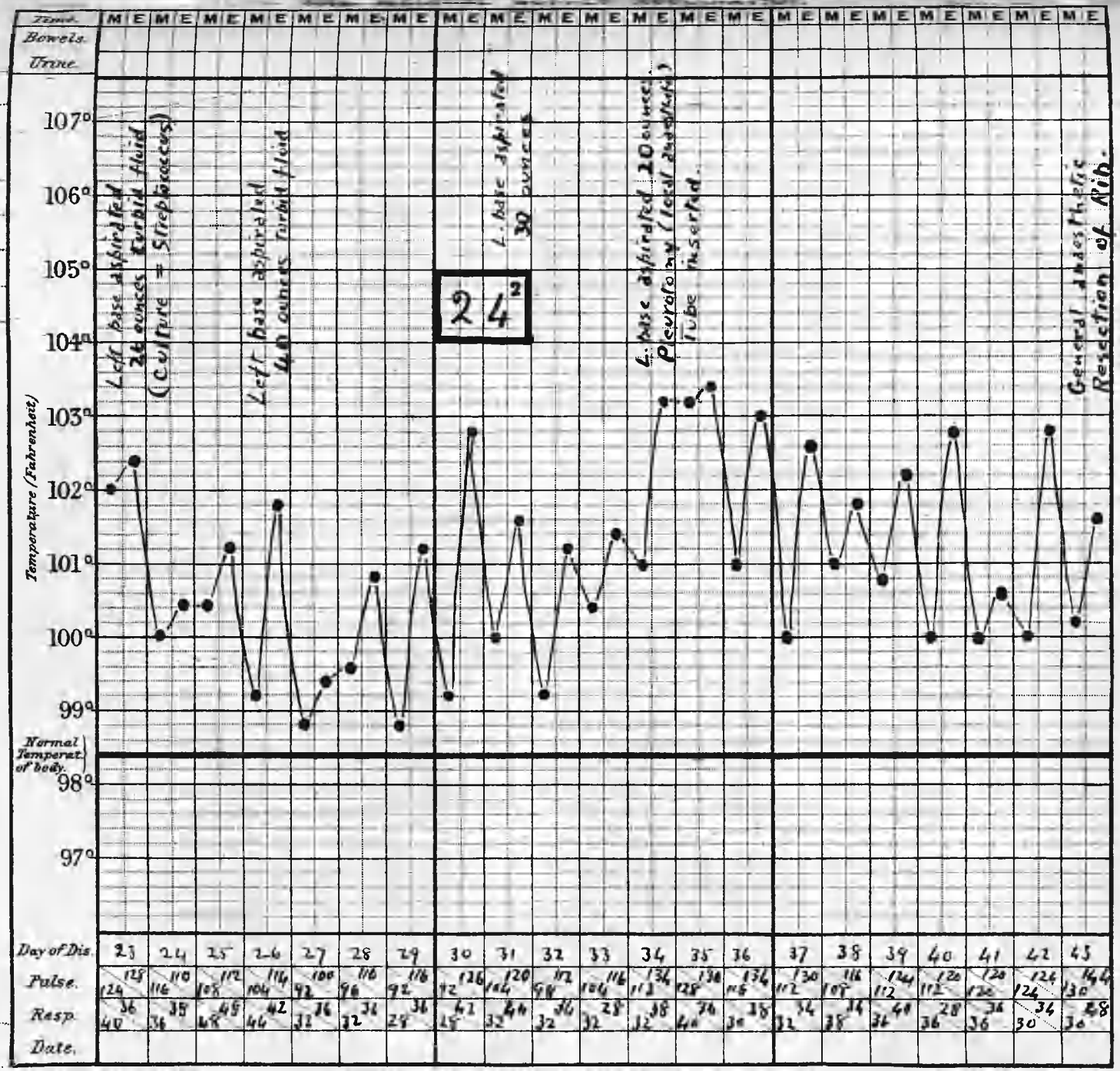
Age

Diet

Case Book N° 24

Date of admission.

Result



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Golden Clinical Chart

DISEASE.

Notes of Case.

Name

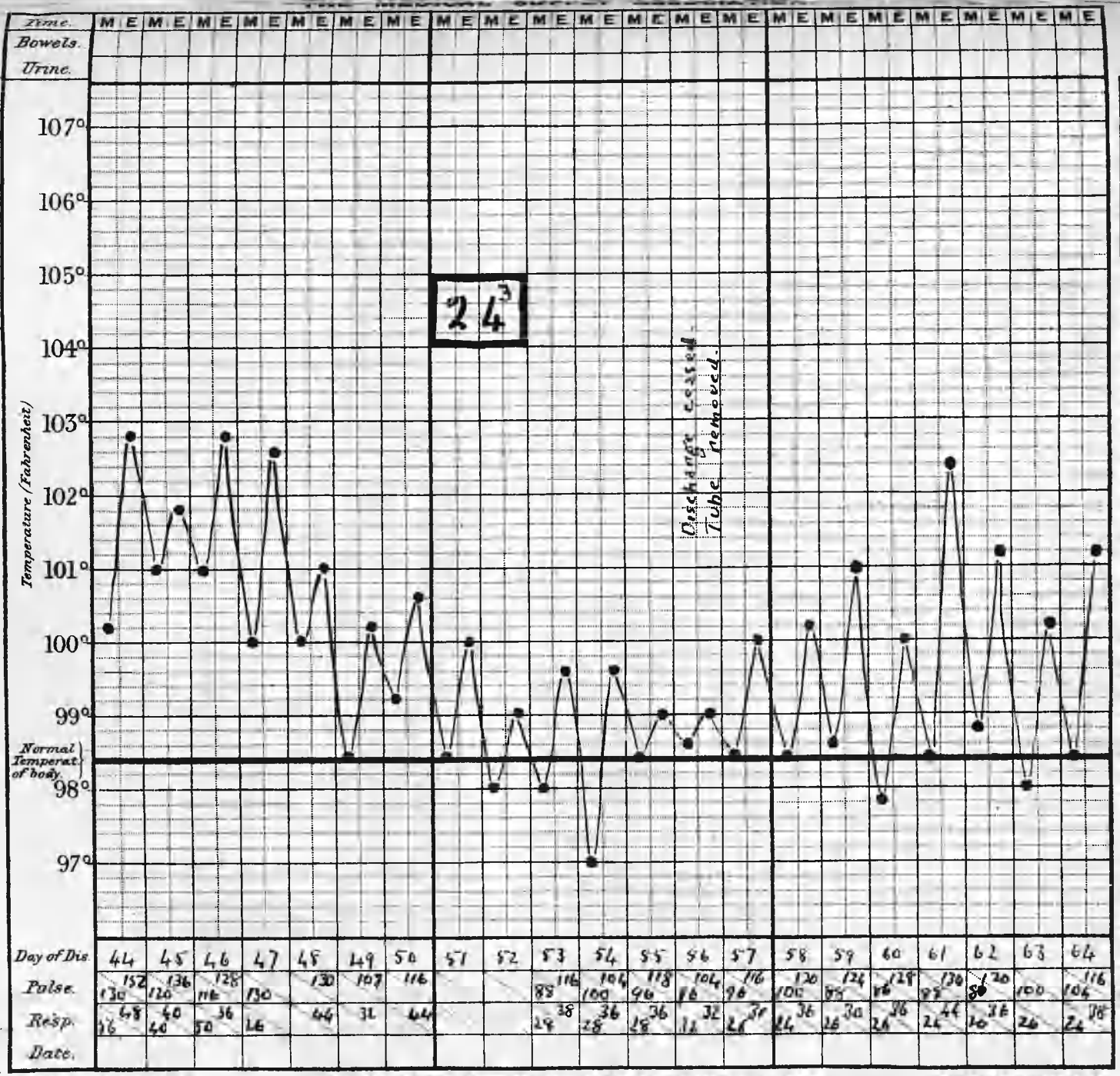
Age

Diet

Case Book N° 24

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

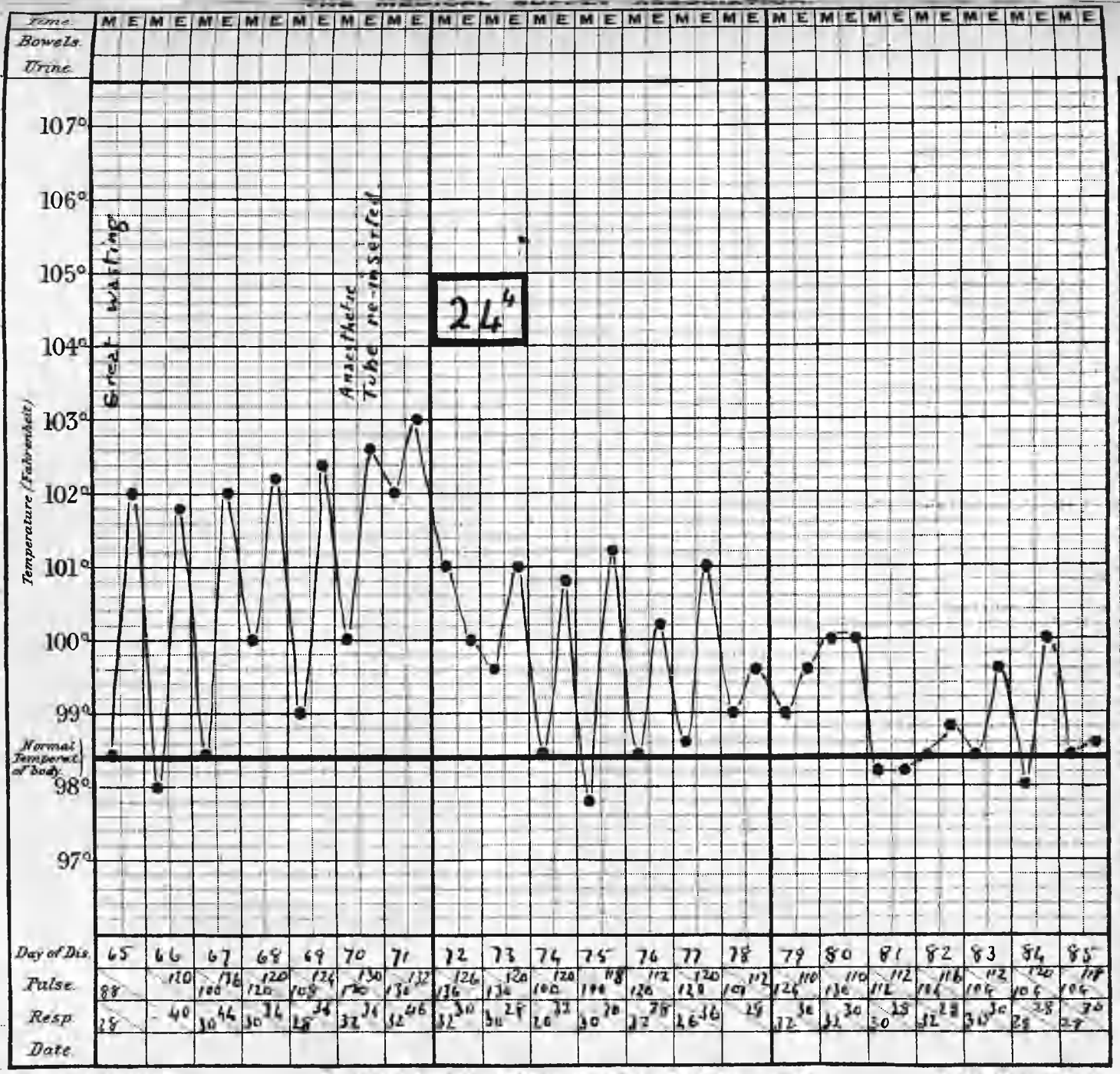
Diet

Case Book N° 24

Temperature settled finally on 96th day. Apyrexia with daily increase in health and strength till 124th day.
124th – 132nd day
Acute Influenzal Bronchopneumonia.
Complete recovery by 150th day—
Sinus healed

Date of admission

Result



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DISEASE.

Notes of Case.

Name

Age

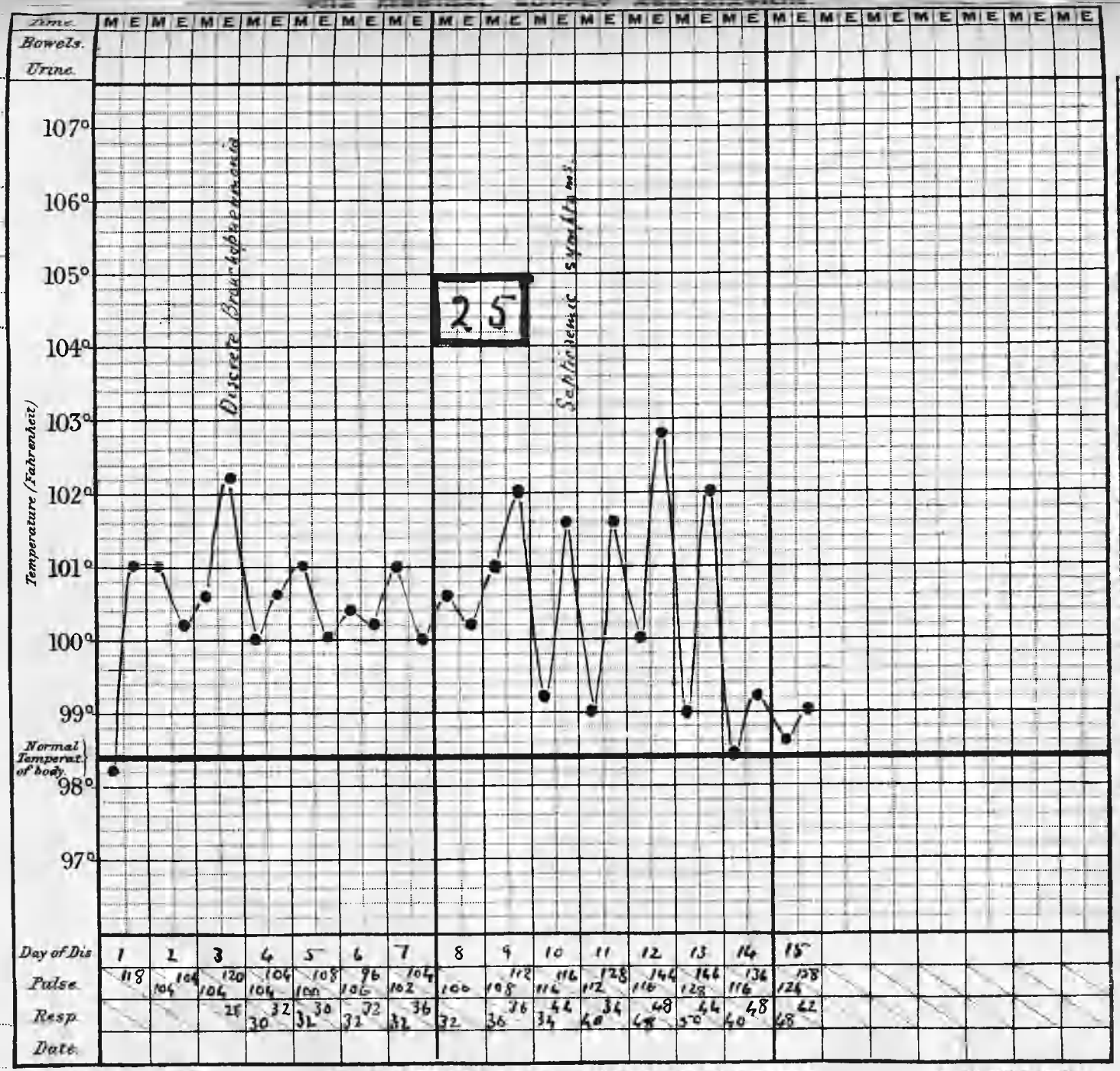
Diet

Case Book N° 25

STREPTOCOCCAL SEPTICAEMIA FROM 9th DAY.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No. 26

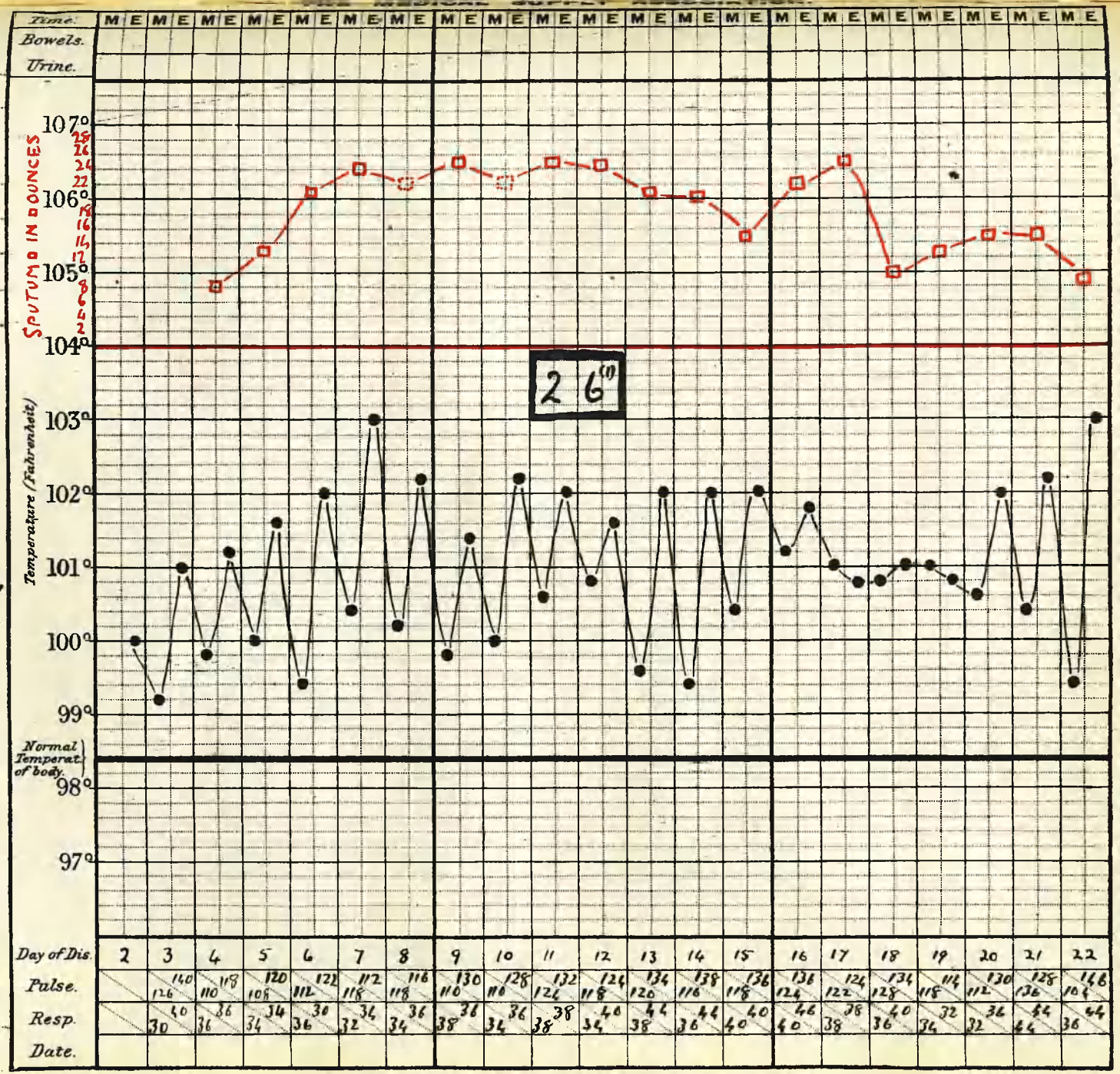
BRONCHIECTASIS.

Discrete Broncho-pneumonia leading to pulmonary necrosis. On 23rd day a foetid smell was noted in the sputum, and symptoms of toxaemia were marked.

After 6 months there was still severe cough with much purulent sputum.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

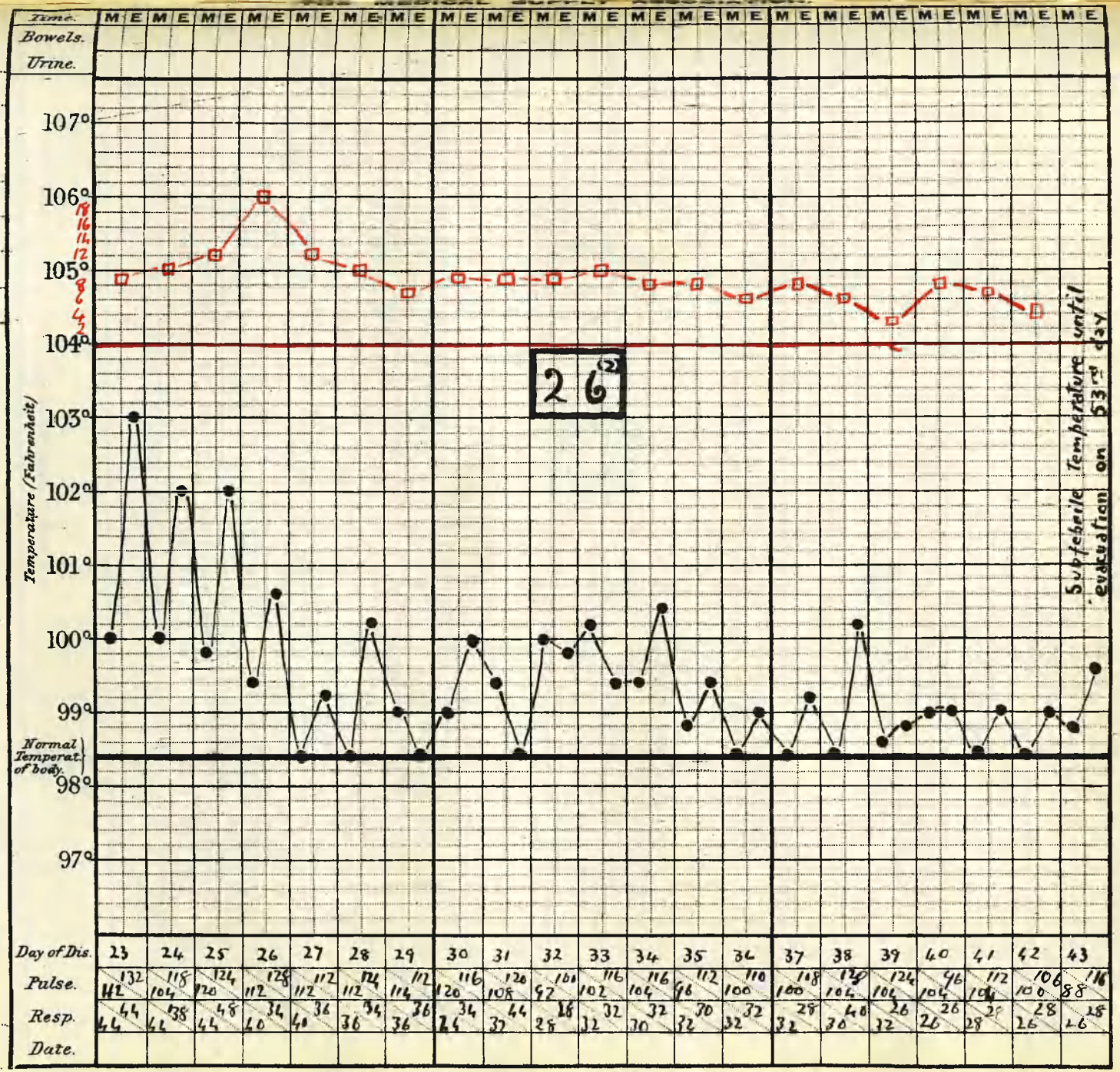
Diet

Case Book No. 26

Note persistently high sputum daily total.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No. 27

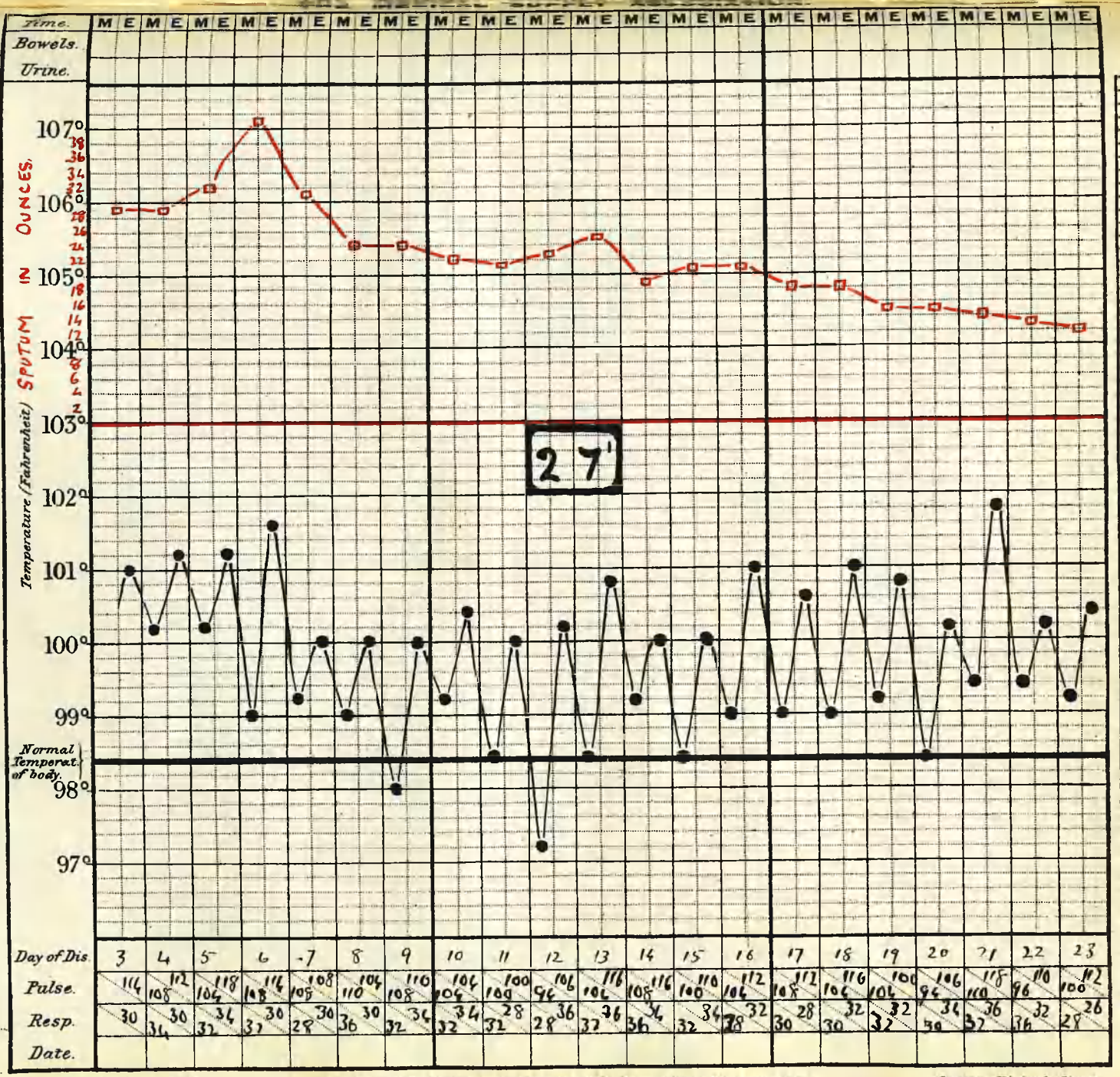
BRONCHIECTASIS.

Initial Right basal Bronchopneumonia followed by signs of pulmonary necrosis — foetid smell of sputum, "hectic" temperature and wasting.

After six months still cough with purulent sputum.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

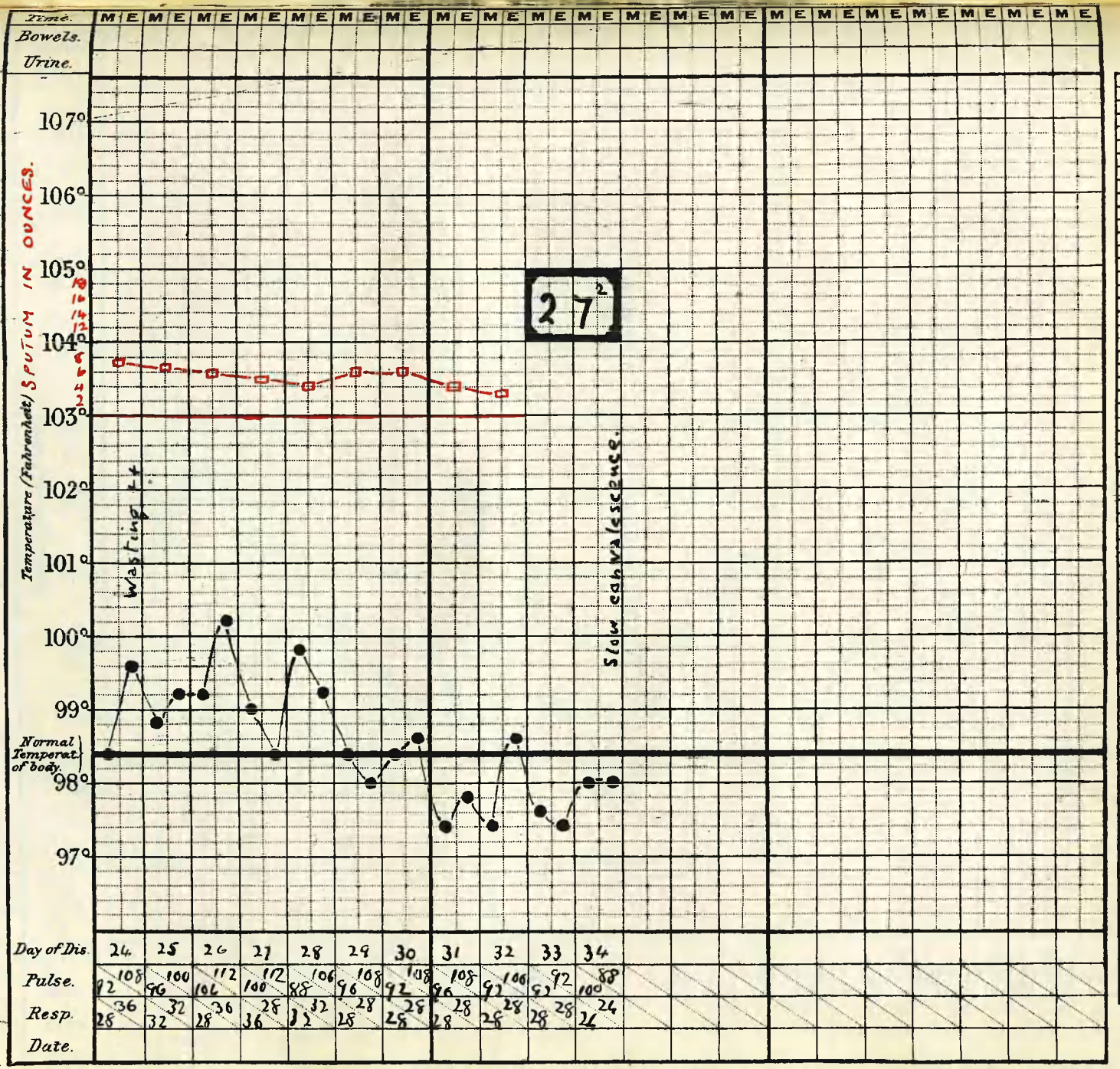
Diet

Case Book N° 27

Gradual (and incomplete) fall in daily sputum total.

Date of admission.

Result



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DISEASE.

Notes of Case

Name

Age

Diet

Case Book N° 28

PULMONARY NECROSIS

? BRONCHIECTASIS.

Initial discrete Bronchopneumonia with remission (of symptoms at least) between 12th and 17th days. On 21st day marked dulness R. Base: blood-stained sputum and 'septic' temperature of inverse type. Very rapid wasting. No T.B. found on repeated examinations. Signs gradually cleared and convalescence established from 40th day.

Date of admission

Result

28

Temperature (Fahrenheit): 107° 106° 105° 104° 103° 102° 101° 100° 99° Normal Temperature of body 98° 97°

Temperature (Centigrade): 42° 41° 40° 39° 38° 37° 36° 35°

Day of Dis	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
Pulse.	96	132	128	120	108	100	100	108	104	100	100	104	104	100	108	104	104	100	104	104	104
		152	128	110	108	90	96	100	140	112	108	108	100	98	94	108	96	122	120	118	116
Resp.		24	28	34	25	26	26	26	32	32	30	26	22	24	24	24	24	28	24	28	34
			26	30	30	34	36	36	40	36			24		24		26	36	32	30	32
Date.																					

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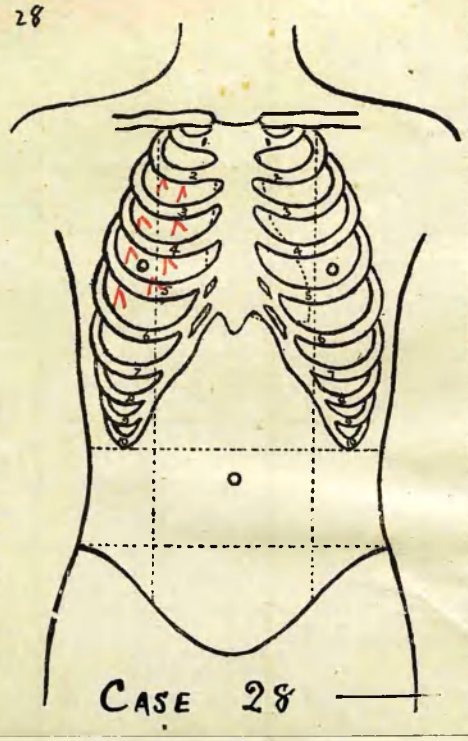
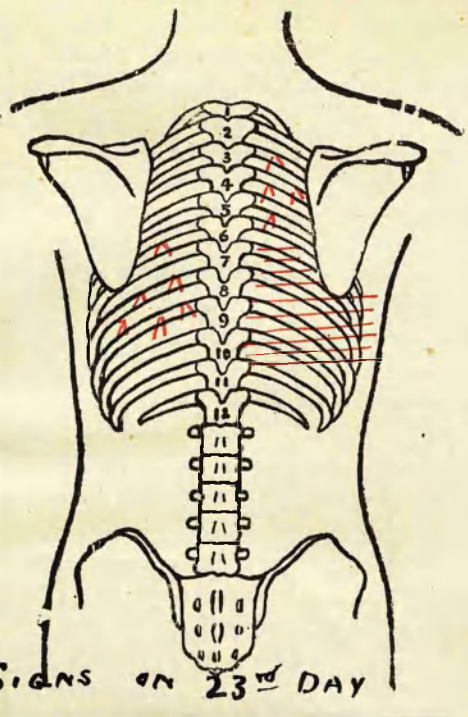
28



CASE 28



SIGNS ON 23rd DAY

DISEASE.

Notes of Case.

Name

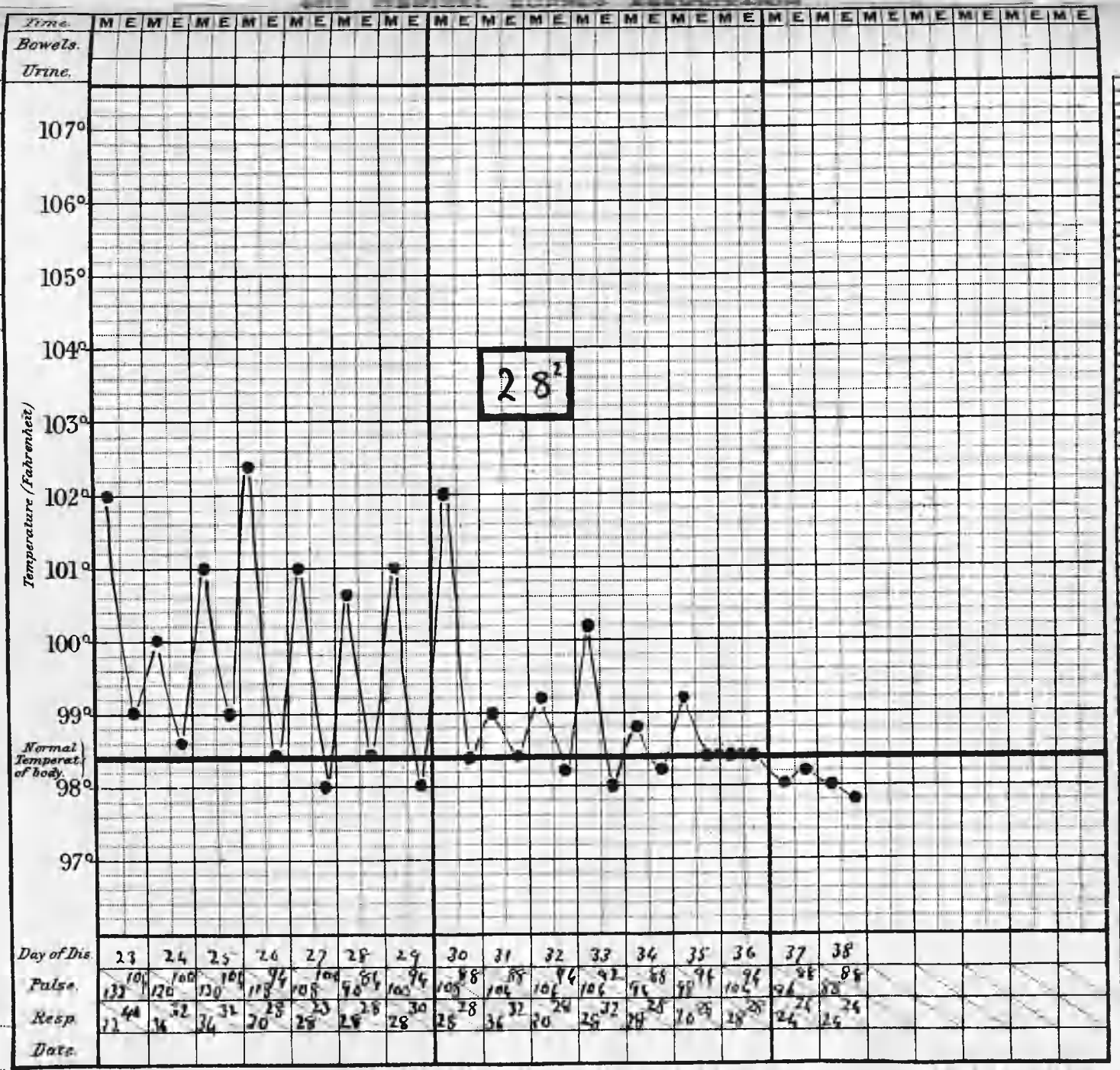
Age

Diet

Case Book N° 28

Date of admission

Result



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DISEASE.

Notes of Case.

Name

Age

Diet

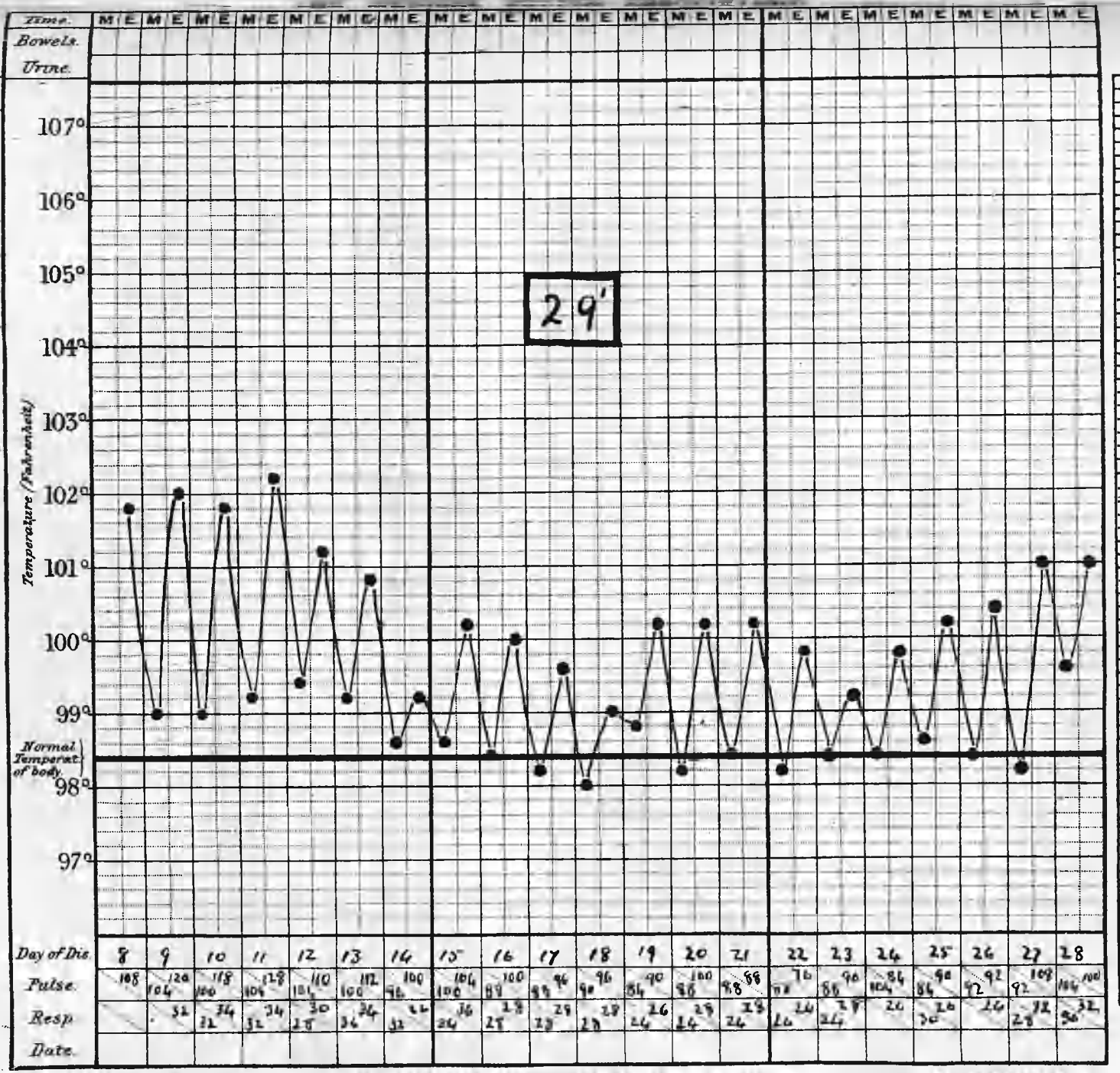
Case Book N° 29

BRONCHIECTASIS.

Initial discrete Bronchopneumonia with remission (partial) between 14th and 24th days.

Date of admission

Result



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DISEASE.

Notes of Case.

Name

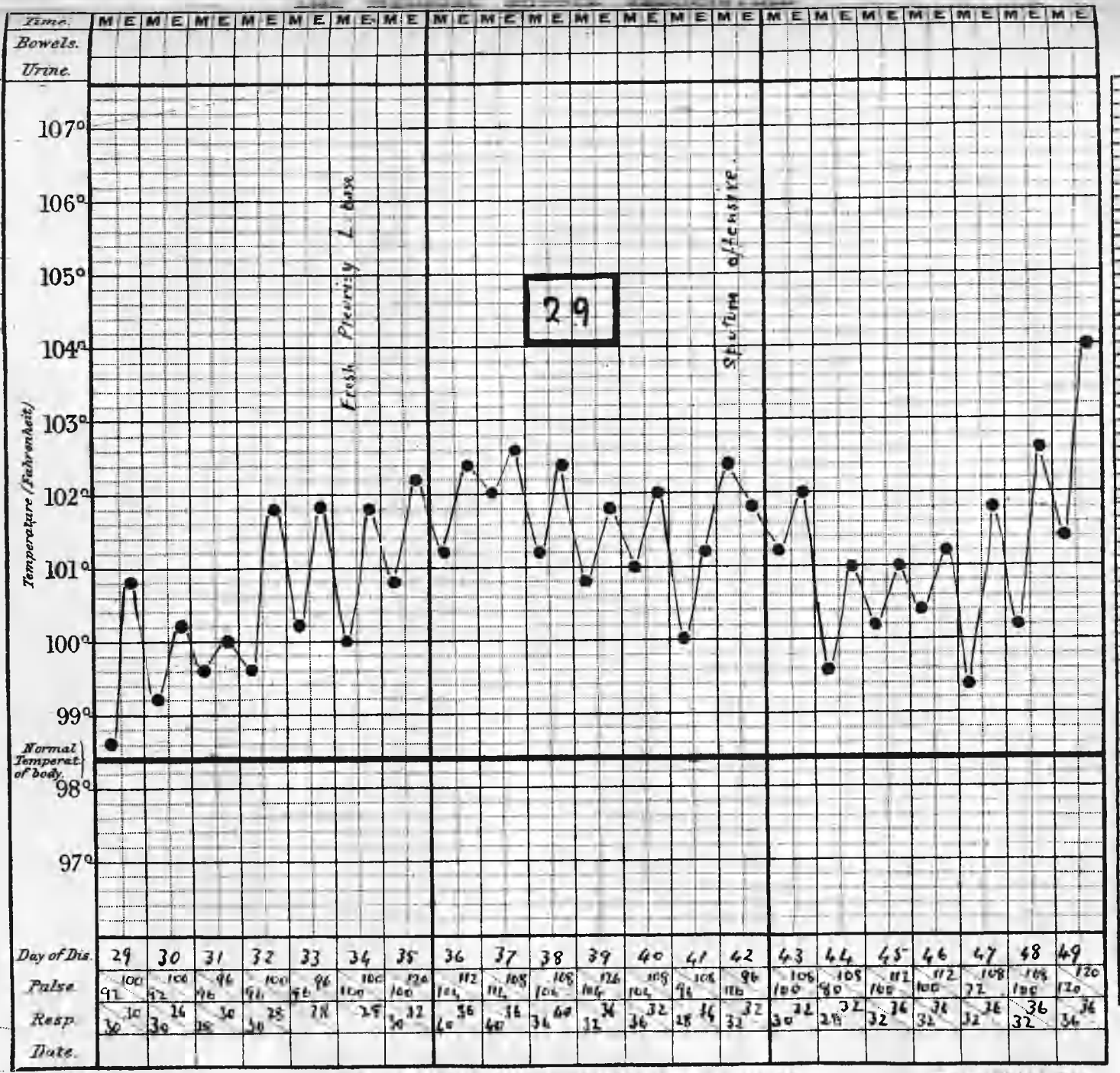
Age

Diet

Case Book N° 29

Date of admission.

Result



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Gould's Clinical Chart

DISEASE.

Notes of Case.

Name

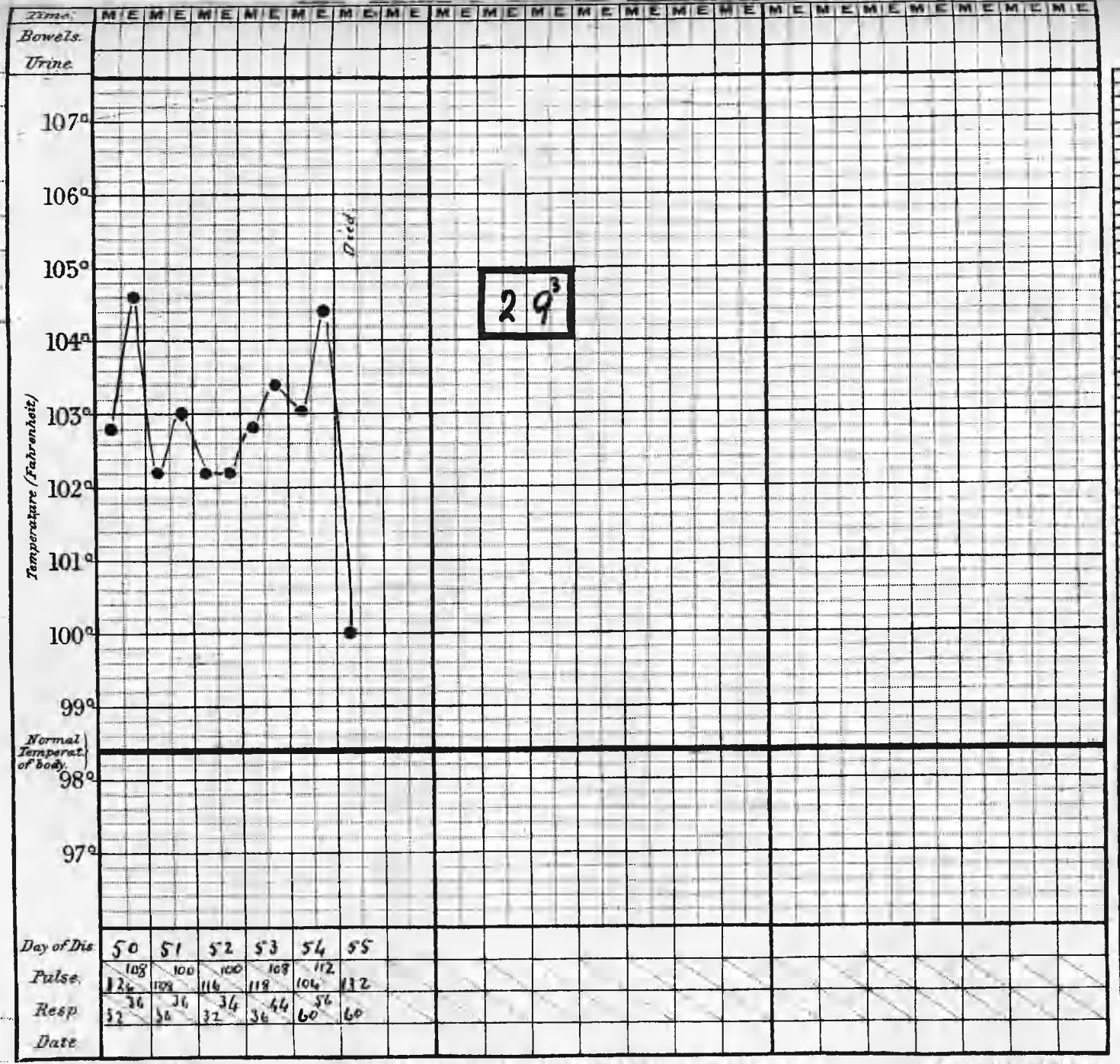
Age

Diet

Case Book N° 29

Date of admission

Result



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DISEASE.

Notes of Case.

Name

Age

Diet

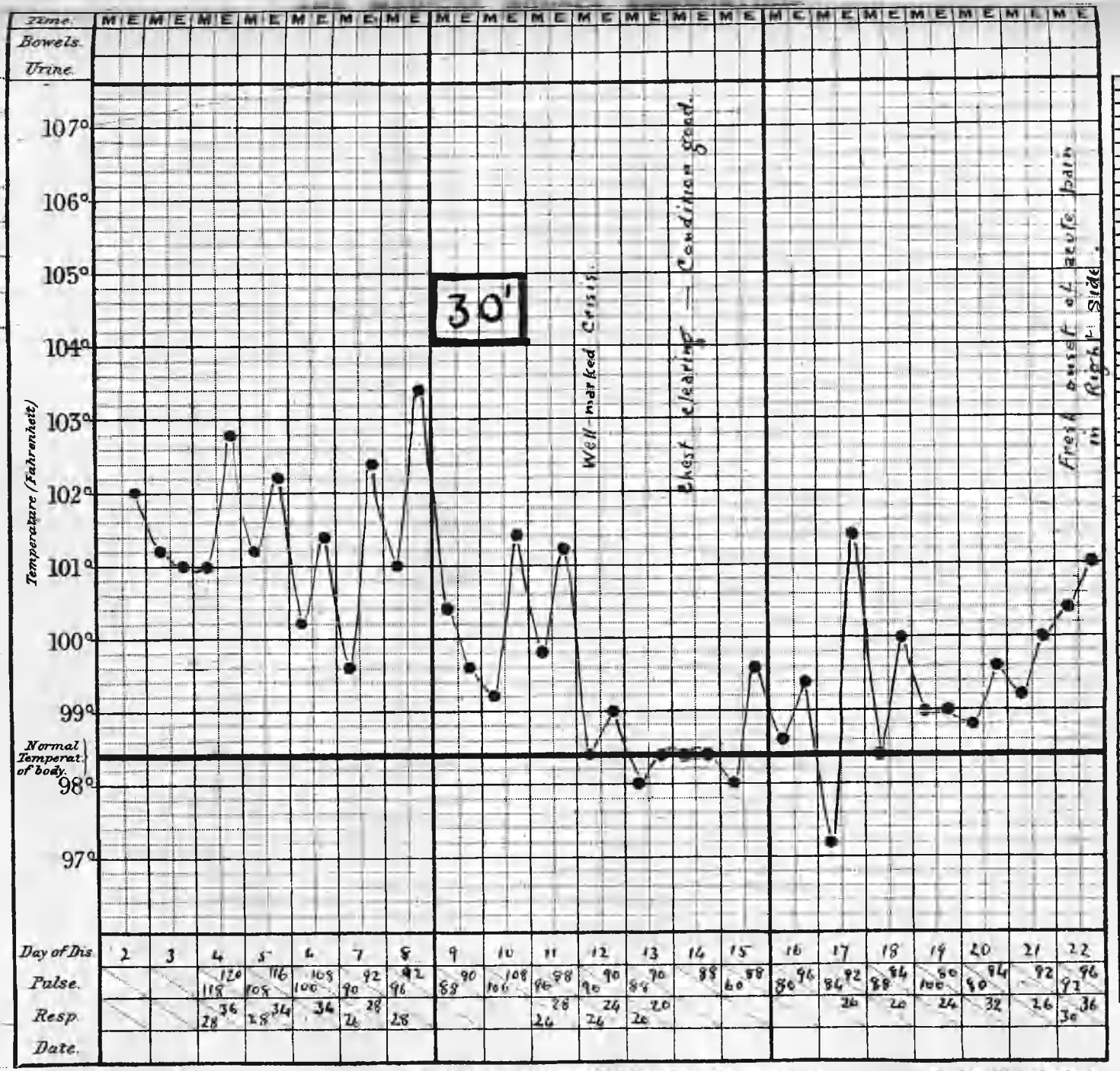
Case Book N° 30

CHRONIC PNEUMONIA

ACUTE STREPTOCOCCAL BRONCHOPNEUMONIA.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

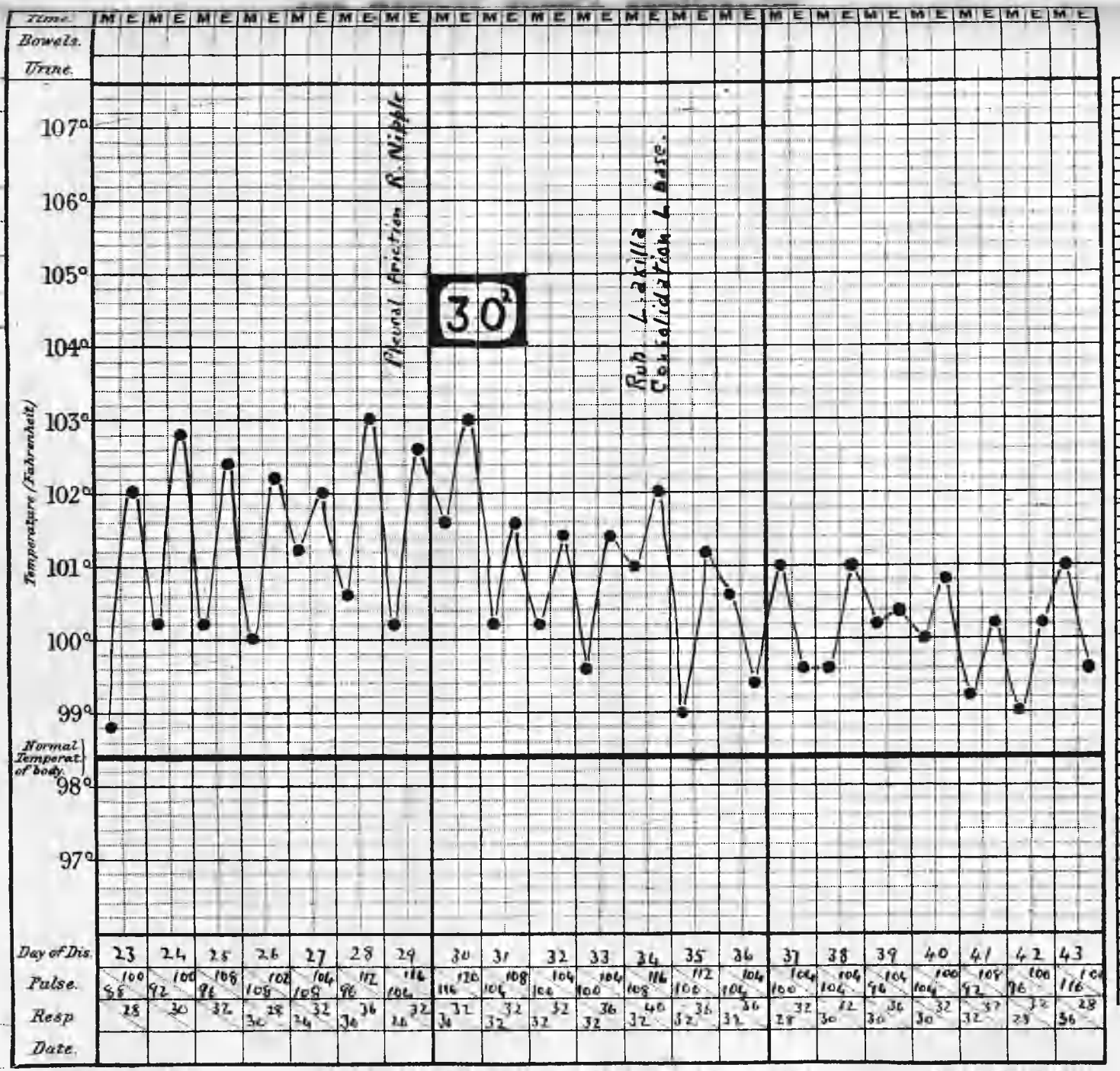
Age

Diet

Case Book N° 30

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

Diet

Case Book N° 30

Date of admission.

Result

Time.	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	M E	M E
Bowels.																										
Urine.																										

Temperature (Fahrenheit)

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

Normal Temperat. of body.

Consolidation L^g Base 1/52.

Died

30²

Temperature (Centigrade)

42° 41° 40° 39° 38° 37° 36° 35°

Day of Dis.	44	45	46	47	48	49	50
Pulse.	120 / 112	116 / 114	124 / 124	120 / 112	120 / 132	148 / 140	124 / 130
Resp	32 36	44 32	32 36	40 36	44 28	40 48	48 44
Date.							

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GROUP F: CUTANEOUS LESIONS:

Case 31: Pte.N. Diagnosed "lachrymatory gas" at Field Ambulance and clothing not changed.
2nd. day: no conjunctivitis: no cough. One vast burn all over back from occiput to lower buttocks. Patient seemed quite comfortable, and general condition remained good until
5th day when the blisters changed to sloughs, the whole skin began to peel off. Symptoms of septic absorption set in. Quite suddenly a large amount of blood and albumin appeared in the urine. There was neither headache nor oedema, and no vomiting until the 10th day.
On the 10th day the temperature fell and the patient felt comfortable. But at the same time the pulse became running and uncountable. No chest symptoms or signs developed at any time. The patient remained perfectly conscious and rational until within an hour of death.

Post-mortem: Eyes, pharynx, larynx, trachea and lungs normal. Skin lesions as on diagram. Kidneys - acute haemorrhagic nephritis.

Case 32: Pte. M.

2nd. day: on admission very collapsed. Vomited and became almost pulseless two hours after admission.
3rd. day: Attacks of faintness and vomiting. No chest signs. Urine clear.
4th day: Slight improvement, but burns not reacting. Trace of albumin in urine, and some blood.
5th day: Slight cough and a few rhonchi in chest. Heart still weak.
6th day: Urine: much albumin. Several syncopal attacks during the day. At 5.30 p.m. vomited and died of syncope.

Post-mortem: Trachea moderately injected but no ulceration. Lungs practically normal (a few areas of consolidation found microscopically). Stomach very acutely congested, with petechial haemorrhages near cardiac end. Kidneys - acute congestion of glomeruli. Charts 33, 34 and 35 are examples of very extensive skin lesions without respiratory involvement. They show well the more uniform progress and evolution of the skin lesions as compared with those of the respiratory tract - the acute stage between the 6th and 10th days, at the time of greatest absorption from the infected surfaces; the gradual steady improvement; and the complete and final settling of the temperature about the 21st day, when regeneration of the epithelium has begun to make headway.

Cases 36, 37 and 38 are examples of severe and extensive burns complicated by respiratory involvement. In the first case (36) the/

the critical stage of the Burns (6th day) preceded the respiratory symptoms by two days. A striking increase in respirations occuss only on the 8th day.

Case 38 is particularly interesting and deserves fuller quotation than I have space to give:

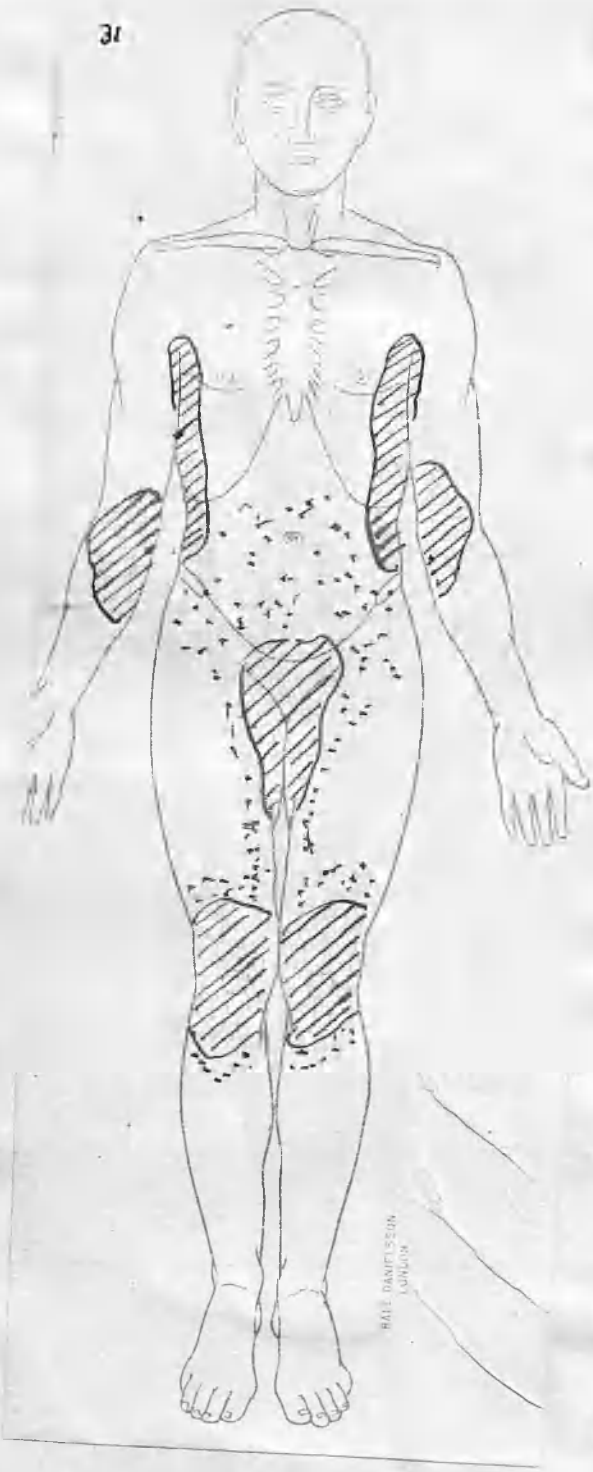
Pte. B. - admitted 2nd. day: Skin lesions as on diagram.
3rd. day. Flushed but not cyanosed. Sputum dark brown, frothy and blood-stained.
Chest: rhonchi all over. Resonance good in front but impaired at both bases. Fine crepitations and diminished breath sounds at left base.
4th day. Burns not reacting well. Consolidation left base.
Heart: Maximum impulse 5½ inches from mid-sternum - slapping in character. Presystolic and systolic murmurs at apex. 2nd sound reduplicated at apex. Becomes faint and giddy on sitting up. Delirious at night.
8th day: General improvement. Patient feels well and is able to read papers, write letters etc. Left base still dull: bronchial breathing.
12th day: Much better. Chest practically clear; but three large areas of skin are stripped of epidermis and their bases are sloughy and infected.
15th day: Severe rigor: Dyspnoea and acute pain left side. Tenderness L. lumbar region. Leucocytosis 44,000.
16th day: Anuria and acute headache. Heart very irregular. Totally unconscious at night and one convulsion. Breathing of cheyne stokes type.
17th day: Conscious: passed a little urine at noon - solid with albumin. Severe headache. Pupils small.
18th & 19th day: Spreading consolidation left base; tenacious 'rusty' sputum. Acute precordial pain.
20th day: Skin regeneration commencing, but bedsore left hip. Patient becoming very thin and wasted. Chest signs as above.
21st day: Loud bronchial breathing left base and axilla. Pleuro-pericardial friction.
23rd day: Massive consolidation extended to left upper lobe. Heart suddenly slowed, pulse 'missing' every third beat. Toxic and delirious.
24th day: Toxaemia: picking at bedclothes. Herpes round lips. Tongue dry, brown and fissured. Signs unchanged.
30th day: Gradual improvement in chest condition and burns healing rapidly. Still a heavy cloud of albumin in urine.
32nd day: Frontal headache. Sudden oedema of left foot, left hand, and left side of face. Chest: coarse crepitations all over left back. Heart beat fuller and stronger.
39th day: Great improvement. Burns now healed and chest almost clear. But the patient is wasted to a skeleton.
42nd - 44th day: An attack of Influenza.

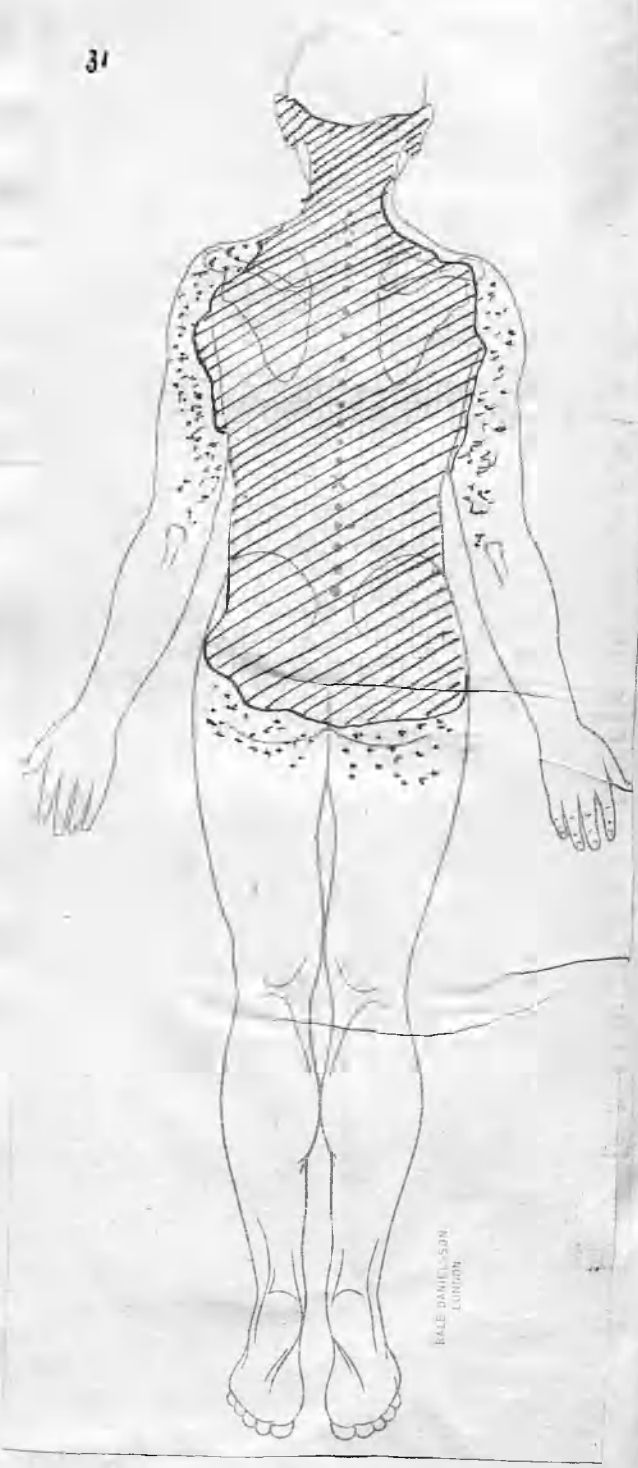
57th/

57th day: Transferred to England - well and cheerful. Urine clear.
5th month: Discharged to duty A1.

Case 87 is very similar to 31 but was complicated by extensive bronchopneumonia. The patient died of acute haemorrhagic nephritis.

----------oOo----------





SKIN LESIONS — CASE 31

DISEASE.

Notes of Case.

Name

Age

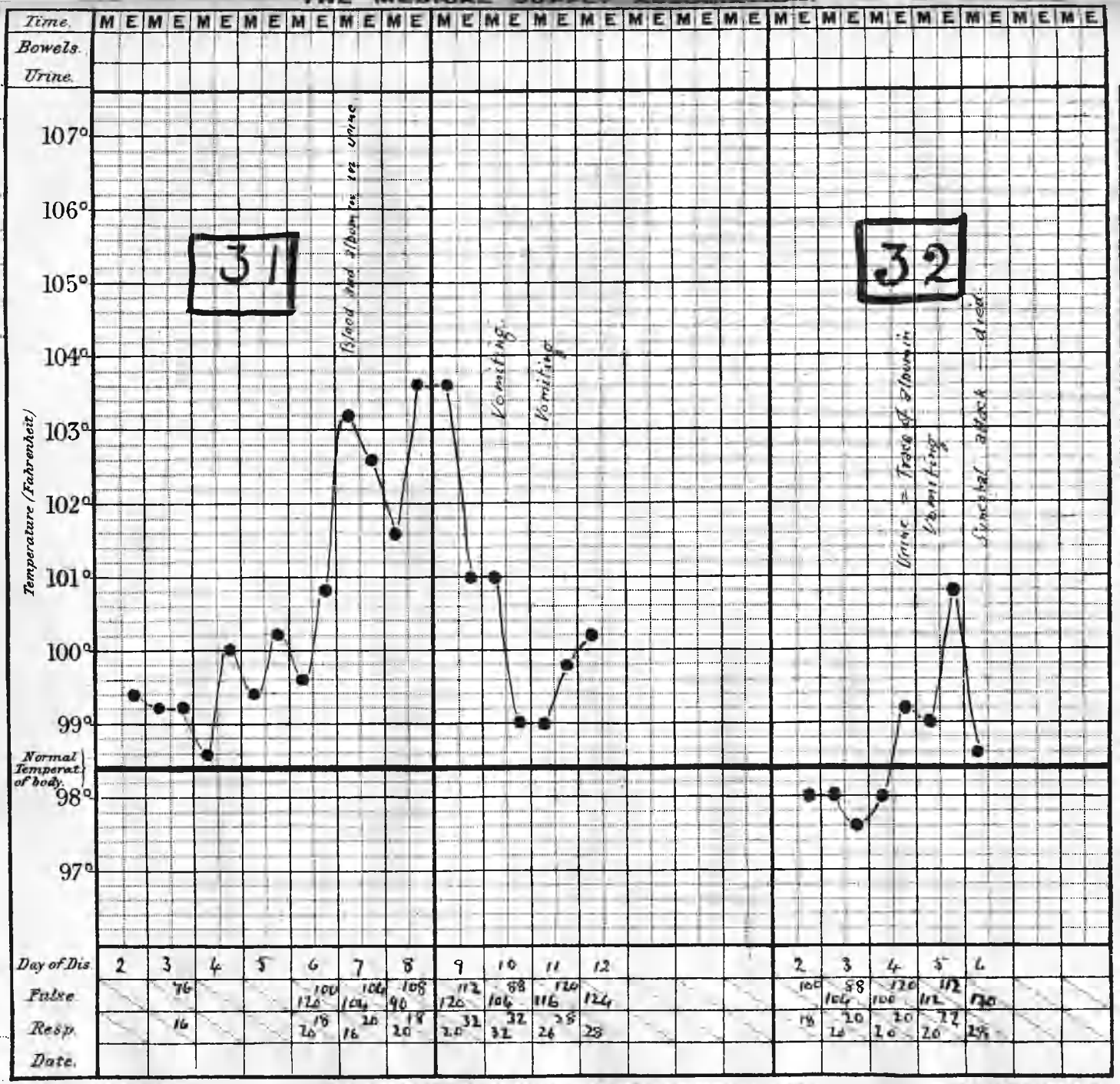
Diet

Case Book N° 31 / 32

Gelatinous Lactic and Milk diets

Date of admission

Result



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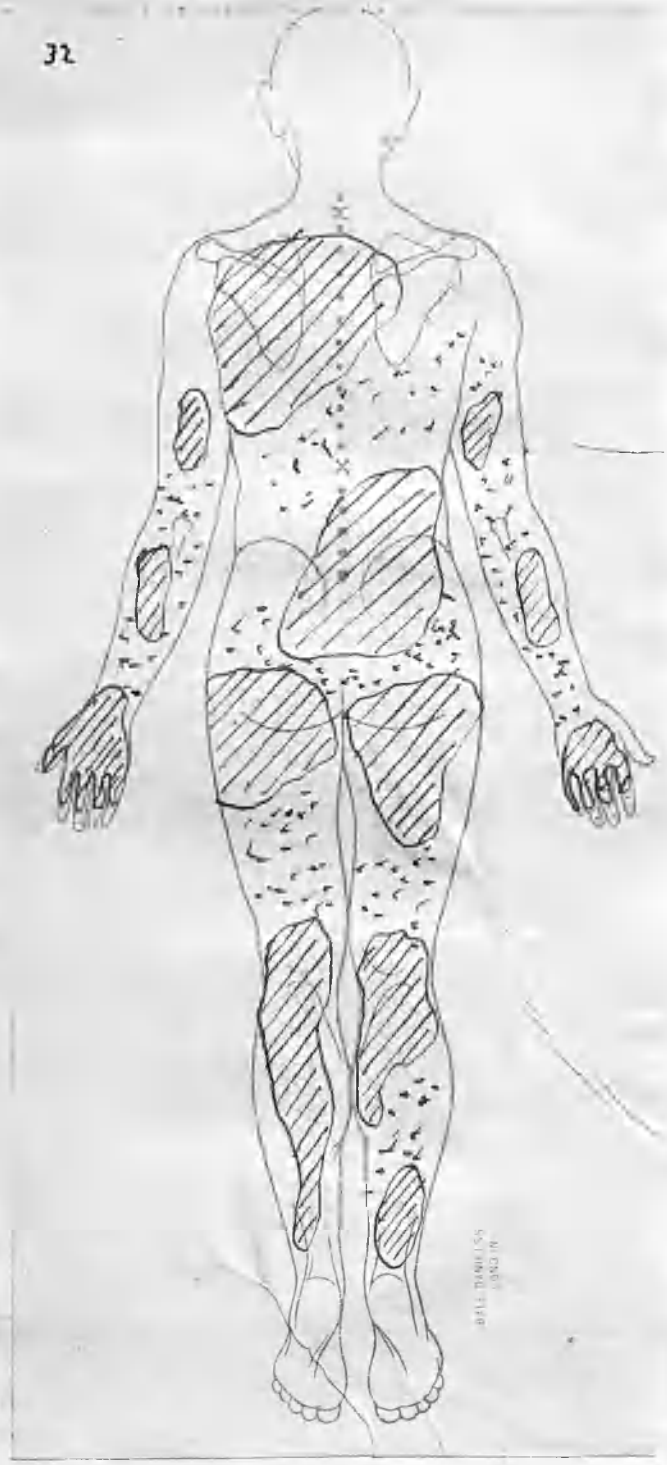
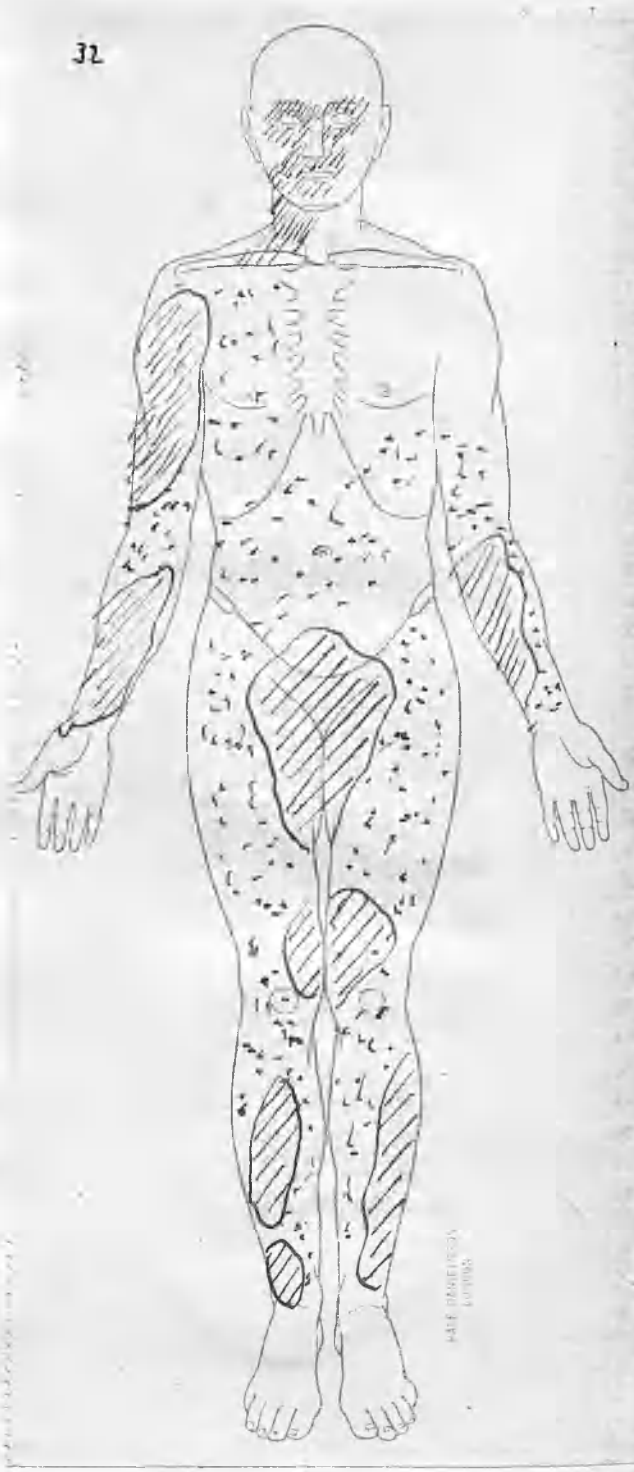




SKIN · LESIONS — CASE 32

DISEASE.

Notes of Case.

Name Pte. J.

Age

Diet

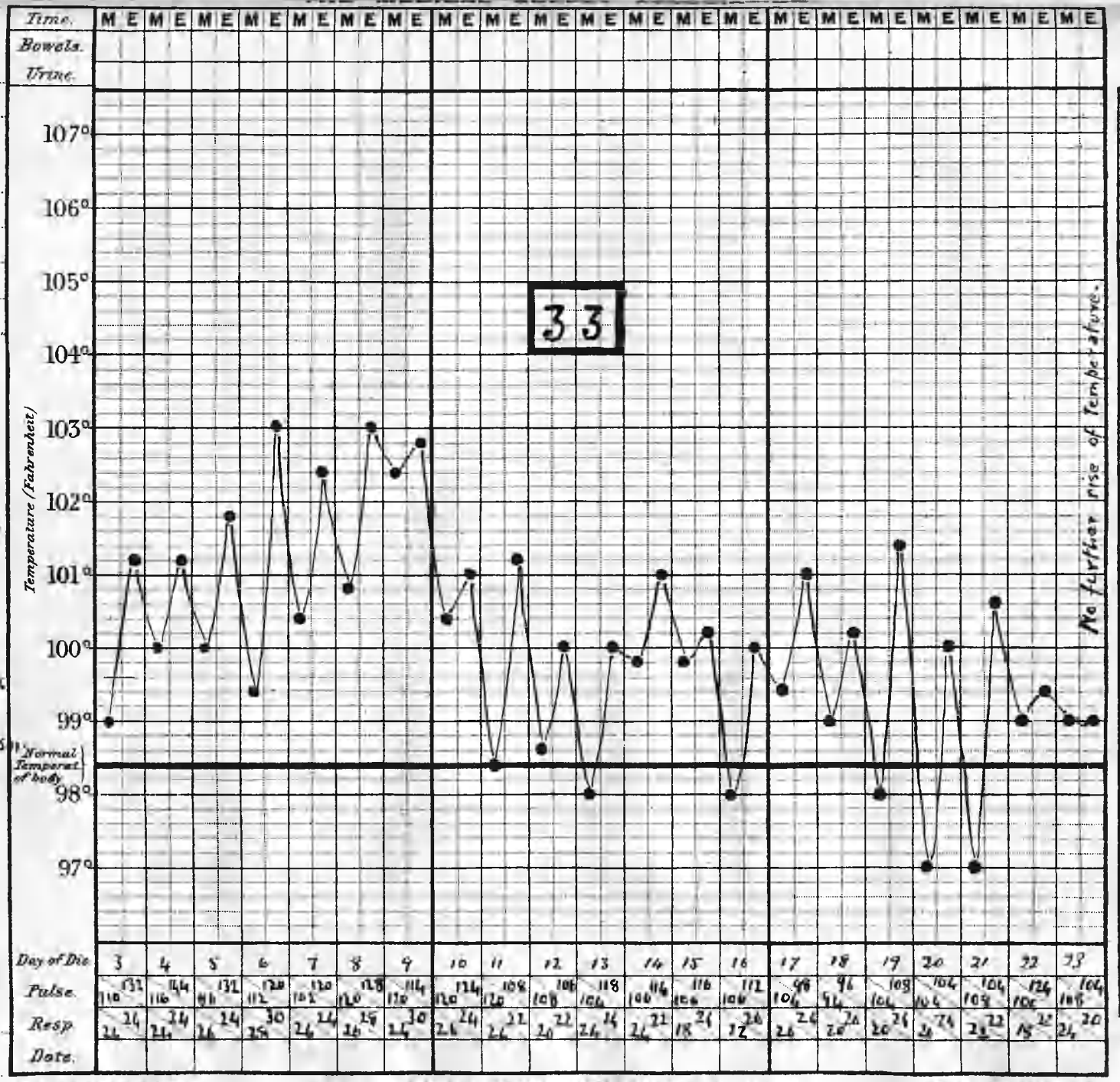
Case Book N° 33

Severe burns (as on Chart overleaf) No signs in chest. On 6th day (critical period for burns) the patient developed hysterical paralysis of both legs.

On the 12th day ulcers developed on both corneae, although by this time there was little blepharospasm. Burns healed on 28th day

Date of admission

Result



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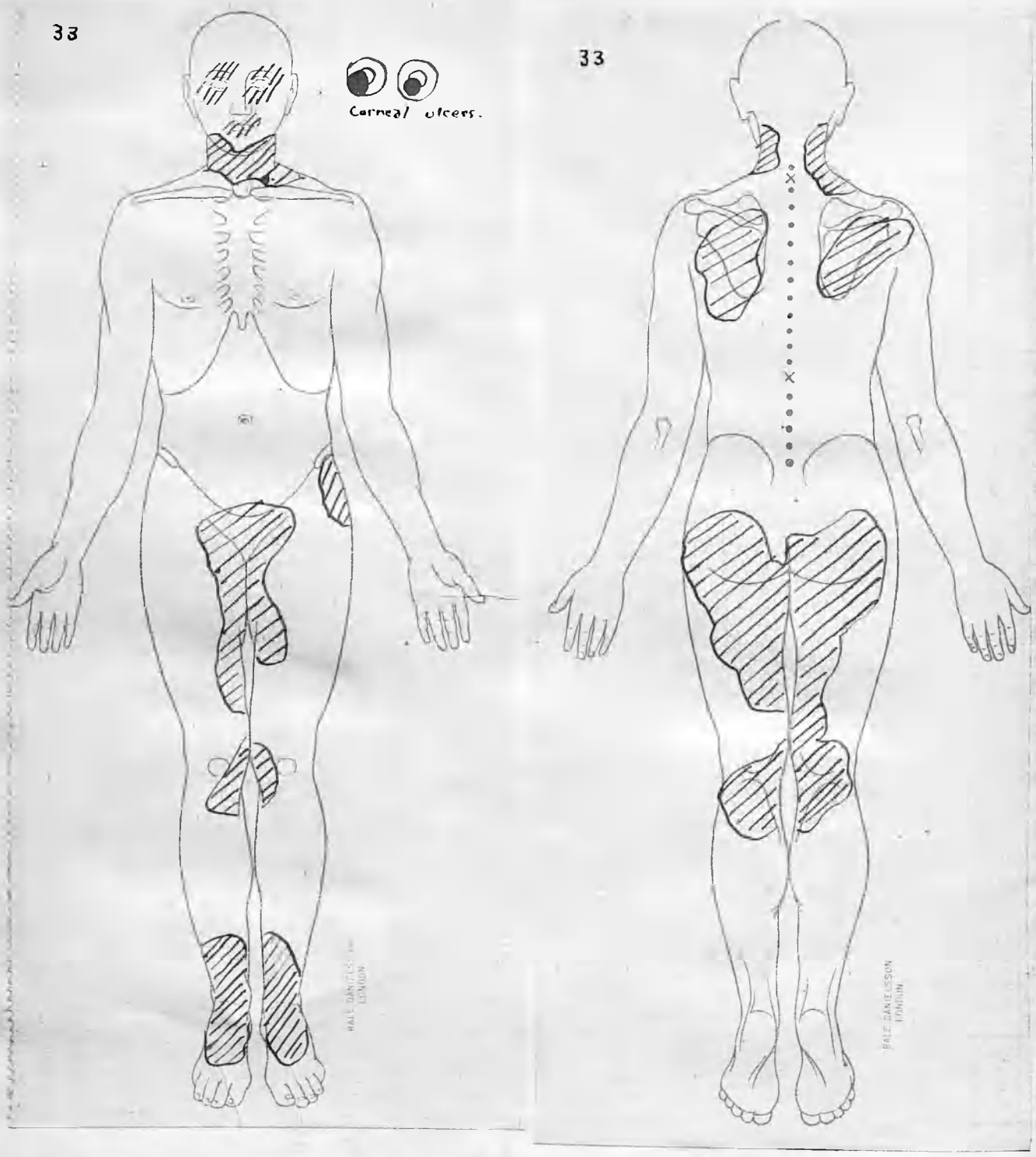


SKIN LESIONS — CASE 33.

DISEASE.

Notes of Case.

Name

Age

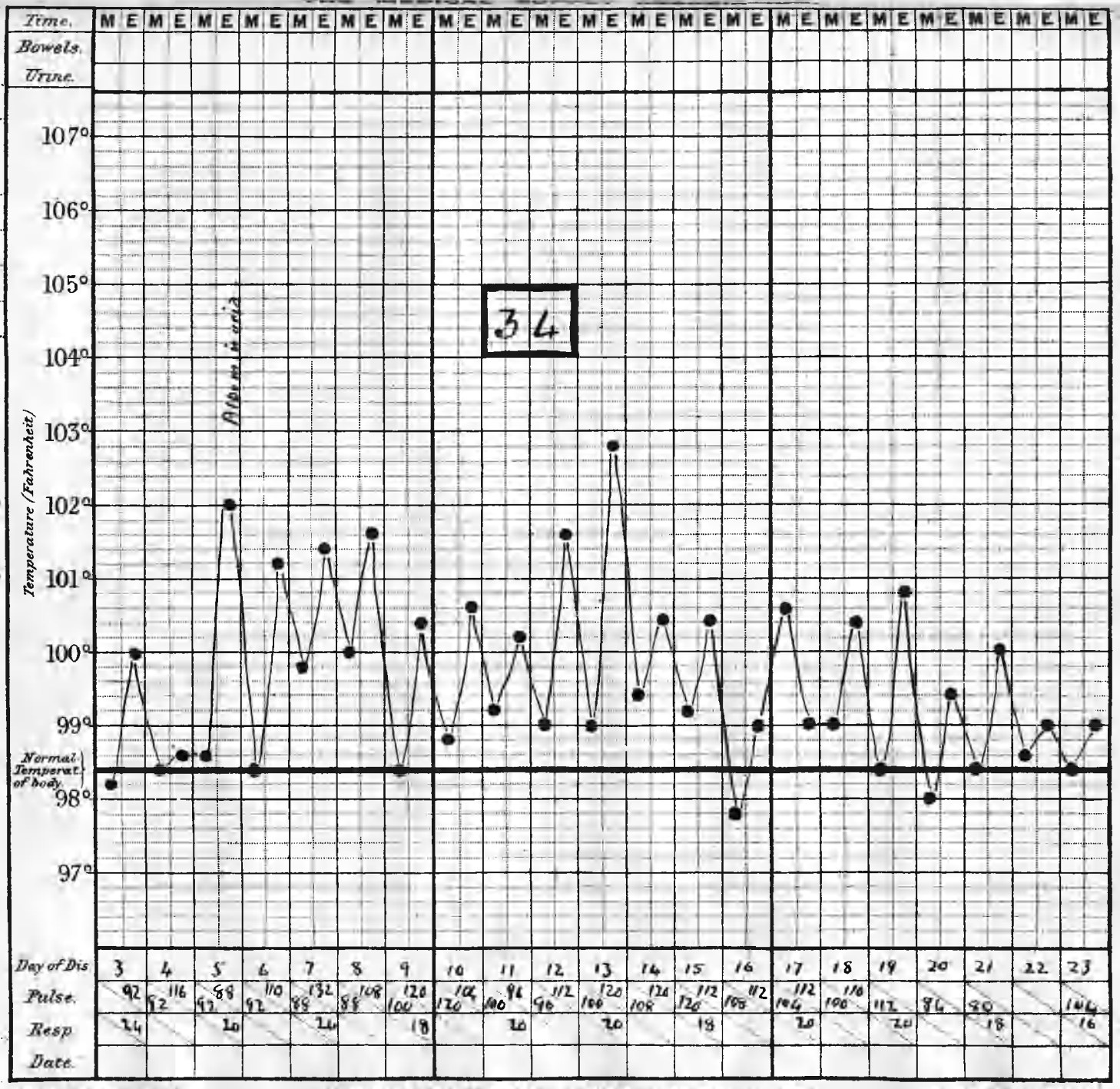
Diet

Case Book N° 34

Isolated severe burn of back from direct contact with the liquid. No injury to eyes or Respiratory tract. Healing complete on 28th day.

Date of admission.

Result



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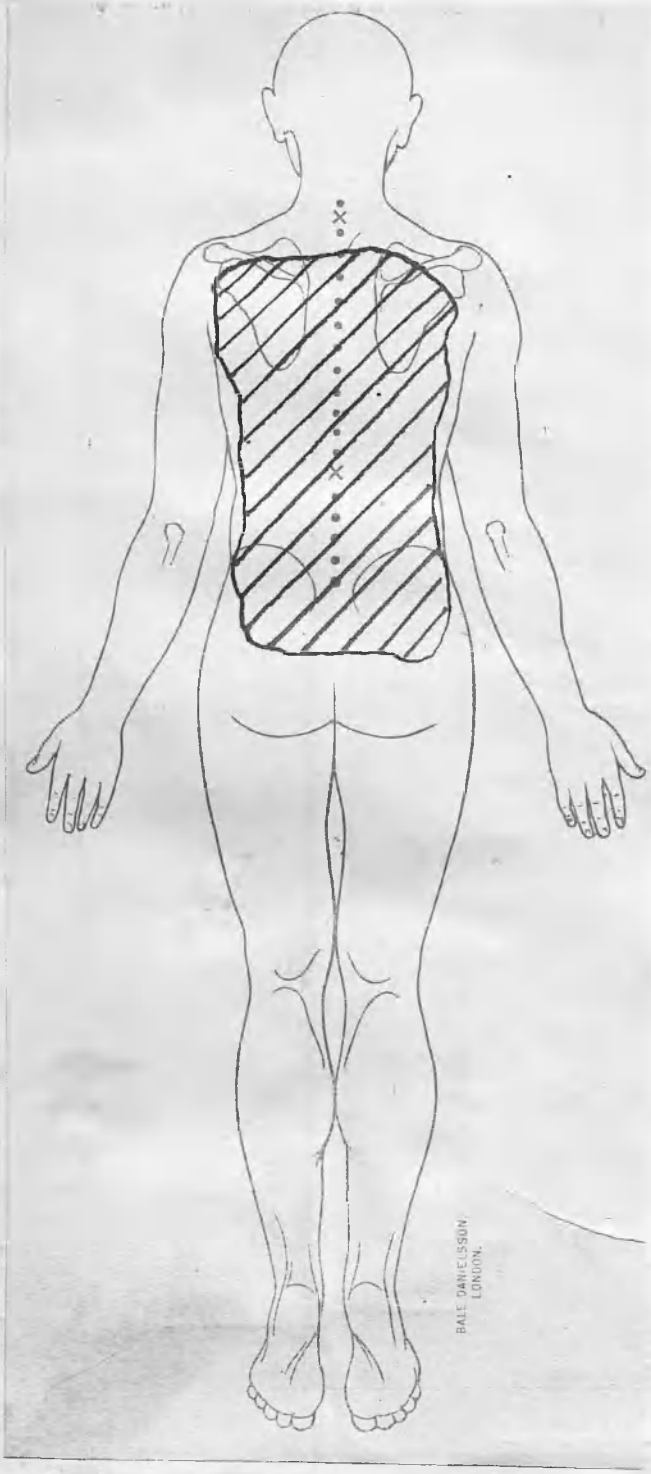


CASE 34.

UNCOMPLICATED BURN

DISEASE.

Notes of Case.

Name

Age

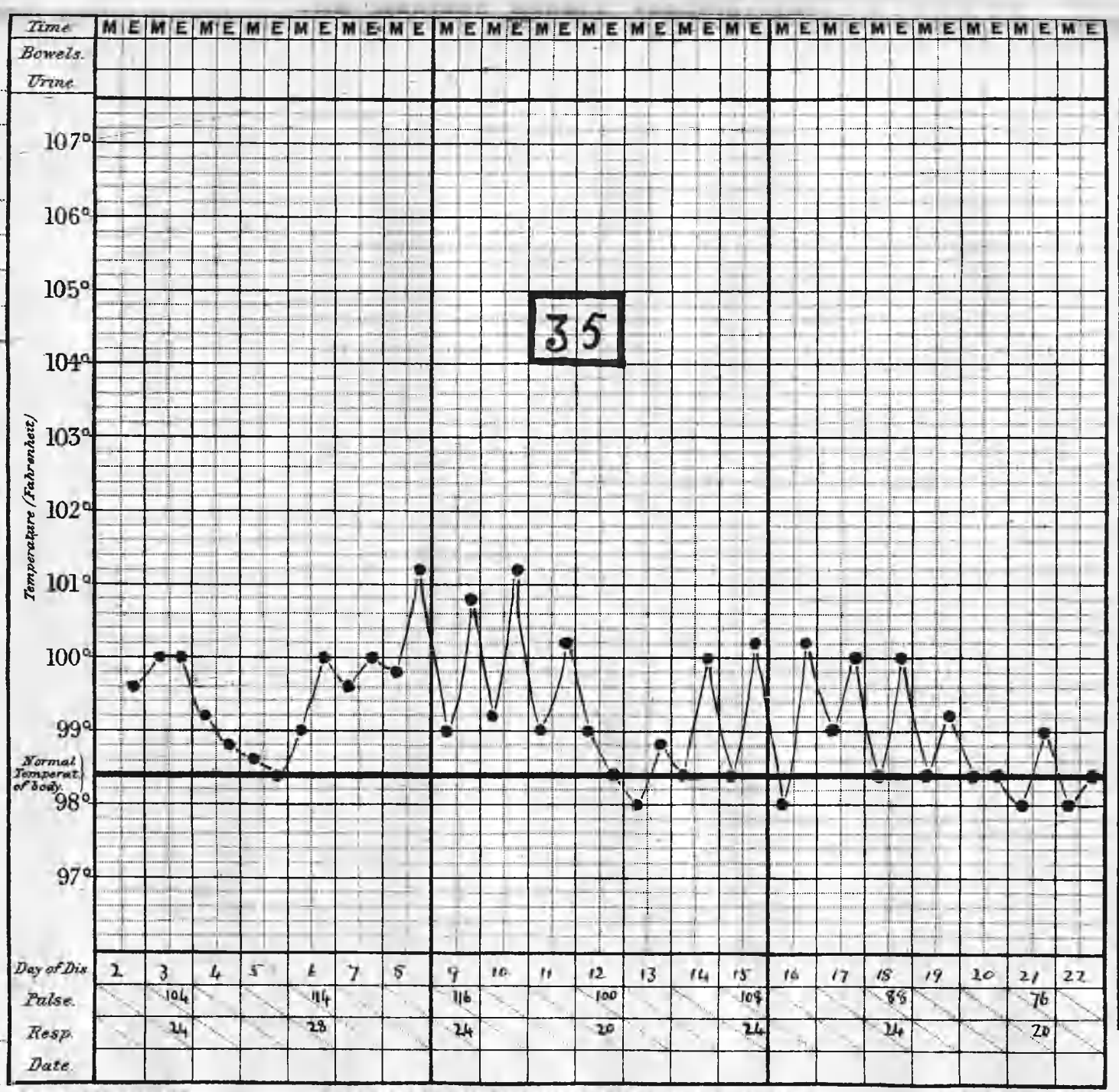
Diet

Case Book N° 35

A case of uncomplicated Burns of Moderate severity — typical of the majority — Complete healing by 22nd day.

Date of admission.

Result



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DISEASE.

Notes of Case.

Name

Age

Diet

Case Book No. 36

Acase of severe burns, terminating with pulmonary infarction. (Quoted in text).

Date of admission.

Result

Time.	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
Bowels.																								
Urine.																								
Day of Dis.	3		4		5		6		7		8		9		10									
Pulse	134 / 112		118 / 124		116 / 120		108 / 110		118 / 104		116 / 112		138 / 116		160 / 148									
Resp.	36 / 28		30 / 30		32 / 30		28 / 28		32 / 30		40 / 28		40 / 40		52 / 48									
Date.																								

Temperature (Fahrenheit): 107°, 106°, 105°, 104°, 103°, 102°, 101°, 100°, 99°, *Normal Temperat. of body.* 98°, 97°

Temperature (Centigrade): 42°, 41°, 40°, 39°, 38°, 37°, 36°, 35°

Sudden change in Respirations

Died.

36

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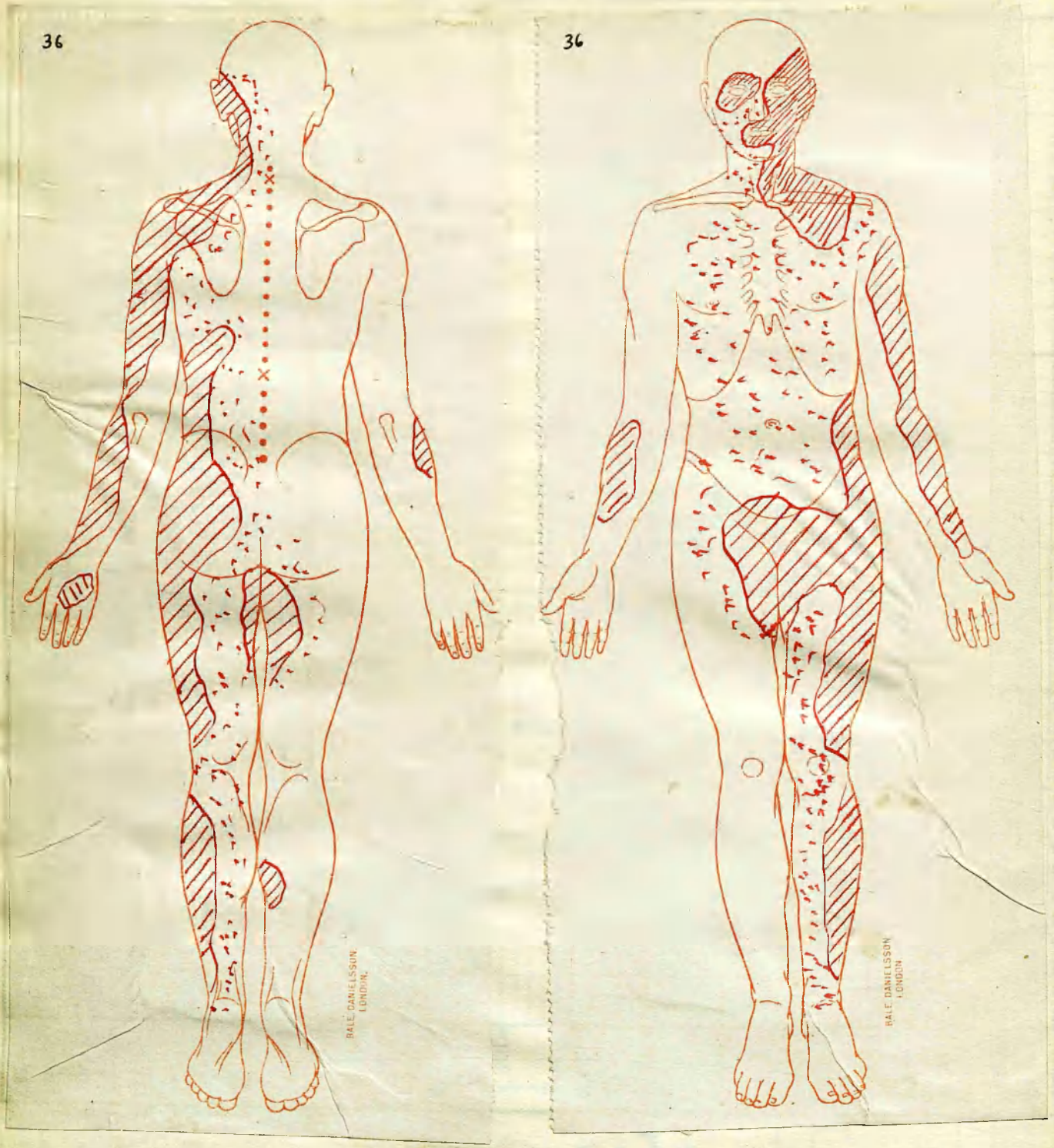
Gould's Clinical Chart



SKIN LESIONS — CASE 36.

DISEASE.

Notes of Case.

Name { Pte B.

Age

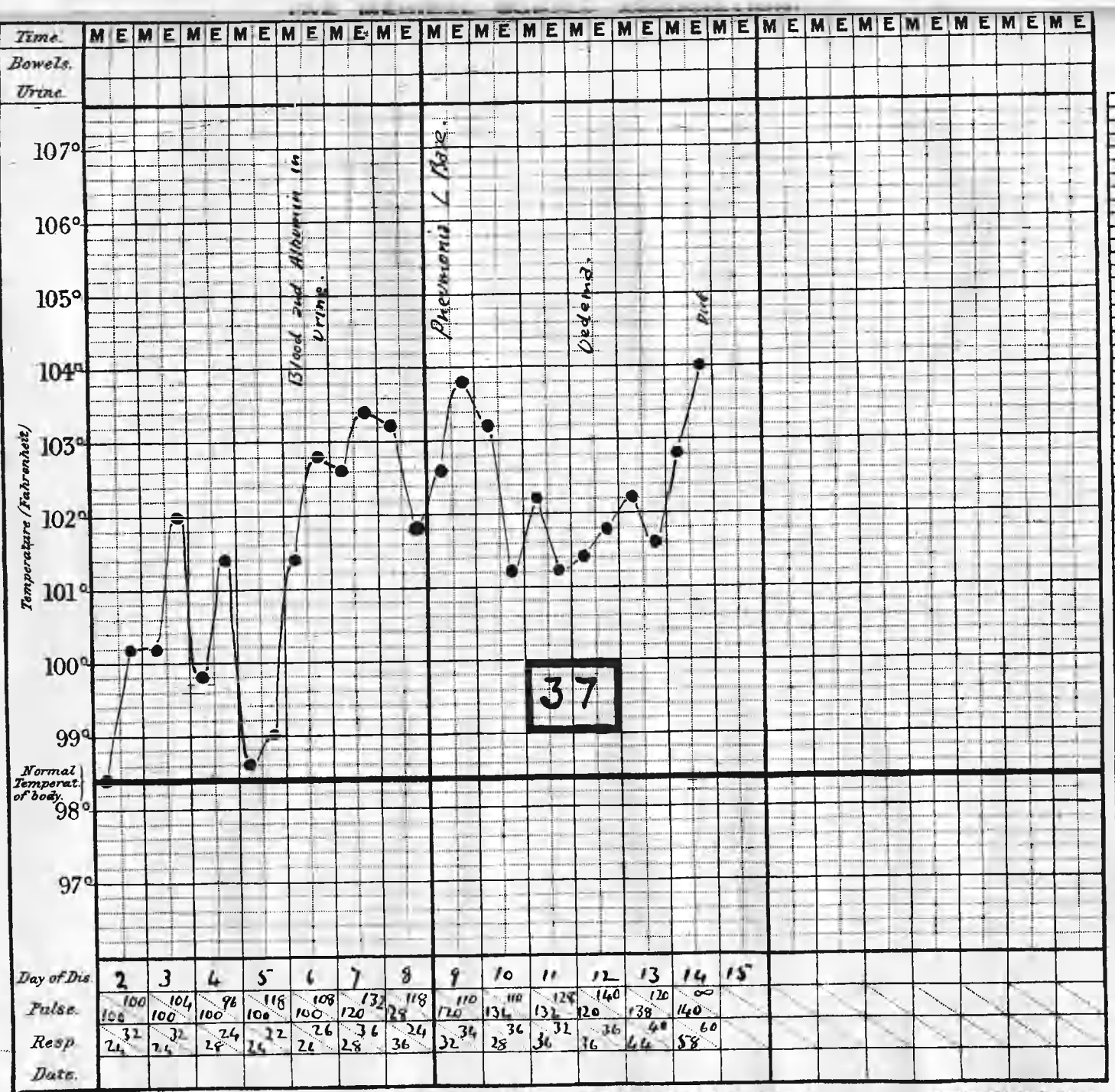
Diet

Case Book No. 37

SEVERE BURNS.
HAEMORRHAGIC
NEPHRITIS (6th DAY)
LEFT BASAL
BRONCHOPNEUMONIA
(diagnosed definitely
on 9th day).

Date of admission.

Result



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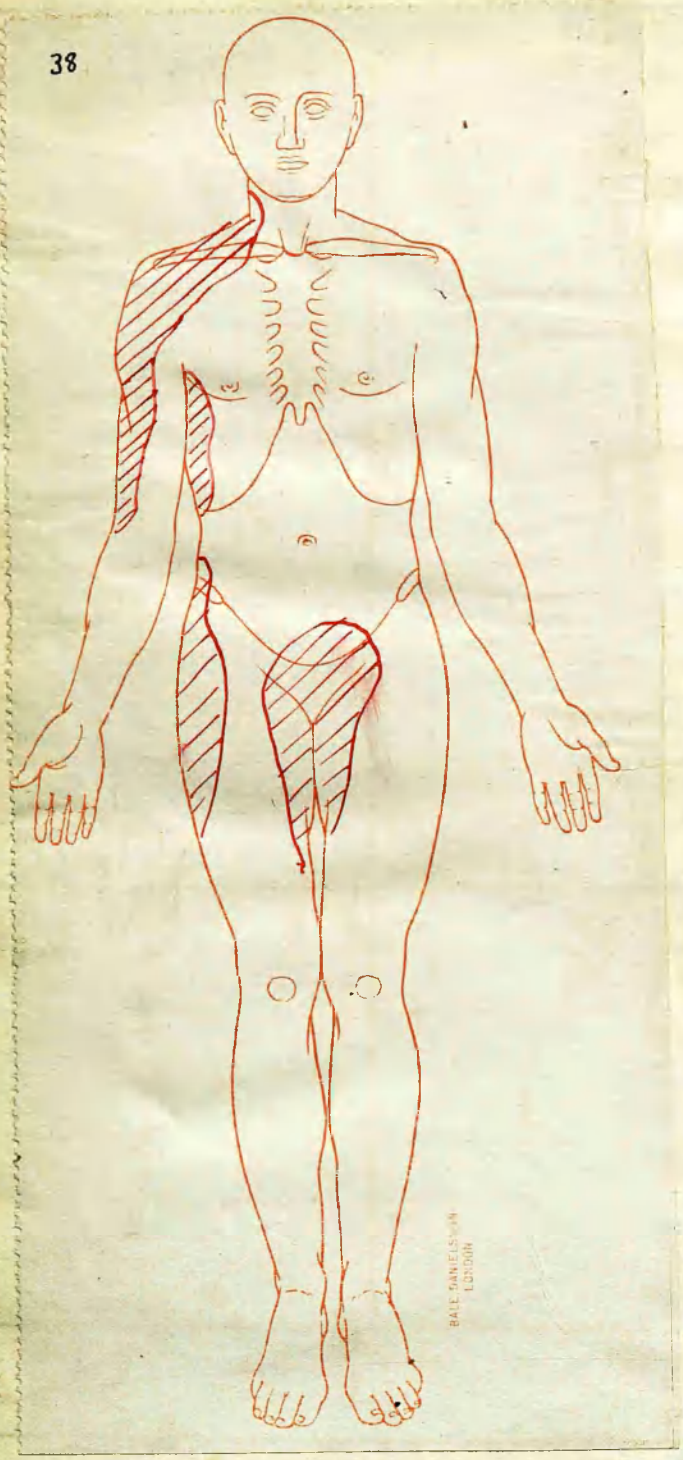
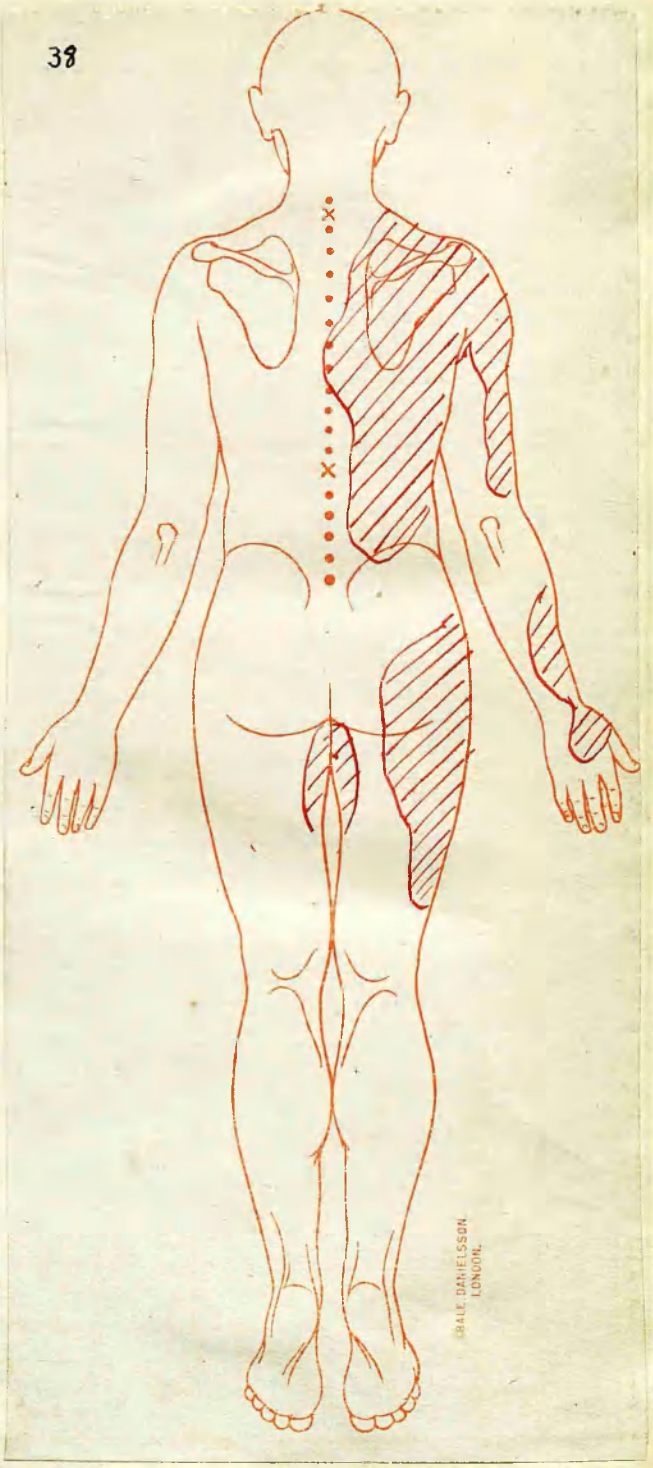




SKIN LESIONS — CASE 38

DISEASE.

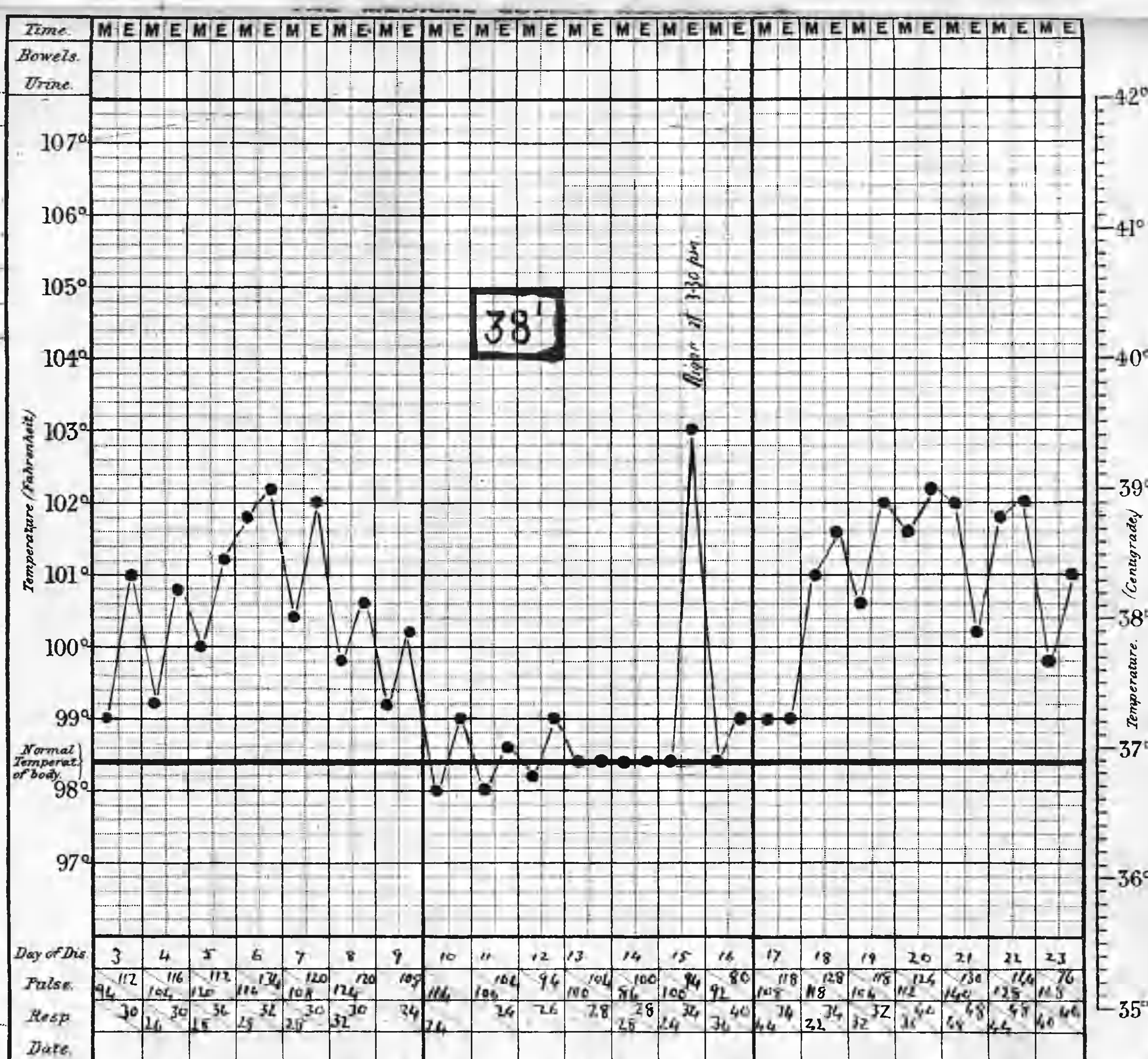
Notes of Case.

Name

Age

Diet

Case Book No. 38



Date of admission.

Result

DISEASE.

Notes of Case.

Name

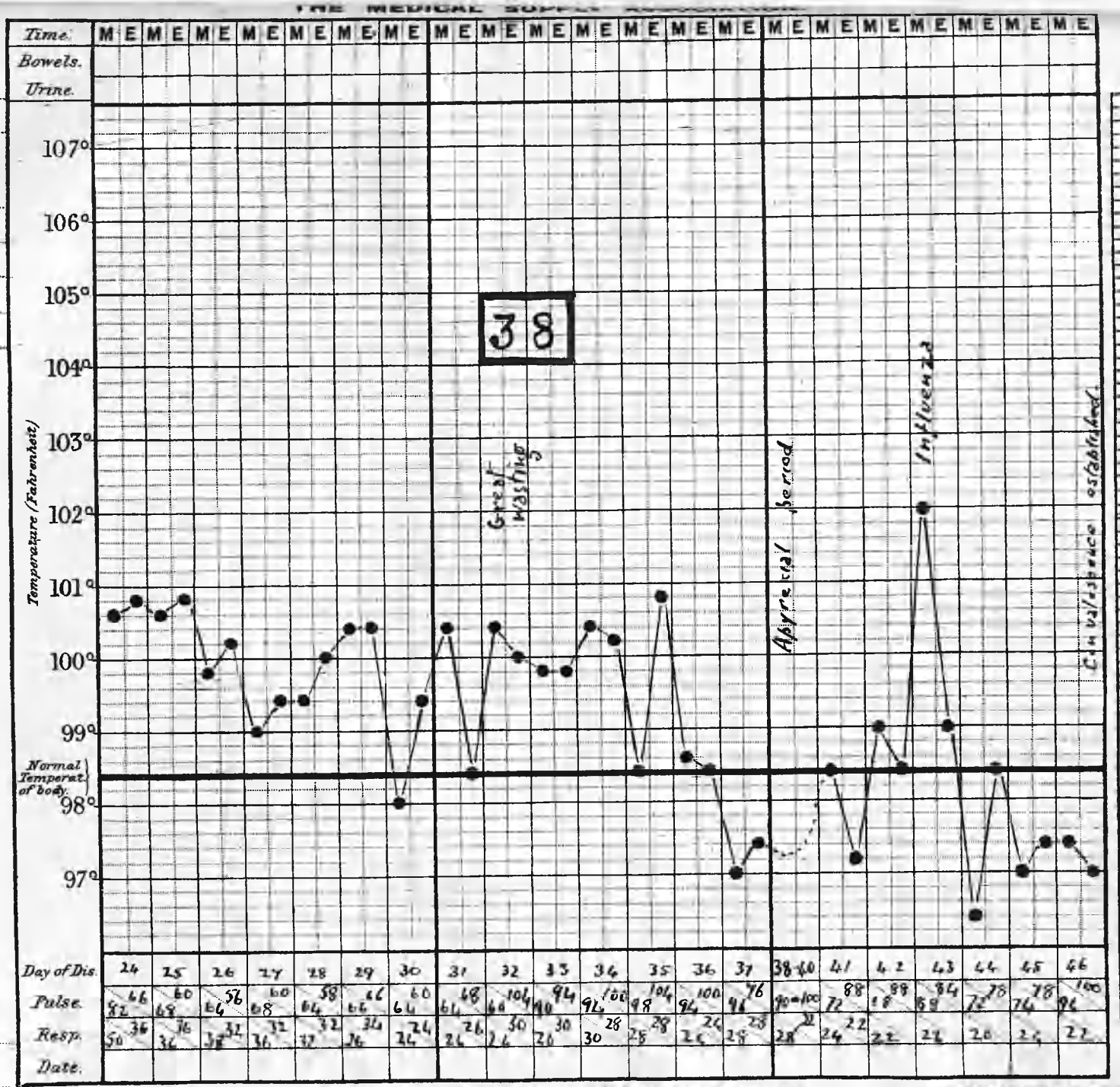
Age

Diet

Case Book No. 38

Date of admission.

Result



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