S T U D I E S I N D I P H T H E R I A

with particular reference to
Suprarenal Cortex Deficiency and
to the Use of Sodium Chloride in Treatment.

-oOo-

by

ALEXANDER MACLEAN, D.P.H., M.B., Ch.B.

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

1 9 3 5

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

INTRODUCTION.

This work is based on clinical, biochemical and pathological observations on diphtheria patients who were under my care in the wards of Ruchill Fever Hospital, Glasgow, between April, 1933, and April, 1935. It is intended to show, in the first place, that the effects, which, in the acute stage of diphtheria, are ascribed to absorption of diphtheria toxin, are similar to effects which are known to be associated with deficiency of the secretion of the cortical part of the suprarenal glands, and in the second place, that a method of treatment, which has been proved to be of value in conditions characterized by suprarenal cortical deficiency, can also be utilized with benefit in diphtheria.

The acute stage of diphtheria is characterised by general and local symptoms. The general symptoms are attributable to absorption into the circulation of toxins elaborated by the organisms which cause the disease. The local symptoms result from tissue reactions to the action of toxins at the site where the infecting organisms are present.

The general symptoms can be classified according to

the manner in which they appear to affect the nervous system, the cardiovascular system and the kidneys.

(a) The effect on the nervous system in the acute stage is the production of lassitude or prostration. Even in the mildest case there is an element of apathy or listlessness, which is evident from the lack of resentment to examination and from the fact that the patient submits to the injection of serum with a minimum of struggling in contrast, for example, to a patient suffering from scarlet fever. The degree of lassitude or prostration appears to vary with the severity of the toxaemia and is extreme in critical cases.

(b) The effect on the cardiovascular system is shown by palpation of the pulse and by physical examination of the heart. The pulse is rapid, soft and easily compressible. In extreme cases there is enlargement of the precordial dulness due to dilatation of the heart, and there may be murmurs due to valvular incompetence. Normally, the first heart sound is long and dull and the second sound is short and clear; in diphtheria, the contracting heart muscle does not make its normal contribution to the composition of the first heart sound which, in consequence, becomes short and sharp and approximates

in character to the second heart sound. The poor action of the heart is also shown by the tendency towards pallor of the face and mucous membranes and towards cyanosis of the lips and finger-tips. Again, the risk of sudden cardiac failure with collapse is present and makes necessary the nursing of the patient in the horizontal position and the avoidance, on his part, of sudden movements.

(c) The effect on the kidneys is shown by the presence in the more severe cases of albuminuria.

Estimates of intoxication in diphtheria, based on clinical observations, have been made, notably by van Bie,(1) who divides cases into three groups, A, B and C, according to the degree of prostration, the extent of cardiac involvement and the amount of albuminuria when considered with the state of foetor of the breath, the severity of lymphadenitis in the neck and the quantity of nasal discharge.

The local symptoms depend on the site and extent of the local lesion. This lesion consists of the presence of a greyish pseudo-membrane, which is formed as a reaction of the patient's tissues to the presence of the causative organisms and is found usually on the mucous membrane of the upper respiratory or alimentary tracts

or, less commonly, on the conjunctiva, external genitalia or wounds. Typically, the pseudo-membrane is present on the mucous membrane in the faucial region and, according to its extent in this situation, cases of diphtheria have been classified by Van Bie (2) in eight groups as follows:

Group 1 - a slight amount of pseudo-membrane is present on the tonsils,

Group 2 - pseudo-membrane is more extensive but does not dover the tonsils,

Group 3 - pseudo-membrane covers the tonsils completely,

Group 4 - pseudo-membrane covers the tonsils and spreads beyond the tonsillar surfaces,

Group 5 - pseudo-membrane covers the tonsils, spreads beyond the tonsillar surfaces and involves the margin of the soft palate,

Group 6 - pseudo-membrane covers the tonsils, spreads beyond the tonsillar surfaces and involves the margin of the soft palate and the uvula,

Group 7 - pseudo-membrane covers the tonsils, spreads beyond the tonsillar surfaces and involves the greater part of the soft palate and uvula,

Group 8 - pseudo-membrane covers the tonsils, spreads beyond the tonsillar surfaces and involves the uvula and the soft palate as far forward as the teeth and the posterior margin of the hard palate.

The local lesion is not at first accompanied by symptoms of discomfort and it is not infrequent to find even a very severe case of diphtheria with a relatively slight complaint. An inflammatory reaction takes place in the regional lymphatic glands, and causes painful swelling of those organs which varies in size and degree of pain with the extent of the local lesion and with the occurrence of secondary infection with other organisms than those which caused the initial lesion. The presence of pseudo-membrane in the mouth, nose or throat causes a characteristic foetid odour to be given to the breath. A sanious nasal discharge is an accompaniment of pseudo-membrane in the nasal cavities.

Neither of the two methods used by Van Bie for estimating severity in the acute stage of diphtheria is entirely satisfactory. The classification based on an estimate of general intoxication fails in that cases with an extensive local lesion occasionally show very little sign of intoxication and yet their clinical condition in the recovery stage is such as usually succeeds a severe attack of diphtheria, and vice versa. Again, the classification derived from the extent of the pseudo-membrane in the faucial region has several drawbacks. In the first

place, cases of diphtheria in which the faucial structures are free from pseudo-membrane are excluded; these include cases of diphtheria of the nose, pharynx, larynx, conjunctiva, genitalia and wounds, which would be included in the classification based on general symptoms of intoxication. In the second place, cases of diphtheria with slight faucial pseudo-membrane and, therefore, belonging to one of the least severe groups in the classification based on the extent of pseudo-membrane, will, if there is a diphtheritic lesion in another region, probably show signs which will warrant placing them in one of the more severe groups in the classification based on general intoxication.

In the present investigation, the severity of the acute stage of each case was assessed by a method based on both the methods introduced by Van Bie. As regards toxaemia, cases were included in Group A if apathy was slight, if the heart sounds were of good tone and tachycardia was absent (taking into account an increase of pulse rate commensurate with the pyrexia of the patient and, also, the age of the patient) and if albuminuria was not found; they were placed in Group C if prostration was evident, if the muscular component of the first heart

sound was poor and tachycardia was considerable or if albuminuria was severe; intermediate cases were included in Group B. In estimating the extent of faucial pseudo-membrane, Van Bie's classification was followed with the exception that when a lesion was mainly unilateral the grouping was estimated for each half of the throat as if the other half were similarly covered with membrane and the average value taken. The fourteen groups actually obtained in this manner were combined, as in the following table, into four main groups containing respectively MILD CASES, MODERATE CASES, SEVERE CASES and VERY SEVERE CASES:

TABLE I.

Classification of Cases of Diphtheria according to the Severity of the Acute Stage.		Groupings of the same Cases according to Van Bie's Classifications.
Group	Clinical Description	
I	Mild	A1, A2, B1.
II	Moderate	B2, B3, B4, B5, C2.
III	Severe	C3, C4, C5.
IV	Very Severe	C6, C7, C8.

This classification, therefore, excludes from consideration all cases in which a faucial lesion was absent and also cases which were only detained in the wards as being

carriers of diphtheria organisms. In addition it was decided to discount patients in whom there was a small faucial lesion and also a laryngeal lesion because in those patients there was usually very little general intoxication, the illness of the patient being due in the main to the concomitant embarrassment of respiration.

The patient was usually given an adequate dose of diphtheria antitoxin soon after he came under treatment; later the symptoms of intoxication disappeared and the extension of the pseudo-membrane ceased with the development of immunity to the toxin of the disease. In the series of cases dealt with here, the average quantity of serum given was as shown in the following table:

TABLE II.

Classification of Cases of Diphtheria according to the Severity of the Acute Stage.		Amount of Diphtheria Antitoxin employed in Antitoxic Units.
Group	Clinical Description	
I	Mild	4,000 - 12,000
II	Moderate	16,000 - 24,000
III	Severe	28,000 - 36,000
IV	Very Severe	40,000 - 200,000

In the majority of cases, a single dose of antitoxin was sufficient to produce a satisfactory change in the

patient's condition; in the remainder, subsequent doses of antitoxin were necessary before the desired effect was attained. The method of intramuscular injection was invariably used for the administration of antitoxin to cases belonging to Groups I, II and III. In Group IV cases, an attempt was made to give at least half the initial dose of antitoxin by intravenous injection and the remainder by intramuscular injection; if subsequent injections of antitoxin were necessary the intramuscular route was chosen.

The results of treatment in the present investigation were assessed by placing the cases in the following eight categories, according to increasing gravity of the ultimate issue:

(1) Satisfactory recovery.
(2) Delayed recovery.
(3) Complicated recovery.
(4) Death after treatment for more than fourteen days.
(5) Death after treatment from the eighth to the fourteenth day, inclusive.
(6) Death after treatment from the fifth to the seventh days, inclusive.
(7) Death after treatment from the second to the fourth days, inclusive.
(8) Death after less than twenty-four hours' treatment.

Cases of SATISFACTORY RECOVERY included all cases in which the patient recovered, did not develop paralysis

and was fit to be dismissed from hospital before the fiftieth day of treatment, or would have been so but for the occurrence of intercurrent illness, such as chickenpox and acute appendicitis, or for the persistence of corynebacterium diphtheriae on the faucial or nasal mucous membrane or in pathological discharges.

Cases of DELAYED RECOVERY included all cases in which the patient recovered and did not develop paralysis but was not, in the physician's opinion, sufficiently recovered to be dismissed until the fiftieth day of treatment or later, always excepting a reason such as the occurrence of intercurrent illness or the persistent presence of C.diphtheriae in cultures. The causes of the delay were usually of an indefinite nature and included such symptoms as slight apathy, anorexia, slight tachycardia or slight bradycardia, the presence of occasional extra-systoles when the pulse rate was slightly increased (thus excepting sinus arrhythmia in children) and albuminuria after the acute stage.

Cases of COMPLICATED RECOVERY included all cases in which the patient recovered and was dismissed after passing through a stage in which cardiac paralysis, muscular paralysis or both occurred.

The cases in the category of COMPLICATED RECOVERY included cases of recovery with mild paralysis and cases of recovery with severe paralysis. Cases were placed in the former subdivision if either mild cardiac paralysis or mild muscular paralysis or both were present and there was no form of severe paralysis; mild cardiac paralysis was taken to be present if after the middle of the second week of treatment there were definite irregularity of the heart beat, poor quality of heart sounds, and either tachycardia or bradycardia; mild muscular paralysis was present if there were symptoms of palatal paralysis or of paralysis of the extrinsic or intrinsic muscles of the eye, or if the gait of the patient when permitted to walk was sudh as to suggest that there was abnormal weakness of the muscles of the lower limbs consistent with a recent paralysis of these muscles. Cases were regarded as being cases of severe paralysis if either severe cardiac paralysis or severe muscular paralysis or both were present whether there was also mild paralysis or not; severe cardiac paralysis was taken to mean that paralysis, such as has been described above as mild cardiac paralysis, was accompanied by signs of cardiac failure such as oedema of the back or extremities, precordial pain, bron-

chitis or albuminuria; severe muscular paralysis included paralysis of the muscles of the pharynx and of the diaphragm; actually, no cases of diaphragmatic paralysis occurred in this investigation.

The division of the fatal cases according to the time of death was made arbitrarily with the exception that the inclusion of the end of the first day as a defining date was made because it was thought that any method of treatment that was employed would have very little chance to exert its influence in cases which ended fatally before that date.

A diphtheria patient who makes a satisfactory recovery is kept lying flat in bed for the first three weeks of treatment. During the fourth week he is permitted to sit up in bed and, during the fifth and sixth weeks, to walk about. He is dismissed from hospital at the end of the sixth week or during the seventh week if he is no longer found to be infectious.

If recovery is interrupted by the development of cardiac or muscular paralyses, the patient must be kept lying flat in bed until all paralysis has disappeared. It is customary to give such a patient by mouth a mixture containing a small quantity of strychnine (e.g. the equi-

valent of one-twentieth grain of strychnine hydrochloride thrice daily) and, sometimes, containing also a small quantity of adrenalin (e.g. five to ten minims of liquor adrenalini hydrochloricus thrice daily); if paralysis of severe type is present, hypodermic injections thrice daily of a hundred-and-twentieth grain to a sixtieth grain of strychnine, according to the age of the patient, are given. Strong coffee is frequently given at a late stage when recovery is retarded by the persistence of slight cardiac irregularity or tachycardia.

Very severe cases may die in an early stage as a result of the direct action of toxins on the cells of important organs, or at a later stage from cardiac paralysis or from paralysis of the diaphragm or pharyngeal muscles with resulting respiratory embarrassment. Very severe cases which recover usually do so only after a protracted illness often characterized by paralysis of severe type.

On the following pages, the subject matter is arranged in four sections. In the first section a description of suprarenal cortex deficiency is given as it has been observed by various workers in animal experiments and in diseases of the human subject. It is shown

in the second section, that there are several points of semblance, including certain clinical, biochemical and pathological changes, between cases of diphtheria and cases in which there is evidence of suprarenal cortex deficiency. Evidence is given in the third section that the intensive administration of sodium chloride, which is of definite value in conditions characterized by suprarenal cortex deficiency, is also an important method of treatment in diphtheria. The results of the investigation are summarised in the fourth section and certain conclusions are reached.

Photographs, a diagram, graphs of blood sugar curves and various tables are interspersed at appropriate places in the text. A bibliography is given at the end of the subject proper. The biochemical methods used are described in the Appendix which also contains tables of the detailed results of the treatment described in the third section.

Section I.

SUPRARENAL CORTEX DEFICIENCY.

In this section a description is given of recent work on Suprarenal Cortex Deficiency as observed both in animal experiments and in conditions of disease in the human subject.

A. Experimental Suprarenal Cortex Deficiency in Animals.

In the past five years much experimental work has been performed and many observations made, notably by American investigators, on animals which have had both suprarenal glands removed at operation. This research has been greatly advanced by the use of potent extracts of suprarenal gland freed from the secretion of the medullary portion of the gland by special treatment. Such extracts have been prepared by Rogoff and Stewart (3) and by Swingle and Pfiffner (4), among others, and, under certain circumstances, they can make good deficiency of secretion from the suprarenal cortex.

Many years ago, Brown-Séquard (5) observed that, if both suprarenal glands were completely destroyed, death followed after eight hours in mice, nine hours in rabbits, thirteen hours in guineapigs and fourteen hours in cats

and dogs; if one suprarenal gland only was extirpated the period of survival was not usually less than twenty-five hours.

Hartman, Brownell and Lockwood (6) found that when both suprarenal glands were removed in an animal a characteristic train of symptoms occurred. The animal at first tired easily and had no inclination for exercise; later there was loss of appetite with vomiting and, perhaps, haemorrhages; next there was a stage of inertia verging on coma or else the animal was irrational and easily irritated; coma followed and, finally, there was sudden collapse and death supervened. The important discovery was also made that the administration of cortical extract at any point, before the final stage was reached, caused the symptoms to disappear; they passed off in the reverse order of their appearance. Such an animal could be maintained in normal health by being given a daily ration of cortical extract.

Further they found that an animal, which was kept in a state of partial cortical deficiency by bilateral suprarenalectomy followed by daily administration of cortical extract in an insufficient quantity to maintain health, presented the following features:

(i) there was extreme muscular weakness;
(ii) there were usually loss of appetite, vomiting and diarrhoea;
(iii) there was a lowering of general bodily activity, shown by a fall in temperature, lowering of the blood-pressure and diminution of the basal metabolism;
(iv) there were increased excretion of urea and chloride in the urine and decreased excretion of water; (there was also decreased intake of water);
(v) there was increased concentration of the blood with increased blood cell volume, increased erythrocyte count and decreased leucocyte count;
(vi) distinctive changes occurred in the chemical constitution of the blood, viz,

diminution of the plasma total base,
diminution of the plasma sodium,
diminution of the plasma chlorine,
diminution of the plasma bicarbonate,
increase of the plasma potassium,
increase of the plasma magnesium,
increase of the plasma inorganic sulphate,
increase of the plasma inorganic phosphate,
decrease of the blood sugar, and
increase of the blood non-protein nitrogen.
(No change occurred in the plasma calcium)
(7)

From similar observations, Britton and Silvette argued that as regards such animals, two facts stood out clearly:

(a) that the suprarenalectomized animal with symptoms of deficiency showed an almost complete and thus very critical depletion of its circulating and reserve carbohydrate materials;

(b) that under the same circumstances there was no lack of water in the animal's body; there was a loss of fluid from the blood stream which was balanced by an increase of water in the hepatic and in the muscular tissues.

Harrop, Widenhorn and Weinstein (8) concluded that the suprarenal cortex had a direct hormonal influence on the function of the kidneys particularly as regards excretion of water and of nitrogen.

Harrop, Weinstein, Soffer and Trescher (9) found that suprarenalectomized dogs which received injections of sodium chloride solutions survived longer than similar animals which were not treated in this manner. They noticed also that suprarenalectomized animals, with well-healed wounds and maintained in good condition by injections of cortical extract, when thrown into a state of deficiency by the withdrawal of these injections showed a typical course characterized by gradual haemoconcentration, increase of the blood non-protein nitrogen and increase of the blood proteins; later, there were fall of blood-pressure, diminution of basal oxygen consumption, diminished blood-flow, lowered bodily temperature and circulatory collapse. These authors regarded haemoconcentration as estimated from increased blood-oxygen capacity, increased erythrocyte count and increased volume of packed red cells, as the primary event in the sequence of symptoms; accompanying it there were falls in the total plasma base, plasma sodium and plasma chlorine and marked increases in the urinary out-

put of sodium and chlorine, the volume of urine excreted being maintained at a normal figure until within a few hours of death. Their conclusions were that the suprarenal cortex exercised a regulatory influence over the excretion of water, sodium and chlorine and that, when this was removed, the kidneys wasted those components not alone from the blood plasma but also in even greater amounts from the extravascular tissue fluids; the resulting dehydration was responsible for the symptoms and fatal ending of suprarenal cortex deficiency following the withdrawal of cortical extract.

Arguing further, Harrop, Weinstein, Soffer and Trescher considered that, if the regulatory influence over the excretion of sodium and chlorine was lost after injections of cortical extract were stopped, it was probable that the animal would go into a state of cortical deficiency much more readily on a salt-poor diet than when the salt-intake was normal. Conversely, an animal on a high salt-intake should require less cortical extract for maintenance of perfect health than an animal on a normal salt-intake. Those conclusions were proved experimentally to be correct.

Again, they found that suprarenalectomized animals on a bare maintenance dosage of cortical extract, i.e.

on the verge of suprarenal cortex deficiency, showed the characteristic clinical, physiological and biochemical signs of deficiency if they were placed on a salt-free diet and, if they were not allowed to sink too deeply, they recovered after the administration of sodium chloride alone.

Examination of this literature therefore appears to show, in the first place, that the main effects of Suprarenal Cortex Deficiency in animals are,

(1) increase of the water content of the liver and of skeletal muscle, decrease of water in the circulating blood and diminution in the excretion of water in the urine;

(2) decrease of sodium and chlorine in the circulating blood and increase in the excretion of sodium and chlorine in the urine;

(3) decrease of carbohydrate in the circulating blood and of the carbohydrate reserve in the tissues.

In the second place, this condition of Suprarenal Cortex Deficiency is best treated by injections of cortical extract, the most useful adjunct to this treatment being the administration of sodium chloride.

B. Suprarenal Cortex Deficiency in Man.

The effects of deficiency of the secretion of the suprarenal cortex have been studied extensively in the condition which is now generally known as Addison's Disease.

In a paper entitled "On the Constitutional and Local Effects of Disease of the Suprarenal Capsules" and read in London in 1855, Addison (10) prefaced his remarks with the following sentence -

> "The leading and characteristic features of the morbid state to which I would direct attention are anaemia, a general languor and debility, a remarkable feebleness of the heart's action, irritability of the stomach and a peculiar change of colour of the skin, occurring in connection with a diseased condition of the suprarenal capsules."

This description has become classical and remains an accurate summary of the symptomatology of Addison's Disease.

According to Rowntree, (11) the lesion found in 80-90% of cases is tuberculosis of the suprarenal gland and in the remaining 10-20% of cases is atrophy of unexplained origin. He states that the immediate cause of the clinical manifestations is deficiency of the cortical

hormone; these appear when approximately four-fifths of the gland-substance has been destroyed by pathological processes.

The disease is rare - it occurred in 16 out of 100,000 successive admissions at the Mayo Clinic. It is seen twice as often in males as in females and is most frequent between the ages of thirty and fifty years. (11) (Rowntree).

In a series of 13 cases treated by Harrop, Weinstein, Soffer and Trescher, (12) the main symptoms noticed were

(1) pigmentation of the skin and mucous membranes;

(2) low blood pressure;

(3) symptoms of associated tuberculosis.

In addition, all presented the following characteristics:

(4) excessive weakness and liability of muscle to fatigue;

(5) low basal metabolic rate reflecting the general inanition;

(6) numbness and tingling in the extremities with pain in the back and thighs;

(7) loss of appetite;

(8) periods of nausea;

(9) loss of weight.

They also found that their patients sometimes had acute relapses or, as they are often called, Addisonian crises. These were precipitated by undue exertion, by intercurrent infection or by the administration of drugs which increased the basal metabolic rate: for example, thyroid extract.

The description now given is summarised from an article by Rowntree.(11)

> The disease usually presents three stages occurring in succession as follows:
>
> (1) A stage of inexplicable weakness and exhaustion often following upon some infection of the upper respiratory tract.
>
> (2) A stage characterized by the typical clinical syndrome with disinclination to any kind of activity.
>
> (3) A stage of collapse (Addisonian crisis) characterized by prostration or coma and by nausea and vomiting with signs of dehydration.
>
> The onset is insidious.
>
> The most prominent symptoms and signs in the usual order of their appearance are,
>
> 1. asthenia and fatigue;
> 2. pigmentation of the skin and mucous membranes;
> 3. anorexia, nausea and vomiting;
> 4. loss of weight.
> 5. arterial hypotension;
> 6. dizziness and syncopal attacks;
> 7. signs of dehydration and circulatory failure.

Although asthenia, debility and languor are usually the earliest symptoms, occasionally pigmentation may precede them. Pigmentation of the skin and mucous membranes is the most striking visible manifestation of Addison's Disease. The cause of the pigmentation is not understood but the pigment is known to be melanin.

Asthenia is invariably present; it is both subjective and objective and mental as well as physical in character; when it is pronounced the patient is bedridden.

The "remarkable feebleness of the heart's action" is shown by the presence of hypotension, circulatory failure, coolness of the body surface, fainting spells and dyspnoea. Mild hypotension is usually present throughout; the systolic blood pressure varies from 90 to 100 mm. of mercury and the diastolic blood pressure from 60 to 70 mm. of mercury. In the terminal phases and crises the systolic blood pressure is often decreased to 60 mm. of mercury.

Symptoms referable to the gastro-intestinal tract are common and usually pronounced; they include nausea, vomiting, gaseous distension and occasional intense diarrhoea. Nausea is most common in the mornings but it may be continuous. As the disease progresses, vomiting occurs and ultimately very little food may be retained. A striking loss of weight, often of 40 to 50 lbs., results and emaciation becomes obvious.

Pain in the loins is often present. As a rule, it is dull and paroxysmal in character but occasionally it is intense and accompanied by rigidity of the abdominal muscles.

Sex changes are sometimes noted, but are not important

Addison's Disease pursues a gradually downward course broken by periods of remission and by exacerbations which may attain the severity of crises.

> Acute crises or even sudden death may follow stress or strain, over-exertion, exposure, acute infections, surgical operations or the taking of purgatives or of drugs which increase the basal metabolic rate. Death may be preceded by excruciating abdominal pain, noisy delusions and dyspnoea. An acute crisis is often ushered by nausea and vomiting with pain in the back, legs or abdomen; later there follow insomnia, mental depression, poor judgment and failure of memory; next comes a stage of coma and ultimately death intervenes.
>
> Indications of early dissolution in a case of Addison's Disease are extreme asthenia, lowered bodily and surface temperature, collapse, protracted vomiting, systolic blood pressure of less than 70 mm. of mercury, blood urea of more than 60 mgm. per 100 cubic centimetres, a short rapid course without much pigmentation, poor response to treatment and the presence of active tuberculosis.

The clinical picture of suprarenal cortex deficiency as seen in Addison's Disease is accompanied by definite biochemical changes. (13) Thus Loeb, who made a complete analysis of the inorganic bases of the blood serum in patients suffering from Addison's Disease, found that the blood serum sodium in three patients had fallen from the normal figure of 138 millimolecules per litre to 131, 109 and 108 millimolecules respectively; in two of these cases the blood serum potassium had risen from the normal figure of 4.8 millimolecules per litre to 8.7 and 7.1 millimolecules per litre and the blood serum

chlorine had fallen from the normal figure of 105 millimolecules per litre to 70 and 73 millimolecules per litre respectively.

Various observers, notably Harrop [12] and Loeb [14,15] and their co-workers, have shown that the asthenic symptoms in Addison's Disease can be dispelled, in the reverse order to which they have occurred, by the administration of large doses of suprarenal cortical extract. Moreover, the symptoms, if mild, can be alleviated by the administration of sodium chloride intravenously or orally; if the symptoms are severe, the administration of sodium chloride as an adjuvant to suprarenal cortical extract enables a return to normal to be made with the use of smaller doses of the latter substance. Again, a patient with Addison's Disease, whether receiving a maintenance ration of suprarenal cortical extract or not, goes into a condition of crisis less readily on a salt-rich diet or if an extra daily quantity of sodium chloride is given than on an ordinary diet.

In a perusal of the scanty literature bearing on disease of the suprarenal glands one cannot but be impressed with the findings of Lucke, Wight and Kime [16] as detailed in a study of a hundred and twenty-six cases of

fatal influenza in the United-States during the pandemic of 1918. In their account of the pathological state of the suprarenal glands these authors noted that in three instances there were macroscopic haemorrhages in the suprarenal substance, the organ being twice its natural size. They found that in twenty cases there were no gross changes. In the other hundred and three cases there was slight increase of size and definite congestion of the suprarenal glands; the outside colour of the organs was pinkish brown and the cut surface decidedly haemorrhagic; the outer zone of the cortex was generally narrow and pale greyish-yellow, rarely presenting the deep orange-yellow tint so frequently seen in the suprarenal cortex; the intermediate zone was deep reddish-brown and exuded blood; the medulla varied from reddish-grey to deep red. Microscopically, in these last cases, extreme congestion and numerous small haemorrhages were found usually involving the medulla and intermediate zone; the cells of the cortex appeared to be swollen and usually to be devoid of lipoid granules while other cells appeared to be in a state of cloudy swelling with indefinite cell-outlines and poorly-staining nuclei; the lipoid exhaustion was observed almost constantly. The interstitial

substance was usually arranged loosely and was definitely oedematous. Focal necrosis with small round cell infiltration was occasionally observed. In a number of the gland cells, especially of the cortex, numerous deep-blue-staining coarse granules were encountered; these seemed to be identical in appearance with similar granules which were found in the liver and which were regarded by the authors as being of the same nature as granules which had been described by Adami (17) as being the final stages of bacterial destruction. Very occasionally, diffuse infiltration with polymorphonuclear cells, suggestive of acute inflammatory suprarenalitis, was seen.

The cases described by Lucke, Wight and Kime formed a representative group of fatal cases in the 1918 pandemic of influenza. When it is remembered that the principal manifestations of this disease included marked asthenia and prostration, the belief is justified that there may have been a definite relationship between the changes in the suprarenal glands and the symptomatology.

Rubinztejn (18) recorded eight illustrative cases of typhoid fever in which, he stated, suprarenal involvement occurred; this was manifested by the typhoid state, prostration, low blood pressure and the occurrence of

such symptoms as hypothermia and pseudo-perforation.

(19)

Pearl and Brunn collected twenty cases of bilateral suprarenal haemorrhage in adults in an exhaustive search of the literature from 1906 to 1928 and added one case of their own. The causes included thrombosis, capillary embolism, congestive diseases, infections and toxins, diseases of the solar plexus, surface burns and the haemorrhagic diathesis. The most common and the most characteristic of the symptoms in all the cases was asthenia. Local symptoms varied in degree from vague tenderness in the lumbar regions or epigastrium to severe abdominal pain with distension.

These authors pointed out that the presence of severe abdominal symptoms with few or no physical signs in a patient who presented definite nervous manifestations, profound asthenia and gradual decline of blood pressure was suggestive of acute suprarenal disease. They stated that the prognosis was hopeless in marked bilateral cases but that small haemorrhages might be absorbed without the occurrence of fatal results. Treatment was unsuccessful in severe cases but the best results were obtained with injections of solutions of sodium chloride and extracts of suprarenal cortex.

The literature contains a number of reports of cases of suprarenal haemorrhage in children. An article by Rabinowitz is perhaps the most informative.

The two cases reported by Rabinowitz were in a state of marked drowsiness and collapse and had a generalized purpuric eruption; post mortem, the only significant findings were in the one case, diffuse infiltration of both suprarenal bodies with bright red blood clot and, in the other, a haemorrhagic sac in the left suprarenal and an intensely haemorrhagic infiltration of the right suprarenal.

Rabinowitz believes that toxin action upon the capillary walls of the skin and suprarenals is responsible for the diapedesis of blood into the tissues in such cases.

It may be concluded that lesions of the suprarenal glands occur more often in human disease than has formerly been realised. The effects of these lesions are profound general disturbances; they are seen in a chronic phase when the morbid process in the suprarenal is slowly progressive as, for example, in the ordinary clinical state in Addison's Disease, and in an acute phase when the lesion is of sudden onset, as in suprarenal haemorrhage, or when chronically diseased suprarenal tissue has

sudden excessive demands made of it, as in the crisis stage of Addison's Disease. In both phases the best measures, whether prophylactic or therapeutic, consist of (1) the administration of suprarenal cortical extract and (2) the exhibition of sodium chloride to the patient.

Section II.

THE SUPRARENAL GLANDS IN DIPHTHERIA.

In this section it is shown that in diphtheria a state suggestive of suprarenal cortex deficiency occurs and is an important feature of the disease. Certain points in the symptomatology are dealt with first; next, an account is given of the morbid anatomy of the suprarenal glands in diphtheria, illustrative cases being reported in detail; thirdly, original experiments on the excretion of water by the kidneys in diphtheria are described; fourthly, the results of studies in carbohydrate metabolism in diphtheria are mentioned and, finally, original work on the sodium, potassium, and chlorine contents of the blood serum in cases of diphtheria is described.

A. Symptoms of Suprarenal Cortex Deficiency in Diphtheria.

There are some features of the clinical state in diphtheria which are reminiscent of suprarenal cortex deficiency, particularly of the acute form as seen in that stage of Addison's Disease to which the name Addisonian Crisis has been given.

Pyrexia is present in the acute stage of diphtheria

but is seldom high. Ker[21] may be quoted as saying: "In a pure diphtheria, if it has any relation to the local condition of the throat at all, ... the worse the lesion the lower the temperature. In septic cases it is more apt to remain moderately elevated for some days, but even here it would seem that the depressing effect of the diphtheria toxins does much to counteract the tendency of the septic infection to cause high temperature." One's own experience is that pyrexia is usually lower in the acute stage of diphtheria than in other acute infedtions with local lesions comparable to those of diphtheria. In the Addisonian Crisis the temperature of the patient is subnormal.

Many observers, including Rolleston[22] have shown that the blood pressure tends to be subnormal in diphtheria, the extent and duration of the depression bearing, as a rule, a direct relation to the severity of the local lesion. Rolleston states that the lowest readings are found in the second week of the disease and that normal tension is not recovered till the seventh week. Lowering of the blood pressure is one of the main features of conditions of suprarenal cortex deficiency, particularly in the Addisonian crisis.

A state of asthenia is present in diphtheria as in conditions in which suprarenal cortex deficiency occurs.

Feelings of dizziness and syncopal attacks are met with in diphtheria and in conditions of suprarenal cortex deficiency.

Nausea and vomiting are found in the early stages of diphtheria; for example, in carrying out an investigation on sugar tolerance in diphtheria, one found it necessary to reduce the quantity of sugar that was given by mouth from 40 grammes to 20 grammes because the administration of the former dose frequently led to a feeling of nausea and vomiting. Those symptoms occur regularly in Addison's Disease, particularly in the stage of crisis.

Pains of varying severity in the lumbar region and abdomen may be complained of by patients suffering from diphtheria in whom death is imminent; these pains are quite different from the precordial pain which is often present also at that stage; the attention of the attendant may first be drawn to their presence by the patient's relatives sitting at the bedside, to whom the patient's complaint is often made, or the attendant may only elicit in conversation with relatives after the patient's death that such pains were present. Such a complaint was made

by the patient whose case is described as Case VI on page 47. Vague pains in the small of the back and abdomen occur in the Addisonian Crisis.

Pigmentation, the true significance of which is not known, is a feature of the clinical picture in Addison's Disease; it is probably, however, to be regarded as an index of chronicity. As it did not occur in the reported cases of acute suprarenal haemorrhage, one would not expect to find it in a disease of such short duration as Diphtheria.

It appears therefore that there is a distinct similarity between the symptoms of the acute stage of Diphtheria and the symptoms of such a condition as Addison's Disease and especially of the crisis stage of that disease.

(B) The Pathological Changes in the Suprarenal Glands in Diphtheria.

In the investigation of a diphtheroid organism, the examination cannot be regarded as complete if the virulence of the organism has not been tested; a diphtheroid organism which is proved to be virulent is accepted as Corynebacterium diphtheriae. Virulence at the present time is usually decided by means of an intradermal test in a guineapig of the toxic filtrate from a culture of

the organism to be tested, the guineapig being an animal which is susceptible to the toxins of C.diphtheriae. Formerly, the method was to inject, subcutaneously, a culture of the organism and its virulence was considered to be established by the finding of haemorrhages in the suprarenal glands of the guineapig; this result was regarded as pathognomonic of a virulent culture of C.diphtheriae. Thaddea (23) has shown that injection of diphtheria toxin into animals causes changes both in the morphology and in the chemical constitution of the suprarenal cortex; haemorrhages and necrosis are found and there is disappearance of cholesterol from the cortex in greater or less degree, according to the degree of intoxication.

Post mortem examination of fatal cases of human diphtheria often reveals, in addition to the recognized changes in the liver, heart muscle, nervous tissue and other structures, characteristic changes in the suprarenal glands. Thus Muir (24) noted that poisoning with diphtheria toxin is associated with marked congestion and minute haemorrhages of the suprarenal glands. Ker (21) stated that the suprarenal glands may show degeneration in diphtheria and that haemorrhages are not infrequently observed in their substance. Rolleston (22) on the other hand, may be quoted

as follows:

> "In contrast with the well-marked haemorrhagic lesions of the suprarenals in the guinea-pig which has died of experimental diphtheria, these organs in the human subject rarely show any gross naked-eye changes, and even microscopical lesions such as cell degeneration and necrosis, small haemorrhages and necrosis, described by some writers, are not constant."

Before describing one's own findings in the suprarenal glands in fatal diphtheria, it is not out of place at this stage to give a short account of the normal state of these organs. The following summary is made from a description in Gray's Anatomy.(25)

The suprarenal glands are a pair of organs situated at the upper poles of and closely apposed to the kidneys. The right gland is often described as having the shape of a cocked hat; the left gland is semilunar and a little larger than the right gland. The average weight of each is between 3 grammes and 4 grammes.

Histologically, each gland is seen to consist of an outer cortex and an inner medulla enclosed in a fibrous capsule which sends prolongations into the interiorto form the interstitial tissue of the organ.

The capsule consists of connective tissue and some smooth muscle fibres.

The cortex consists of a fine connective tissue network imbedding glandular epithelium which is made up of polyhedral cells with rounded nuclei and with protoplasm which may contain coarse granules and lipoid granules. According to differences in the arrangement of the glandular cells, three zones are described:

(i) the outer zone, or zona glomerulosa, which is placed beneath the capsule and consists of polyhedral cells, containing spherical or oval nuclei; the cells are arranged in rounded groups and show evidences of alveolar structure;

(ii) the middle zone, or zona fasciculata, which is composed of radial columns of polygonal cells having spherical nuclei and protoplasm which contains fine granules and often globules of lipoid material;

(iii) the inner zone, or zona reticularis, which is formed of irregularly arranged columns of cells which are somewhat larger than those of the middle zone and the protoplasm of which often contains pigment-granules.

The medulla consists of a mass of large finely granular chromaffin cells permeated by venous sinusoids.

The suprarenal glands get their arterial blood-supply from,

(i) the superior suprarenal arteries, branches of the inferior phrenic arteries which arise from the aorta,

(ii) the middle suprarenal arteries, branches of the aorta, and,

(iii) the inferior suprarenal arteries, branches of the renal arteries which arise from the aorta.

These arteries anastomose in the capsule and, in the cortical part of the gland, break up into arterioles and capillaries. The latter empty into the sinusoids of the medulla and these in turn empty into the veins near the centre of the medulla. The veins in the medulla contribute to the formation of a single suprarenal vein from each gland. The right suprarenal vein enters the inferior vena cava; the left suprarenal vein joins the left renal vein which empties into the inferior vena cava.

The lymphatics follow the course of the veins and end in the lumbar group of lymphatic glands.

The numerous non-medullated nerve fibres which reach the suprarenal glands are derived from the splanchnic nerves (through the coeliac and renal plexuses) from the phrenic nerves and from the vagus nerves. They enter the lower and medial part of each capsule, traverse the cortex and end around the cells of the medulla, in which portion of the organ, numerous small ganglia develop upon the nerves.

The function of the secretion of the cortical portion

of the suprarenal gland has been indicated on an earlier page; according to Hale-White, (26) the function of the secretion of the medullary portion, which is commonly known as adrenalin, is to stimulate the myoneural junctions of the nerves of the true sympathetic system.

In thirteen cases in which a fatal issue followed diphtheritic infection and in which permission for autopsy was obtained, a special study of the morbid anatomy of the suprarenal glands was made. In one of these cases, diphtheria was merely a contributory factor to the fatal issue as the patient had shown several manifestations of a late syphilitic state and had had a strongly positive Wassermann reaction of the blood; most of the organs showed degenerative lesions; the suprarenal cortices contained islets of parenchymatous tissue separated by thickened trabecular connective tissue and haemorrhages were not noticed. Two other cases were examples of death from severe membraneus croup, the diphtheritic pseudo-membrane in each case extending into the narrower parts of the bronchial tree; the suprarenal glands had sustained no visible damage (see Figures 2 and 3). In cases of membranous croup it is to be noted that diphtheritic in-

Fig. I.

Coloured photograph of mounted specimen of left kidney and suprarenal gland from a case of malignant diphtheria. (Natural size).

Note the marked enlargement of the supra-renal gland and the extensive haemorrhages on its surface.

Fig. 2.

Photomicrograph of suprarenal gland from a case of Laryngeal Diphtheria (low power magnification). Compare with Fig.4.

Note the ease with which the three layers of the cortex can be distinguished, the prominence of the nuclei of the cortical cells and the absence of cloudy swelling and haemorrhagic infiltration in the cortex. The medulla is seen as a thin band because the section has been made near the border of the gland.

Fig. 3.

High power photomicrograph of part of the field shown in Fig.2. Compare with Fig.5.

The photomicrograph shows the capsule, zona glomerulosa, zona fasciculata and outer edge of zona reticularis. Note the absence of cloudy swelling and haemorrhagic infiltration of the cortex, and that the nuclei of the cortical cells are prominent and the cells easily differentiated from one another. There is no evidence of cloudy swelling in the capsule.

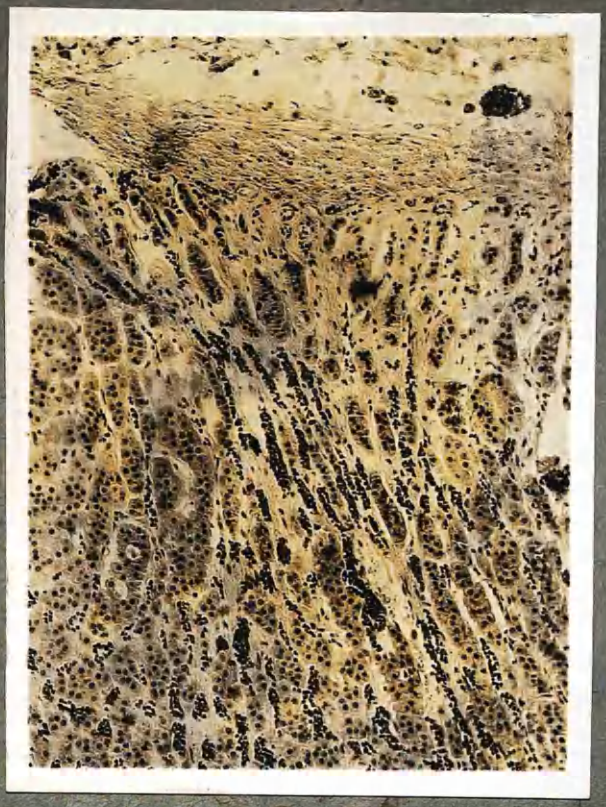


Fig. 4.

Photomicrograph of suprarenal gland from a case of Malignant Diphtheria (low power magnification). Compare with Fig.2, which is on the same scale.

Note that the cortical layers are distinguishable with difficulty. The zona reticularis is seen at the foot; its cells are in a state of cloudy swelling and the cell-columns are separated by clumps of red corpuscles. The zona fasciculata is seen in the lower half above the zona reticularis; the columns to the left and to the right have suffered cloudy swelling: in the centre the columns are isolated by radial haemorrhages. The zona glomerulosa is less damaged but small haemorrhages and cloudy swelling are visible. The capsular tissue is also showing slight evidence of cloudy swelling but no haemorrhages. The medulla is not visible.

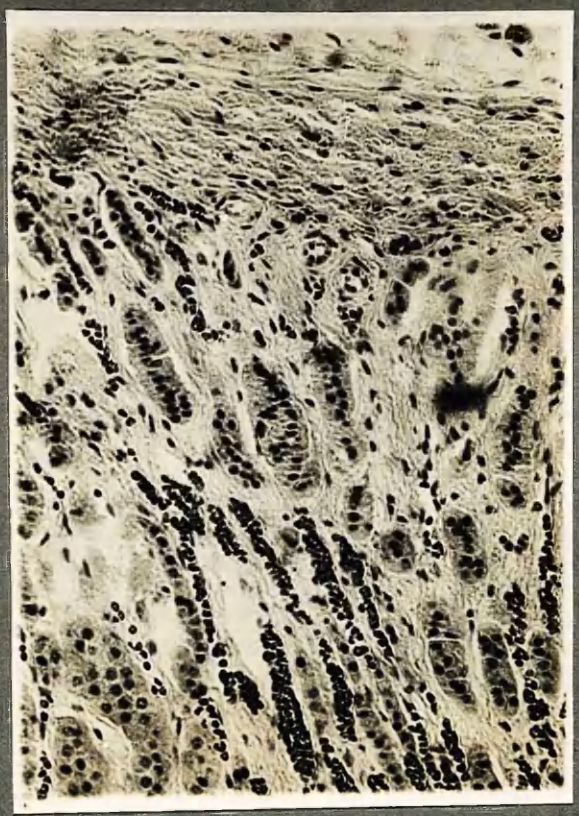


Fig. 5.

High power photomicrograph of part of the field shown in Fig.4. Compare with Fig.3 which is on the same scale.

The capsule, zona glomerulosa and the outer part of the zona fasciculata are visible. Note the cloudy swelling of the cortex but absence of haemorrhages. The zona glomerulosa shown cloudy swelling (note the difficulty in distinguishing cell-margins and the poorly staining nuclei). The zona fasciculata shows cloudy swelling and radial haemorrhages between the columns.

Fig. 6.

Photograph of mounted specimen of left kidney and suprarenal gland from a case of faucial diphtheria (natural size).

Note the absence of haemorrhages in the suprarenal substance. The specimen has been partly dissected to show the glandular enlargements along the suprarenal vein which is seen issuing from the hilum of the gland.

toxication is at a minimum and that the element of asphyxia predominates.

The other ten cases had extensive faucial lesions with severe general symptoms; post mortem definite changes in the suprarenal glands were found in all. A short summary of the main features of those ten cases with the appearances of the suprarenal glands at autopsy follows:

Case I.

J.M., a boy, 9 years of age, was admitted on the fourth day of severe diphtheria, pseudo-membrane being present on the tonsils, fauces, soft palate, uvula, posterior pharyngeal wall and in the nasal cavity; the glands of the neck were markedly enlarged, there was heavy foetor of the breath, the urine was loaded with albumen and contained acetone, the heart sounds were poor and lassitude obvious. During the first three days in hospital, the patient received 45,000 units intravenously and 135,000 units intramuscularly of diphtheria antitoxin; the general symptoms became less severe and the pseudo-membrane commenced to separate. On the morning of the fifth day of hospital treatment the patient suddenly complained of severe epigastric pain, collapsed and within five minutes was dead.

At autopsy, it was found that the left suprarenal gland was enlarged and that it had at its upper end an area of haemorrhage; microscopically the capsule did not appear to have suffered toxic change; the parenchymatous cells of the cortex were in a state of cloudy swelling; the columns of epithelium in the inner zone and the inner part of the middle zone were separated by masses of red blood corpuscles and the outer part of the middle zone and the outer zone showed the same change in a lesser degree; some haemorrhage was present in the medulla but only at that part which bordered on the inner zone of the cortex. (see Figs. 4 and 5).

The right suprarenal gland appeared to be somewhat congested. Microscopically, the cells of the cortex seemed to have suffered cloudy swelling of more intense nature than in the left suprarenal gland - thus the nuclei under a low magnification were hard to distinguish from the protoplasm and the cell outlines were lost; the nuclei under a higher magnification were seen to be in a state of karyorrhexis. These changes were present in all the layers of the cortex but were particularly obvious in the inner and middle zones. The medulla showed no change except the presence of a small zone of haemorrhage at

its border.

Case II.

W.A., a boy, 2½ years of age, was admitted to hospital in a moribund condition on the third day of illness; he had extensive diphtheritic membrane on the mucous membrane of the throat and great enlargement of the upper cervical glands. He died seventy minutes later.

Macroscopically the suprarenal glands at autopsy appeared normal. Histologically similar changes were found in both glands. There were haemorrhages between the fasciculi in the inner zones and the inner parts of the middle zones of the cortices and occasional haemorrhages in the outer parts of the middle zones and the outer zones of the cortices and in the extreme borders of the medullae. The cells of the cortical parenchyma gave evidence of cloudy swelling; those of the outer zones possessed nuclei which were deficient in chromatin and those of the two inner and middle zones had nuclei which varied in staining reaction, some being dark-staining and others light-staining with basic stains and others again having only a nuclear membrane to show the site of the nucleus; the cell outlines in the outer zone could be distinguished but not those in the

middle or inner zone. The cells of the medulla were little damaged.

Case III.

J.C., a boy, 4 years of age, was admitted to hospital on the third day of illness; he had an extensive diphtheritic condition of the throat and nasopharynx, haemorrhages from the throat and nose and a petechial rash; the glands of the neck were greatly enlarged and painful; there was heavy foetor of the breath; the heart sounds were poor and the pulse soft and rapid; prostration was extreme; the urine was scanty and contained albumen and acetone. The patient was given 72,000 units of diphtheria antitoxin intramuscularly; bleeding occurred at the site of injection later. The patient failed to respond and died twenty-seven hours after admission.

The left suprarenal gland was enlarged and found to be the seat of gross haemorrhages (see Fig.1).

The right suprarenal gland showed no macroscopic change; microscopically, however, changes similar to those described in the right suprarenal gland in Case I were discovered, i.e. there was cloudy swelling of the cortical parenchyma with haemorrhage particularly in the inner and middle zones; very

little change was detected in the medulla, haemorrhage being confined to the region in the vicinity of the cortex.

Case IV.

J. M'Q., a boy, 4 years of age, was admitted to hospital on the fourth day of illness. He had extensive diphtheritic patching of the throat, pseudo-membrane being present on the tonsils, fauces, palate, uvula and pharyngeal wall and a profuse sanious discharge issuing from the nostrils. General symptoms of profound intoxication were not present at first; the condition however, failed to respond to treatment, severe general symptoms developed and, on the fourth day in hospital, petechial haemorrhages were present in the skin of the legs and ecchymoses were noticed at the sites of needle-punctures. The patient collapsed and died on the seventh day in hospital; he had been given an aggregate of 200,000 units of diphtheria antitoxin during his stay in hospital.

At autopsy, the left suprarenal gland was enlarged and showed gross haemorrhages; the microscopic findings were as in the left suprarenal gland in Case I. The right suprarenal gland showed no macroscopic changes; histologically, cloudy swelling was noted in the cortex and haemorrhages

were present in the inner and middle zones of the cortex and to a lesser extent, in the outer zone of the cortex and in the outer region of the medulla.

Case V.

A.W., a boy, 5 years of age, was admitted to hospital on the third day of diphtheria and died eleven days later. On admission, general symptoms of toxaemia were severe; pseudo-membrane covered the tonsils, pillars of the fauces, palate, uvula and pharyngeal wall; there was a slight nasal discharge; the breath was foetid; the glands of the neck were greatly enlarged; the urine contained albumen and acetone. The patient responded to intramuscular injections of diphtheria antitoxin (144,000 units) and on the seventh day in hospital no pseudo-membrane was visible. On the ninth day in hospital, however, the patient became drowsy: next day his pulse became soft and rapid and the heart sounds were poor in tone. Early on the morning of the eleventh day in hospital he complained of a severe aching pain in the right lumbar region posteriorly and, a few hours later, he collapsed and died.

At the post mortem examination, small haemorrhages were visible in the right suprarenal gland but not in the

left suprarenal gland, which seemed normal in appearance. Histological sections of the right suprarenal gland showed that the areas of haemorrhage were focal in site and were placed in the cortex the shape of each being triangular with the apex being placed in the inner zone and the base in the outer zone; the remainder of the cortex and the medulla appeared to be normal. Similar sections of the left suprarenal gland showed freedom from haemorrhage and a minimum of degenerative change in the cortical cells, the cell-outlines being well maintained.

Case VI.

J.M'G., a boy, 8 years of age, was admitted to hospital on the fifth day of diphtheria. Two days previously an incision had been made through the left anterior pillar of the fauces because of a mistaken diagnosis of quinsy. On admission, general symptoms of diphtheria were severe and there was extensive pseudo-membrane on all the faucial structures and on the roof of the mouth as far forward as the posterior third of the hard palate; the incision in the fauces could not be detected; there was a profuse sanious nasal discharge, marked foetor of breath and great enlargement of the lymphatic glands of the neck. During the next

twelve hours the patient was given 100,000 units of diphtheria antitoxin intravenously and 112,000 units intramuscularly; he failed to rally and died fifteen hours after admission.

At autopsy, no gross changes were seen in the suprarenal glands. Microscopically small haemorrhages were present only in the zona reticularis of each gland and very little evidence of degenerative changes in the cortices was seen; the medullae seemed normal.

An interesting feature of this case, in view of the known close association between the suprarenal glands and the pituitary, was the finding of upward bulging of the roof of the sella turcica due to an excessive accumulation of colloid material in the pituitary gland; degenerative changes were not found in the gland and the significance of the finding was not apparent.

Case VII.

F.McD., a girl, 7 years of age, was admitted to hospital on the sixth day of diphtheria and died thirteen hours after admission. Pseudo-membrane was present on the left tonsil, left pillars of the fauces, the uvula, the greater part of the soft palate and the left pharyngeal wall and

there was a purulent nasal discharge; the lymphatic glands on the left side of the neck were greatly enlarged and tender; there was distinct foetor of the breath; general symptoms of diphtheria were marked. The patient was given 80,000 units of diphtheria antitoxin intramuscularly.

At autopsy, the suprarenal glands appeared normal. On section, however, focal haemorrhages were visible in various parts of the cortices, all three layers being involved; the cortical tissue between those foci showed very little change except in one or two places where early degenerative lesions were found and, in these areas, small accumulations of blood clot were present in the zona reticularis; in neither gland was the medulla involved.

Case VIII.

J.C., a boy, 7 years of age, and a brother of the patient whose case has been reported as Case III, was admitted to hospital on the fifth day of diphtheria. On admission, general symptoms were only of moderate severity; pseudo-membrane was present on both tonsils and on the contiguous surfaces of the pillars of the fauces; there was a profuse nasal discharge and the lymphatic glands of the neck were moderately enlarged and tender. 32,000 units

of diphtheria antitoxin were given by the intramuscular method on admission and repeated twelve hours later. The boy's general condition improved and the pseudo-membrane separated gradually till none was visible on the seventh day of treatment. After that day, however, the patient failed to improve and signs of cardiac failure were evident. On the ninth day in hospital petechiae were found on the skin of the arms and the patient became very drowsy; he died later that day.

Post mortem examination revealed no definite change in the right suprarenal gland and a haemorrhagic condition in the left suprarenal gland on macroscopic examination. Under the microscope, the cortices of both glands showed signs of cloudy swelling; haemorrhage was present between the columns of the inner zone of the cortex in the right suprarenal gland and between the columns of all three zones of the cortex in the left suprarenal gland, being most severe in the inner zone and least extensive in the outer zone; the medulla in both glands was damaged only at its boundary with the zona reticularis of the medulla and in this region haemorrhages were present.

Case IX.

T.S., a boy, 7 years of age, was admitted to hospital with diphtheria on the sixth day of illness, pseudo-membrane being present on the tonsils, palate, uvula, pillars of the fauces and pharyngeal wall; the lymphatic glands of the neck were greatly enlarged and tender, there was a sanious discharge from the nose, the breath was foetid and general symptoms of diphtheria were severe. The patient was given 60,000 units of diphtheria antitoxin intravenously and 64,000 units intramuscularly. His condition improved until the seventh day in hospital when palatal paralysis was found to be present and signs of cardiac failure commenced. On the following day he collapsed and died.

Post mortem, the most significant findings were enlargements of lymphatic glands including the lymphatic nodes related to the suprarenal glands (see Fig.6). Neither suprarenal gland revealed gross damage; the only microscopic changes in the right suprarenal gland were small haemorrhages in the zona reticularis; the left suprarenal gland was not examined microscopically, being retained for mounting as a specimen.

Case X.

I.M., a girl, 7 years of age, was admitted to hospital on the second day of diphtheria. Pseudo-membrane was present on the tonsils, pillars of the fauces, palate and pharynx; the glands of the neck were enlarged and tender and general symptoms were profound. The patient received in all 148,000 units of diphtheria antitoxin and on the eighth day in hospital all pseudo-membrane had disappeared. Two days later the patient became very drowsy and signs of cardiac failure appeared. On the eleventh day in hospital, there was heavy albuminuria and acetonuria; later that day the patient became comatose, her temperature rose to 105°F and she died.

The suprarenal glands showed no gross changes. The zonae reticulares contained small haemorrhages. Haemorrhage in the brain was sought but not found.

Correlation of the clinical state and the appearances of the suprarenal glands at autopsy in each of the above cases leads to the following conclusions:

It appears that the suprarenal glands, like other organs of the body, suffer from the presence in the body of diphtheria toxin. In cases in which intoxication is at a

minimum, e.g. cases of membranous croup, there is either no visible change or alteration is represented at most by a small amount of haemorrhage between the fasciculi of the zona reticularis of the cortex. In more severe cases, the suprarenal lymphatic glands along the suprarenal vein are inflamed, haemorrhages are found in all three layers of the cortex and in that region of the medulla which is in proximity to the cortex, and cloudy swelling of varying degree affects the parenchymatous tissue of the cortex; the haemorrhages and the cloudy swelling are most intense in the zona reticularis and the inner part of the zona fasciculata. In the most severe cases, haemorrhage in one or other suprarenal gland is of such a degree as to be apparent on the surface of the organ.

In one case, the patient before death complained of pain in one side and later, the suprarenal gland on this side was found to be the seat of macroscopic haemorrhages (Case V). In other cases, it is not improbable that diffuse haemorrhagic infiltration of the suprarenal cortex occurred as the patient's general condition deteriorated and that actual rupture of the haemorrhage externally coincided with the final collapse of the patient (Cases I,

III, IV, VIII).

The nervous supply of the suprarenal glands is derived from the splanchnic nerves from the sympathetic, the phrenic nerves and the vagus nerves (particularly the right vagus) through the suprarenal plexus and that of the heart from the vagus nerves and the sympathetic through the cardiac plexus (Gray's Anatomy)[25]. It is thus evident that the nervous connections of the suprarenal glands and of the heart are closely related and it seems possible that, through those connections, a sudden change such as a haemorrhage, in a suprarenal gland may cause the patient to manifest signs of acute cardiac failure ending in death.

(C) Water Excretion in Diphtheria.

A special study of the excretion of water in the urine in cases of diphtheria was made. The method employed was the simple Water Test introduced by Volhard and Koranyi[41] for the estimation of renal efficiency.

Briefly, the test is carried out as follows:

On the previous day, intake of fluid is restricted and after 9 pm. is stopped. At 9 pm. the bladder is emptied and the urine discarded.

From 9 pm. till 9 am. on the day of the test, all urine passed is kept, the final quantity be-

ing added at 9 am.; the volume and specific gravity of this, the night urine, are measured.

At 9 am. the patient drinks thirty ounces of water. Urine is passed at 10 am., 11 am., 12 am., 1 pm., 2 pm. and 3 pm. The volume and specific gravity of each specimen are measured.

Normally, under the conditions of the test, the night urine does not exceed 20 oz. and its minimum specific gravity is 1025, the average figures being 15 oz. and 1032 respectively. During the day, the entire thirty ounces of water, drunk at 9 am. are excreted in five hours and the maximum specific gravity is 1003; fifteen ounces are usually excreted within the first two hours.

Poor concentration of the night urine, which is shown by excretion of more than 20 oz. of urine of which the specific gravity is less than 1025, is regarded as an early sign of renal disease.

Renal impairment is shown by failure to excrete the test dose of water in five hours, by a persistent specific gravity of 1010 or more in successive hourly specimens and by equality in the quantity of the hourly specimens; the extent of renal impairment is proportional to the deficiency of output.

In thirty-two patients who were under my care in the

Diphtheria wards, the Water Test was carried out as described above at weekly intervals during the illness until the test gave a normal result. The specimens of urine were obtained naturally, catheterization not being employed.

The deficiency in excretion at two hours after the ingestion of the test dose of water was calculated as a percentage of 15 oz., and the deficiency in excretion at five hours after the ingestion of the test dose of water was calculated as a percentage of 30 oz. The lesser of these two percentages was taken as a measure of the deficiency in water excretion because that figure was less likely to be affected by mechanical difficulties in the expulsion of urine such as incomplete emptying of the bladder by an ill patient.

The detailed results are presented in the Appendix under the headings Cases XI to XLII. It was found that three cases failed to reveal any deficiency of water excretion and one of these, being merely a diphtheria carrier, was apparently in good health; in a fourth case, as also in the carrier case, the initial test was void because of the presence of menstruation. In the remaining twenty-eight cases, deficiency of water excretion was present in all in the acute stage of the illness.

It may be argued that this deficiency of water excretion is due to damage to the kidney tissue, such as might be presumed to follow the presence of diphtheria toxin in the circulating blood. It is noteworthy, however, that if the two cases (Cases XX and XXIV) in which menstruation occurred are excluded, in only two of the remaining cases (Cases XXXI and XXXVII) was there albuminuria and in both of these the albuminuria was transient. Again, in three cases only (Cases XII, XXIII and XXV) did the night urine exceed 20 oz.; in one of these (Case XV), there was no deficiency in water excretion during the daytime and therefore, presumably, no kidney damage; in the other two (Cases XII and XXIII) the specific gravity of the night urine in each case was less than 1025. Thus in the whole series, the number of cases in which a presumption of the existence of kidney damage might be made is four (Cases XII, XXIII, XXXI and XXXVII).

Discounting these cases in which some kidney damage might exist and the cases in which menstruation interfered with the results, it is found that there are twenty-six cases in which the presence of kidney damage was not proved; of these, only two cases did not show deficiency of water in the acute stage (Cases XXV and XXVI).

The conclusion is reached that in this series of mild and moderately severe cases of diphtheria, there was a group of twenty-six cases in which kidney damage was not demonstrated and, of this group, twenty-four cases, or 92%, showed diminution of water excretion during the acute stage of the illness.

(D) The Blood Sugar in Diphtheria.

The normal blood sugar in the fasting state, as found in blood taken from the finger-tip after needle-puncture, is about 90 milligrammes per 100 cubic centimetres of blood if the method described in the Appendix is used.

After ingestion of glucose, the usual test dose of which is 40 grammes, the blood sugar varies in a definite manner which is explained by Peters and Van Slyke, (27) among others, as follows:

> (i) there is an immediate rise of blood sugar due to absorption of glucose from the alimentary system to the blood;
>
> (ii) about fifteen minutes later, the tissues, particularly the liver and the skeletal muscles, begin to convert glucose received from the blood, into glycogen which is stored; this process tends to reduce the amount of glucose in the circulating blood and is attributed to the action of the internal secretion of the pancreas;
>
> (iii) the second process goes on at a more rapid rate than the first with the result that

> about forty minutes after the ingestion of glucose, the blood sugar begins to fall; in the normal person the blood sugar is back to the fasting level about sixty minutes after the ingestion of the glucose; not infrequently for the next few hours, the blood sugar is below the fasting level.

Sugar in appreciable quantity is excreted in the urine only when the blood sugar reaches or exceeds a value which, in the majority of cases, is about 180 milligrammes per 100 cubic centimetres.

The ingestion of galactose or laevulose is followed by a similar succession of events, with the exception that conversion of the sugar into glycogen and consequent storage as glycogen takes place so rapidly that, normally, no rise of blood sugar occurs.

In cases in which hepatic inefficiency is present, it is stated by Monro (28) that the blood sugar curve following laevulose ingestion may approximate in shape to the blood sugar curve following glucose ingestion. Again, when the laevulose test is employed, Hurst (29) regards a rise of blood sugar of more than 20 milligrammes per 100 cubic centimetres of blood in one hour as being significant of hepatitis. Dodds (30) considers that the galactose tolerance test is superior to the laevulose tolerance test in the same connection; his conclusions are similar to those of Hurst.

It is now well recognized that disturbances affecting the blood sugar are present in Diphtheria. Schwentker and Noel (31) found that in the early part of the acute stage of Diphtheria hypoglycaemia and reduction of glycogen in the liver and muscles occurred as a result of increased breakdown of stored glycogen and increased utilization of circulating carbohydrate; at a later period in the acute stage, they found that increasing difficulty in the assimilation of glucose from the blood stream occurred with resulting hyperglycaemia. Various French observers (32, 33, 34) found that hypoglycaemia, increased blood urea, initial decrease and later increase of blood cholesterol, and diminution of blood chloride occurred in Diphtheria. Benn, Hughes and Alstead (35) observed that in the acute stage of Diphtheria the blood-sugar curve after intravenous injection of glucose approximated to that seen in cases of diabetes. Brems (36) noted that the blood sugar curve following ingestion of glucose was of the diabetic type in the acute stage of diphtheria and for a period of two to three weeks afterwards. The most recent important communication on the subject is made by Begg; (37) he notes afresh the abnormalities in the glucose tolerance curves and quotes experimental evidence which suggests a close relationship between the type of blood sugar curve and the degree of cardiovascular failure in

the early stages of diphtheria.

Three groups of diphtheria cases were chosen and blood sugar estimations were made after the ingestion of glucose, laevulose and galactose, respectively. The test was carried out in each case on the day after admission and, again, on the day before the patient was allowed to leave bed. The quantity of sugar given was restricted to 20 grammes, as it was found that the usual 40 grammes dose almost invariably caused nausea and often vomiting in the acute stage of the disease. The estimations were carried out on blood from the pricked finger by Herbert and Bourne's (38) modification of the Folin-Wu method of blood sugar estimation (see page 102).

The detailed results are presented in the Appendix under the headings Cases XLIII to LXXIV. It will be found that in thirty-one cases (excluding Case LVII which was that of a healthy carrier), the average fasting blood sugar on admission was 80.4 milligrammes per 100 cubic centimetres of blood and at the time that the patient was allowed to leave bed (i.e. about four weeks later), was 84.1 milligrammes per 100 cubic centimetres blood.

In sixteen cases, the test sugar was glucose. In one of those cases (Case LVII) the patient was a healthy carrier and the range of his blood sugar on admission was within

normal limits. In the remainder, the fasting blood sugar on admission was generally at a hypoglycaemic level and the subsequent curve took the form that has frequently been called "the lag curve"; several weeks later the fasting blood sugar was higher and the blood sugar curve was approaching the recognized normal form.

In the other sixteen cases, the test sugar was either galactose or laevulose. Of the five cases in which galactose was employed, there was an initial hypoglycaemia in four cases; in two cases, the blood sugar curve on admission fell within normal limits, in one case it resembled in shape a normal blood sugar curve after the ingestion of glucose and in two cases it resembled a lag curve such as was found in the glucose tolerance test above. Of the eleven cases in which laevulose was used, there was a fasting hypoglycaemia in eight cases, a normal fasting blood sugar in one case, and a fasting blood sugar which was at the higher limit of normal in two cases at the time of admission; the blood sugar curves were normal in five cases, resembled a normal glucose tolerance curve in one case, and resembled a lag curve in five cases. With the exception of two laevulose tolerance curves which were still tending to be at a high level after four weeks' treatment, repetition of the galactose and laevulose tolerance curves revealed

normal results at the time when the patients were allowed out of bed.

From consideration of these findings, it appears that in cases of Diphtheria which come under treatment early there is a state of hypoglycaemia during the fasting stage; where the patient does not receive treatment until later the fasting blood sugar tends to rise to normal again and it is possible that, as Schwentker and Noel have stated, there are cases in which hypoglycaemia is present in the fasting stage; the fasting blood sugar in any case becomes normal as convalescence advances.

Again, as Benn, Hughes and Alstead, (35) Brems (36) and Begg (37) have previously shown, glucose tolerance curves in the acute stage in all the cases of Diphtheria investigated tended to approximate to the "lag" form which suggested that there was little or no loss of the power of absorption of glucose into the blood but that there was a diminution of the power of assimilation of glucose by the tissues from the blood.

Galactose and laevulose tolerance curves were abnormal in the acute stage in eight out of sixteen cases in which these curves were plotted, i.e. in 50% of cases. This suggests that the power of assimilation of sugar by the liver is at fault only in half the cases.

Finally, consideration of these two last conclusions

leads to the belief that the rise of blood sugar in a glucose tolerance test in the acute stage of Diphtheria is mainly due to diminished assimilation of glucose by the peripheral tissues and not by the liver.

(E) The Serum-Sodium, Serum-Potassium and Serum-Chlorine in Diphtheria.

The basic elements which are present in normal blood serum are sodium, potassium, calcium and magnesium. They are balanced by the acid radicles, which are chlorine bicarbonate, phosphate, protein, sulphate and organic acid. Peters and Van Slyke (27) give the relative quantities of each of these in the following formula:

Na	K	Ca	Mg	=	Cl	HCO_3	PO_4	protein	(SO_4 & organic acid)
142	5	5	3	=	103	28	2	16	6

where Na = sodium,
K = potassium,
Ca = calcium,
Mg = magnesium,
Cl = chlorine,
HCO_3 = the bicarbonate radicle,
PO_4 = the phosphate radicle,
SO_4 = the sulphate radicle,

and the figures given below each chemical symbol represent the quantity of the chemical substance represented by the symbol present as milli-equivalents of that chemical substance in a litre of normal blood serum. It is to be noted

that one milli-equivalent is equal to a thousandth part of a gramme-equivalent.

It has been found that in conditions in which supra-renal cortical deficiency occurs there are deviations of these values from the normal and that the changes occur particularly in the serum sodium and serum chlorine, both of which are decreased, and in the serum potassium which is increased. (12, 13).

A number of cases of Diphtheria were investigated with regard to the sodium, chlorine and potassium contents of their blood serum during the period from March 1934 till April, 1935. In general, the cases were chosen according to the ease with which it was thought specimens of blood could be obtained for examination; it was not expedient to include any patients under the age of seven years and many patients under the age of ten years.

Specimens of blood were taken from the median basilic vein in sufficient quantities to yield at least three cubic centimetres of serum on four occasions during the hospital treatment of the patient, viz. on admission, one week after admission, two weeks after admission and on the day before dismissal.

The methods used for the estimation of serum sodium, serum chlorine and serum potassium are those described by

Peters and Van Slyke (39) and are described in the Appendix.

The results are given in Tables 3 to 7 and are grouped according to the severity of the cases and according to whether the cases were receiving the ordinary treatment of diphtheria patients or were also receiving an extra quantity of sodium chloride in the manner which is described in Section III.

It was found that 84 cases of Diphtheria, which recovered, were examined for the sodium, chlorine and potassium contents of their blood serum; 55 of these received extra sodium chloride and 29 did not receive extra sodium chloride.

The average serum sodium on admission was 120.82 milli-equivalents per litre or 17.18 milli-equivalents per litre below the normal level taken by Loeb. The average serum chlorine was 101.83 milli-equivalents per litre or 3.17 milli-equivalents per litre below the normal level. The average serum potassium was 6.83 milli-equivalents per litre or 1.83 milli-equivalents above the normal level.

The respective average values on dismissal were 130.75, 104.43 and 5.85 milli-equivalents per litre, i.e. the serum sodium was 7.25 milli-equivalents per litre below normal, the serum chlorine was 0.57 milli-equivalent per litre below normal and the serum potassium was 0.85 milli-equiva-

lent per litre above normal. The three values had approached to normal by 9.93, 2.60 and 0.98 milli-equivalents per litre respectively, during the period of residence.

There is, therefore, in Diphtheria, a condition of the blood serum resembling that which is found in cases of suprarenal cortical deficiency such as occurs in the acute phase of Addison's Disease. The change appears to affect particularly the sodium content the fall in which, however, is also accompanied by a fall in the chlorine content and by a rise in the potassium content; these latter changes are not of sufficient magnitude to balance the fall in sodium content.

The administration of extra sodium chloride does not appear greatly to influence the course of the return of the serum constituents to normal.

The severity of the acute stage is not particularly associated with the degree of abnormality of the serum constituents although it can be stated that, roughly, the more severe the acute stage the more removed from normal are the values of the serum sodium, serum chlorine and serum potassium. There is, however, a very definite fall in serum sodium and serum chlorine in cases which will shortly prove fatal (see Table 7).

It may, therefore, be concluded that there are, in Diphtheria, certain pieces of evidence which suggest that dysfunction of the cortical part of the suprarenals is present. The clinical appearances in Diphtheria, particularly those that depend on prostration and asthenia, are common in diseased conditions of the suprarenal glands. Definite lesions in the suprarenal glands are proved to be present in Diphtheria and these lesions are found most characteristically in the cortical tissue. There are deficiency in water-excretion, upsets of carbohydrate metabolism and changes in the bases of the blood-serum such as have been proved to be cardinal features of conditions of suprarenal cortical deficiency, and these abnormalities tend to approach normality as convalescence proceeds. Finally, it is shown in the next section that a method of treatment intended to benefit the element of suprarenal cortical deficiency is a potent means of producing improved results in a series of cases of Diphtheria.

Section III.

SODIUM CHLORIDE IN THE TREATMENT OF DIPHTHERIA.

As the experimental work described on the previous pages developed and the form that the conclusions would take became apparent, it was decided to apply the knowledge obtained in the treatment of cases of Diphtheria. It was thought that, if deficiency of the secretion of the suprarenal cortex occurred in Diphtheria, measures which had been found to have a beneficial effect on other clinical states characterized by such deficiency would also improve the condition of patients suffering from Diphtheria. Two such methods of treatment were considered, viz.,

(i) the administration of suprarenal cortical extract, and

(ii) the administration of sodium chloride.

In view of the prohibitive cost of suprarenal cortical extract, it was impossible to contemplate an experiment on a large number of patients with quantities of extract sufficiently adequate to be of therapeutic value. It is interesting, however, to know that Donato (32) has noted that the administration of total suprarenal extract has a favourable effect on the course of Diphtheria and on the disordered

carbohydrate and cholesterol metabolism which occurs in the disease. More recently, too, Thaddea[23] has observed that the changes produced in guineapigs during experimental intoxication with diphtheria toxin can be mitigated by treatment with suprarenal cortical extract if combined with large doses of ascorbic acid (vitamin C).

In the last few years clinicians have regarded the abnormal carbohydrate metabolism, which occurs in Diphtheria, as a cardinal feature of the disease and have attempted to improve the treatment by methods which aim at the rectification of this state. Thus, for example, Schwentker and Noel[31] gave intravenous injections of 20 grammes of glucose in a 50% solution in saline at intervals of twelve to twenty-four hours in the early part of the acute stage and as a 10% solution later in the acute stage; at the same time, hypodermic injections of small doses of insulin were given on the theory that the patient's tissues in Diphtheria cannot assimilate glucose properly. Later Schwentker and Noel's work was repeated by Benn, Hughes and Alstead, and Begg produced results which suggest that insulin is of little value while glucose is of undoubted benefit in treatment. The latter observers, also, gave the therapeutic doses of glucose in 50 cubic centimetres of normal saline. It is note-

worthy that those workers who have produced good results by the intensive administration of glucose to their patients have been giving not only glucose but also sodium chloride. Further, it is conceivable that the excellence of their results has been due in part, if not wholly, to the sodium chloride which the patients have been given.

During the period from 1st May 1934 till 30th April 1935, two series of cases in the Diphtheria wards of Ruchill Hospital were taken; one was given an extra quantity of sodium chloride while the other did not receive this. The cases which composed these two series were not selected in any way apart from the necessity for filling a vacancy in a ward by the admission of a fresh case but, as a consequence of natural reluctance to use an untried method of treatment, only one quarter of the available accommodation was reserved for the cases which received additional sodium chloride; it may be said, therefore, that the cases were chosen at random.

In the first series each case, which was permitted to take nourishment by mouth, was given a quantity of sodium chloride in addition to that present in the ordinary diet; the amount of sodium chloride administered varied according to the age of the patient from one heaped teaspoonful to three heaped teaspoonfuls per diem and the treatment was employed

for the period of three weeks succeeding the patient's admission.

In certain more severe cases in which it was possible to use intravenous injection, sodium chloride was given daily for fifteen days in the form of 20 to 30 cubic centimetres of 5% or 10% hypertonic solution, the injected saline solution being at body temperature and the injection being made through a hypodermic needle from a 30 cc. Record syringe. Hypertonic saline was used in preference to isotonic saline because the tests for the excretion of the water (see Section II) gave results from which it appeared that there is in the acute stage of Diphtheria a diminution in the volume of water excreted by the kidney and because the use of hypertonic saline meant that a larger quantity of sodium chloride could be given in a given volume than by the use of isotonic saline.

In critical cases, the method employed, but used in conjunction with intravenous hypertonic saline injection where that was possible, was that of continuous rectal glucose saline irrigation which will now be described.

Continuous Rectal glucose Saline Irrigation.

This mode of treatment was introduced originally by John B. Murphy, (40) the noted American surgeon in 1882 and has

been used mainly for the treatment of shock. In this method a saline solution is introduced into the lower colon drop by drop at intervals of about five seconds. The mucous membrane is able to assimilate each drop as it reaches the colon and thus to absorb in a few hours several pints of fluid, whereas if the same volume of fluid had been delivered in larger quantities, bowel movement would almost certainly occur and the saline solution be evacuated.

There are several advantages to be gained by using continuous rectal irrigation in Diphtheria. Firstly it is important that a large quantity of fluid is not allowed to lie within the intestine and retard the action of the heart, an organ which is severely taxed as it is. Secondly, it is superior to four-hourly rectal feeding because, once the method has been established, there is no necessity for rousing the patient at intervals and, more important still, it is less likely to cause spontaneous evacuation of the intestinal contents, an event which is especially liable to occur in cases of severe Diphtheria. Thirdly, it can be used at all ages, unlike methods of treatment involving intravenous injections which can rarely be employed on repeated occasions in children under seven years of age.

The apparatus used in the treatment of the cases in

Fig. 7.

Diagram of Apparatus used in Continuous Examination of Glucose-Saline per rectum.

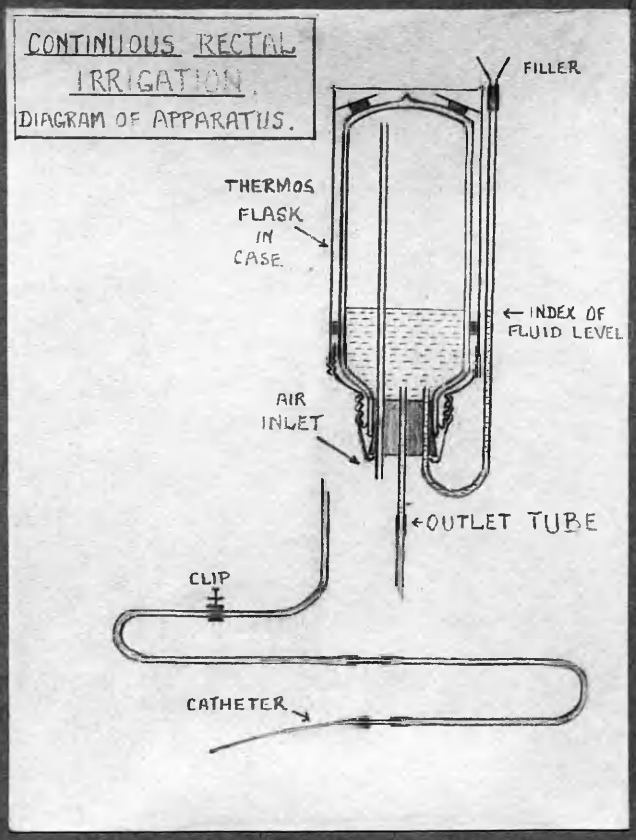


Fig. 7.

Diagram of Apparatus used in Continuous Administration of Glucose Saline per rectum.

this series is depicted diagrammatically in Fig.7. The main part of this apparatus consists of a "Thermos" flask, of one-pint capacity, with the cork replaced by a rubber stopper through which three holes have been bored. A glass tube passes through one hole and reaches almost to the bottom of the flask inside while it ends freely outside, one inch of its length being visible; the purposes of this tube are to adt as an overflow if the flask is overfilled and to allow air to enter while the flask is in use. Through a second hole passes another glass tube which reaches for about half-an-inch inside the flask and which curves on itself outside to run close to the outside cover of the flask, the free outside end being joined to a small glass funnel by means of a soft rubber connection; the purposes of this tube and its funnel are to act as a channel for refilling the flask and, during use, by means of the level of fluid in it, to be an index of the quantity of fluid in the flask. Through the third hole passes a short glass tube which extends for about half-an-inch inside the flask and for about two inches outside the flask; this tube is continuous with a length of rubber tubing, three to four feet long, which is connected by a short piece of glass tubing to a no.1 soft rubber catheter. The "bakelite" or metal cap of the "Thermos"

flask is screwed on after its central portion has been cut out and it serves to hold the rubber stopper in position.

In use the flask is placed in an inverted position about two to three feet above the level of the bed. The soft rubber catheter is lubricated with soft paraffin and is inserted through the anal orifice as far as possible, the object being to leave the nozzle in the lower end of the colon. The rubber tubing is provided with an adjustable clip which regulates the rate of flow through the tubing; a degree of compression of the clip which allows one drop of saline to pass through in five seconds usually gives the best results at the commencement; later a flow of one drop in four seconds or even of one drop in three seconds may be tolerated quite well.

The flask is filled with normal saline containing not more than 10% of glucose; a greater concentration of glucose tends to irritate the intestinal mucous membrane with results fatal to the procedure. The primary object of the glucose is that it should act as a source of food for the patient; secondarily, it is intended to counteract the hypoglycaemia of the early stages of the disease.

The temperature of the saline in the flask should be higher than the body temperature of the patient but it should

not exceed 110°F. Fluid placed in the flask at such a temperature usually cools in passing through the rubber tubing and enters the patient's body at a temperature below normal body temperature; this, however, is preferable to the risk of a dangerous injury to the rectal mucous membrane if saline, at a higher temperature than 110°F, is allowed to enter the rectum as a result of a breakdown in the regulation of the flow; besides, it is found in practice that the patient tolerates the saline quite well at a subnormal temperature. The size of the flask is such that its contents must be replenished at frequent intervals by the nurse in attendance, thus ensuring that the temperature of the fluid is kept as high as possible within the limits defined.

Certain precautions must be taken before the institution of continuous rectal irrigation. Thus, it is very important that the patient should be given a large enema before the commencement of the treatment. Again, the patient should receive nothing by mouth from the moment it is decided to treat him in this way although, if the patient becomes clamorous, he may be allowed to suck small pieces of ice or be given orange-juice in teaspoonful doses. Further, the foot of the bed should be raised because the position thus attained does not encourage a spontaneous

evacuation of the bowel contents. Finally, the administration of sedatives is important and in the present series of cases the drug that was most often used was Syrup of Chloral; the patient was usually given half-a-drachm by mouth at the institution of treatment and two to four drachms were added to the first pint of saline given rectally and a smaller dose, according to the degree of restlessness, was added to each later pint of saline.

In the first twenty-four hours of treatment, there may be difficulty in making the patient understand that he must not evacuate the bowels. If several bowel-movements should occur it is wise to give a second enema after which there is seldom any further trouble. When the treatment is thoroughly instituted it has been found possible to maintain the patient on it for more than two weeks and it has proved adequate for the patient's immediate food requirements for that time.

When the patient has passed the critical acute stage of his illness and the time comes to stop continuous rectal irrigation, he is given gradually increasing quantities of arrowroot by mouth for several days. The saline is finally stopped when natural bowel movements recommence.

By the use of this method, a varying quantity of fluid can be introduced in different persons. A minimum quantity of five to six pints in the twenty-four hours

should be aimed at, which is equivalent to a consumption of about 300 grammes of glucose and 27 grammes of sodium chloride in the time mentioned.

Patients subjected to this treatment not infrequently develop subcutaneous oedema, usually over the back and sacral region; several patients have developed general anasarca and in one there was ascites of such amount that embarrassment was caused to the patient, to relieve which fluid required to be withdrawn from the peritoneal cavity. It is a noteworthy clinical observation, however, that the occurrence of oedema seemed to coincide with amelioration of the general symptoms of diphtheria intoxication. It is a matter for conjecture whether, if hypertonic saline had been used for the irrigation, this oedema could be avoided but one's training supports the belief that the intestinal mucous membrane would be intolerant of such a solution.

The results of treatment have been analysed statistically, the method used postulating that the difference between the percentage values of a given characteristic in two parallel series must be at least twice the standard error of that difference before it can be asserted that the difference is significant. The standard error of the difference between the percentages in each series was calculated

from the quantity

$$\sqrt{\frac{P_1 Q_1}{N_1} + \frac{P_2 Q_2}{N_2}}$$

where P_1 and P_2 are the percentages in the two series of a given characteristic; Q_1 and Q_2 are the differences between 100 and P_1, and 100 and P_2, respectively, and N_1 and N_2 are the numbers of observations in the two series.

The conclusions will now be given in detail, reference being made at various points to the appropriate tables of results which are to be found in the Appendix.

During the period from 1st May 1934 to 30th April 1935 775 cases were admitted to Ruchill Hospital and came under the purview of the present investigation. Of these, 575, or 74.19%, were treated in the usual manner and were not given sodium chloride beyond what their food contained; the remaining 200, or 25.81%, were treated in the usual manner and were also given an extra quantity of sodium chloride by one of the methods already described. Again, 740 cases, or 95.48%, recovered and were dismissed well; 35 cases, or 4.52%, died.

Of the 740 recovered cases 555 or 75% did not receive extra sodium chloride and 185, or 25%, received extra sodium chloride. Of the 35 fatal cases, 20, or 57.14%, did not

receive extra sodium chloride and 15, or 42.86%, received extra sodium chloride.

It is noteworthy also that the fatal results all took place in Group IV, the group containing cases which had a very severe acute stage (see Table I in the Introduction).

RECOVERED CASES.

(a) SATISFACTORY RECOVERY (see Table 8)

If cases of SATISFACTORY RECOVERY (as defined in the Introduction) are compared in the series of patients who received extra sodium chloride and in the series of patients who did not receive extra sodium chloride, it is found that if all recovered cases are considered the percentage of satisfactory recovery was higher in the former series but the difference in the percentages is not significant. The percentage of recovered patients in Group I who made a satisfactory recovery was higher in the series of cases which did not receive extra sodium chloride but the difference between the percentages was not significant. The percentage of recovered patients in Group II who made a satisfactory recovery was higher in the series of cases which received extra sodium chloride but the difference between the percentages was not significant. In Group III

the percentage of recovered patients who made a satisfactory recovery was higher in the series of cases which received extra sodium chloride and the difference between the percentages was definitely significant. In Group IV the percentage of recovered patients who made a satisfactory recovery was higher in the series of cases which did not receive extra sodium chloride but the difference between the percentages was not significant.

Therefore, it may be concluded that so far as making a satisfactory recovery was concerned, mild cases did not benefit, moderate cases benefitted in a small degree and severe cases in a marked degree from inclusion in a series of cases which received extra sodium chloride; very severe cases did not benefit from such inclusion but the reason for this result becomes apparent when later results are analysed.

(b) UNCOMPLICATED RECOVERY (see Table 9)

If cases of UNCOMPLICATED RECOVERY (see Introduction) are next taken, it is found that when all recovered cases are considered the percentage of uncomplicated recovery was very slightly higher in the series of patients who did not receive extra sodium chloride than in the series of patients who received extra sodium chloride; the difference between

the percentages was not significant. The percentage of recovered patients in Group I, whose recovery was uncomplicated, was higher in the series which did not receive extra sodium chloride but the difference between the percentages was not significant. The percentage of recovered patients in Group II whose recovery was uncomplicated was higher in the series which received extra sodium chloride but the difference between the percentages was not significant. In Group III, the percentage of recovered patients who made an uncomplicated recovery was higher in the series which received extra sodium chloride and the difference between the percentages was significant. In Group IV, the percentage of recovered patients who made an uncomplicated recovery was higher in the series which did not receive extra sodium chloride but the difference between the percentages was not significant.

The conclusion here is that in making an uncomplicated recovery mild cases did not benefit, moderate cases benefitted to a small extent and severe cases to a great extent from inclusion in a series of cases which received extra sodium chloride. Again, very severe cases did not appear to benefit for a reason which will be apparent later.

(c) DELAYED RECOVERY (see Table 10).

It is obvious from the definitions of terms given in the Introduction that cases which come under the heading of DELAYED RECOVERY represent the difference of cases which come under the headings of SATISFACTORY RECOVERY and UNCOMPLICATED RECOVERY. Hence, it follows that an increase in the number of such cases in either series may result from diminution of the number of cases which make a satisfactory recovery or from diminution of the number of cases which recover after developing complications; similarly, a decrease in the number would accompany increase in the latter two categories. Again, if an influence is at work in a given series of cases which tends to improve equally the condition of the series as a whole, it follows that a decrease in the number of cases which make a complicated recovery means an addition to the number of cases which make a delayed recovery and a decrease in the number of cases which make a delayed recovery means an addition to the number of cases which make a satisfactory recovery; the net result is that no alteration will occur in the number of cases making a delayed recovery. Further, if the hypothetical beneficial influence exerts itself particularly on the cases which make a complicated recovery, the result is

that a greater number of cases are transferred from the cases which make a complicated recovery to the cases which make a delayed recovery than are transferred from the cases which make a delayed recovery to the cases which make a satisfactory recovery; the net result is an increase in the number of cases which make a delayed recovery. On the other hand, if the beneficial influence exerts itself particularly on the cases which make a satisfactory recovery, the tendency will be for a smaller number of cases to be transferred from the cases which make a complicated recovery to the cases which make a delayed recovery than will be transferred from the cases which make a delayed recovery to the cases which make a satisfactory recovery and the net result will be a decrease in the number of cases which make a delayed recovery.

When all recovered cases which make a delayed recovery are considered it is found that a lesser percentage of such cases occurred in the series which received extra sodium chloride; this suggests that more cases were removed from the cases of delayed recovery to the cases of satisfactory recovery than were received from the cases of complicated recovery to the cases of delayed recovery. The same conclusion is reached in the groups of moderate, severe and

very severe cases but is not made in the group of mild cases. On no occasion, is the conclusion of statistical significance.

It may be said however, that the benefit produced in a series of cases by giving them extra sodium chloride is more in those cases which would not naturally make a complicated recovery and most in those cases the recovery of which would not naturally be delayed.

(d) COMPLICATED RECOVERY - TYPE OF PARALYSIS (See Table 11)

If cases of complicated recovery are divided into cases of recovery with mild paralysis and cases of recovery with severe paralysis (as defined in the Introduction) it is found that the percentage of cases with mild paralysis was higher in the series of cases which did not receive extra sodium chloride but the difference between the percentages in the two series was not significant. In Group I, the results in the two series were identical; in Group II, there was a greater percentage of recovery with mild paralysis in the cases which did not receive extra sodium chloride, but the difference between the percentages in the two series was not significant; in Group III there was a greater percentage of recoveries with mild paralysis in the cases which received extra sodium chloride and the observed difference between the percentages in

the two series is statistically significant; and in Group IV there was a greater percentage of recoveries with mild paralysis in cases which did not receive sodium chloride but the difference between the percentages in the two series is not significant.

Thus the type of paralysis in mild and moderate cases did not tend to mildness, whereas in severe cases it definitely did, in the series which received extra sodium chloride. In very severe cases the paralysis showed a tendency to be of the severe type but in this group again the reason for this result will become obvious when later results are analysed.

FATAL CASES.

For purposes of analysis the fatal cases in Group IV in which group all the deaths occurred, are divided into each of the original sub-groups from which Group IV was formed (see the Introduction). It is now seen that no deaths occurred in sub-group C6 among the cases which received extra sodium chloride; hence sub-group C6 and sub-group C7 are combined for analysis of the fatal cases.

(a) DEATH WITHIN 24 HOURS OF ADMISSION (see Table 12)

It is unlikely that cases which die after a period

of less than twenty-four hours in hospital will have been long enough under treatment to have shown any beneficial effect which may have been present. It is not surprising therefore that the results given in Table 12 are contradictory to the general findings. Here the result in the less severe sub-groups (C6 & C7) appears to be favourable to the cases which received extra sodium chloride and in the more severe sub-group (C8) to the cases which did not receive extra sodium chloride while, in Group IV as a whole, the balance of favour is towards the cases which did not receive sodium chloride. There is no statistical significance in the results obtained.

(b) DEATHS WITHIN FOUR DAYS OF ADMISSION (see Tables 13 & 14)

Here it is evident that a smaller percentage of deaths occurred within four days of admission in cases which received extra sodium chloride but the result was not significant. In the less severe sub-groups (C6 and C7) a smaller percentage of deaths within four days of admission occurred in the cases which received extra sodium chloride and the result was significant; in the more severe sub-group (C8) a smaller percentage of deaths within four days of admission occurred in the cases which did not receive extra sodium chloride but the result was not significant.

If cases which ended fatally within twenty-four hours of admission are discounted on the ground that they cannot have been influenced by treatment, the results are the same with the exception that in the more severe sub-group (C8) the percentage of deaths within four days of admission is identical in the two series of cases.

It may be concluded, therefore, that inclusion of cases which ended fatally in the series which received extra sodium chloride tended to delay the fatal ending beyond the end of the fourth day.

(c) DEATHS WITHIN SEVEN DAYS OF ADMISSION (see Tables 15 & 16)

A smaller percentage of deaths occurred within seven days of admission in the cases which received extra sodium chloride but the result was not significant. In the less severe sub-group (C6 and C7) a smaller percentage of deaths occurred within seven days of admission in the cases which received extra sodium chloride but the result was not significant; in the more severe sub-group (C8) a smaller percentage of deaths occurred within seven days of admission in the cases which did not receive extra sodium chloride but the result was not significant.

Again, if cases which ended fatally within twenty-four hours of admission are discounted for the reason al-

ready given it is seen that, in the main group and in both the sub-groups, there is a smaller percentage of deaths before the end of the seventh day in the cases which received extra sodium chloride.

The conclusion here is that inclusion of cases which ended fatally in the series which received extra sodium chloride tended to delay the fatal ending beyond the end of the seventh day.

(d) DEATHS WITHIN FOURTEEN DAYS OF ADMISSION (see Tables 17 and 18)

A smaller percentage of deaths occurred within fourteen days of admission in the cases which received extra sodium chloride when the deaths were considered as a whole and in each of the sub-groups; the results however were not significant. The same result was obtained if deaths within the first twenty-four hours were excluded.

The conclusion, therefore, is that inclusion of cases which ended fatally in the series which received extra sodium chloride tended to delay the fatal ending till after the end of the fourteenth day.

To summarise the conclusions, it seems that the placing of very severe cases of Diphtheria, in whom a fatal ending is to occur, in a series of cases which receive extra sodium chloride has the effect of delaying the time

of death in these cases beyond the time at which death will occur if they are placed in a series which do not receive extra sodium chloride. Following this conclusion further it appears probable that some cases which will end fatally at a late stage,(e.g. after the fourteenth day),if they are not included in the series which receive sodium chloride,will be able to make a complete recovery as a result of this inclusion. But the recovery of very severe cases of this kind will probably occur only with the accompaniment of either cardiac or muscular paralyses, or of both.

It will be remembered that in the various groups of recovered cases the results were generally better in the series of cases which received extra sodium chloride and that the improvement in the results became more definite as the severity of the acute stage increased from that in Group I to that in Group III while the improvement in Group IV was less obvious or was entirely absent. It will be remembered further that all the deaths in the present investigation occurred in Group IV cases. The conviction therefore grows that the number of recovered cases in Group IV, which received extra sodium chloride, has been added to by cases which would have ended fatally had they

not been given extra sodium chloride and that the presence of these cases made the results among recovered cases in Group IV appear less impressive.

At this point reference should be made to Table 19 which shows that in Group IV, the greatest proportion of cases, in the series which received extra sodium chloride, occurred in the most severe sub-group, whereas the greatest proportion of cases in the series which did not receive extra sodium chloride occurred in the least severe sub-group. These facts indicate that the natural tendency is for the results to be less favourable in the former series than in the latter, because Begg has shown that results become more serious as the severity of the acute stage increases. A similar result is found when deaths of cases with less than twenty-four hours' treatment are discounted as in Table 20.

Finally, reference should be made to Table 21 which shows the distribution of recovered cases according to the severity of the acute stage within Group IV. In the group as a whole a greater percentage of recoveries occurred in cases which received extra sodium chloride but the result was not statistically significant. In the least severe subgroup (C6) a greater percentage of recoveries

occurred in the series of cases which received extra sodium chloride and this result was almost of statistical significance. In the most severe sub-group (C8) a greater percentage of recoveries occurred in the cases which received extra sodium chloride and the result was statistically significant. In the intermediate sub-group (C7) the greater percentage of recoveries occurred in the cases which did not receive extra sodium chloride but the result was not statistically significant. Similar conclusions are reached by consideration of Table 22 which is calculated in a similar manner but which excludes cases which ended fatally within twenty-four hours of admission to hospital.

The benefits of the administration of sodium chloride to a series of cases of Diphtheria may therefore be stated to have been,

(1) reduction in the total number of deaths,

(2) postponement of death in those cases which ended fatally,

(3) reduction in the number of cases with paralysis,

(4) diminution in the incidence of severe paralysis,

(5) increase in the number of cases which recovered without developing paralysis and which were considered to be physically ready for dismissal at the minimum period of hospital residence.

Careful survey also revealed that increase in the number of satisfactory recoveries happened as a result of reduction in the number of delayed recoveries but that actually very little change occurred in these latter cases because of an influx of cases which occurred as a result of reduction in the number of recoveries with paralysis.

A quantitative estimate of the improved results which were produced in this series of cases of diphtheria by the administration of sodium chloride was made in the following manner:

> The cases in each series, according to whether they received extra sodium chloride or did not receive extra sodium chloride, were arranged in their respective sub-groups A1, etc., B1, etc., C2, etc. The cases in each series and in each sub-group were then subdivided according to age-groups and according to the date of death or the type of recovery.
>
> In each age-group thus arranged the total number of cases in both series were found; the results in the series of cases which did not receive extra sodium chloride were calculated as if the total number of cases in this series was equal to the total number of cases in the series which received extra sodium chloride. The effect of this procedure was to produce an array of results which was theoretically a measure of what would have happened in the series of cases which received extra sodium chloride if they had not received this treatment.
>
> The summation of the results obtained in each age-group and in each sub-group in this fashion

gave a new set of values for the whole series of cases which did not receive sodium chloride; this gave an estimate of what would have occurred in the other series if sodium chloride had not been given.

The detailed results of the calculation are given in Tables 23 to 38 inclusive. Table 39 shows the final summation and Table 40 shows the results which would have been expected to occur if each series had consisted of two hundred similar cases.

Using whole numbers, the quantitative estimate of the results in a hundred cases which received extra sodium chloride and in a hundred similar theoretical cases which did not receive extra sodium chloride is given in the following Table which is derived from Table 40.

DESCRIPTION OF CASES	CASES WHICH RECEIVED EXTRA SODIUM CHLORIDE (Actual %)	CASES WHICH DID NOT RECEIVE EXTRA SODIUM CHLORIDE (Comparative %)
Cases of satisfactory recovery	59	51
Cases of uncomplicated recovery	70	63
Cases of recovery without severe paralysis	89	84
Cases of recovery	93	87
Cases alive after 14 days' treatment	94	87
Cases alive after 7 days' treatment	96	94
Cases alive after 4 days' treatment	98	96
Cases alive after 24 hrs.	99	99
TOTAL CASES	100	100

This table shows that the improvement in the results of a series of cases which received extra sodium chloride was present throughout the series.

Section IV.

SUMMARY AND COMMENTS.

The subject of suprarenal cortex deficiency has been examined in detail with reference to Diphtheria. In Section I, an account has been given of suprarenal cortex deficiency as it has been observed by various workers in animal experiments and in human disease. In Section II particular attention has been given to Diphtheria and the opportunity has been taken of describing various clinical, biochemical and pathological features of the disease, some of which have not been shown previously, with the object of demonstrating the definite connection which is present between the results of general intoxication in Diphtheria and the results of suprarenal cortex deficiency. In Section III, it has been shown that the placing of cases in a regime which includes the administration of a definite additional quantity of sodium chloride to them is of undoubted benefit to these cases and this, again, is consistent with the idea that suprarenal cortex deficiency is present in Diphtheria because sodium chloride has been shown to be of value in cases of suprarenal cortex deficiency.

It may be objected that the work described in Section

III has not sufficiently differentiated between sodium chloride and glucose when giving credit to the substance which has produced the improved results in one series of cases. This was, however, considered during the actual investigation and one is quite decided that sodium chloride is the beneficial agent for the following reasons:

(1) In the Groups of mild, moderate and severe cases (Groups I, II and III) there were no cases which received continuous rectal glucose saline irrigation; the only difference between the treatment of two parallel series of cases was that one series received extra sodium chloride by mouth and the other did not; (2) in the Group of very severe cases (Group IV) there was a small group of sixteen cases which was given sodium chloride only by intravenous injection of hypertonic saline solution; thirteen of these cases recovered completely without developing paralysis and the other three recovered after developing severe paralysis; of sixteen similar cases admitted at corresponding times, all developed some degree of paralysis; (3) in the cases in Group IV which received continuous rectal glucose saline the quantity of glucose ingested, viz. about 300 grammes per day which is sufficient to produce in that time 1240 Calories, is scarcely sufficient to supply the

demands of a normal patient far less the requirements of a patient who is acutely ill; (4) in three cases, in my experience, the administration of pure glucose in repeated small doses intravenously failed to avert a fatal issue.

The author is very much indebted to Dr. W.M. Elliott, Physician-Superintendent of Ruchill Fever Hospital, for the facilities which he gave for conducting this investigation.

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

BIBLIOGRAPHY.

(1) Van BIE: Deut.Med.Woch, 1929: 55, 563.

(2) Van BIE: Acta.Med.Scand., 1930: 56, 539.

(3) ROGOFF and STEWART: Jour.Amer.Med.Assoc., 1929: 92, 1569.

(4) SWINGLE and PFIFFNER: Science, 1930: 72, 75.

(5) BROWN-SEQUARD: quoted by RABINOWITZ: Amer.Jour. Med.Sci. 1923: 166, 516.

(6) HARTMAN, BROWNELL and LOCKWOOD: Endocrinology, 1932: 16, 521.

(7) BRITTON and SILVETTE: Amer.Jour.Phys. 1933: 104, 399. Ibid., 1934: 107, 190.

(8) HARROP, WIDENHORN and WEINSTEIN: Münch.Med.Woch (Jan.1929).

(9) HARROP, WEINSTEIN, SOFFER and TRESCHER: Jour.Exp. Med. 1933: 58, 1.

(10) ADDISON: A collection of the published writings of the late Thomas Addison, Practitioner, 1903: 71, 472.

(11) ROWNTREE: the article on Addison's Disease in a Text Book of Medicine by American authors, third edit., edited by Russell L.Cecil, 1933, p.1249.

(12) HARROP, WEINSTEIN, SOFFER and TRESCHER: Jour.Amer. Med.Assoc. 33⅓ 100, 1850.

(13) LOEB: Science, 1932: 76, 420.

(14) LOEB: Proc.Soc.Exp.Biol.and Med., 1933: 30, 808.

(15) LOEB, ATCHLEY, GUTMAN and JILLSON: Ibid., 1933: 31, 130.

(16) LUCKE, WIGHT and KIME: Arch.Int.Med., 1919, 24, 19.

(17) ADAMI: Jour.Amer.Med.Assoc., 1899: 33, 1506 and 1572., and Principles of Pathology, vol.1, 1909, p.425.

(18) RUBINZTEJN: These de Paris, 1934, no.134.

(19) PEARL and BRUNN: Surg.,Gyn., and Obstet., Sept.1928, p.393.

(20) RABINOWITZ: Amer.Jour.Med.Sci., 1923: 166, 517.

(21) KER: Infectious Diseases, revised by Rundle, 1929.

(22) ROLLESTON: Acute Infectious Diseases, 1925.

(23) THADDEA: Klin.Woch, 7.9.35 (Annotation in British Medical Journal, 23.11.35).

(24) MUIR: Text Book of Pathology, 1924.

(25) GRAY'S ANATOMY: 22nd Edition, 1923, edited by Howden.

(26) HALE-WHITE: Materia Medica, 18th Edition, 1924,p.673.

(27) PETERS and Van SLYKE: Quantitative Clinical Methods, vol.1.

(28) MONRO: Manual of Medicine, fifth Edition, 1925.

(29) HURST: in discussion at the meeting of the Medical Society of London, 24.11.31.

(30) DODDS: in discussion at the meeting of the Medical Society of London, 24.11.31.

(31) SCHWENTKER and NOEL: Bull.Johns.Hopkins Hospital, June 1930, p.358.

(32) DONATO: These de Paris, 1930, no.323.

(33) LEREBOULLET, GOURMAY and DONATO: Ann.de Med., May 1931, p.547.

(34) POLANDOWSKI: These de Paris, 1932, no.35.

(35) BENN, HUGHES and ALSTEAD: Lancet, 1932, i, 281.

(36) BREMS: Acta.Med.Scand., 14.12.32, p.1.

(37) BEGG and HARRIES: Lancet, 2nd March, 1935, p.480.

(38) HERBERT and BOURNE: British Medical Journal, vol.1, 1931: 94.

(39) PETERS and Van SLYKE: Quantitative Clinical Methods, vol.II.

(40) MURPHY: reference in Da Costa's Modern Surgery, ninth Edition, 1925.

(41) VOLHARD and KORANYI: As described by T.C.Hunt in a post-graduate Lecture at St.Mary's Hospital, London, 1929.

A P P E N D I X A.

Micro-Method for Blood Sugar Determination.

(Folin-Wu Method modified by Freda K Herbert and Margherita C. Bourne, B.M.J., I, 1931, p.95)

Reagents.

1. 3% sodium sulphate ($Na_2SO_4.10H_2O$)
2. 10% sodium tungstate ($Na_2WO_4.2H_2O$)
3. 2/3N sulphuric acid.
4. Folin-Wu copper reagent.*
5. Phosphomolybdic acid reagent.**
6. Standard glucose solution (a stock solution of 1% glucose in 0.3% benzoic acid is diluted to 0.01% for use)

* Alkaline-Copper Reagent. Dissolve 40 gm. anhydrous Na_2CO_3 in about 400 cc. H_2O and place in 100 cc. flask; add 7.5 gm. tartaric acid, and when dissolved, add 4.5 gm crystallized $CuSO_4$; mix and make up to 1000 cc. With impure carbonate a sediment may form in a week or so; in such a case, transfer the clear supernatant fluid to another bottle.

** Molybdic-Phosphate Solution: Put 35 gm. molybdic acid in a 1000 cc. beaker. Add 5 gm. sodium tungstate, 200 cc. 10% NaOH and 200 cc. distilled H_2O. Boil vigorously 20 to 40 minutes to remove NH_3, cool and dilute to 350 cc. c distilled H_2O. Add 125 cc. 85% phosphoric acid and dilute to 500 cc. c distilled water.

Protein Precipitation.

Take 3.6 cc. sodium sulphate solution and measure into it 0.2 cc. blood. Add 0.1 cc. tungstate solution and 0.1cc 2/3NH_2SO_4. Mix the reagents without undue shaking and separate the fluid at once by centrifuging and pouring the

supernatant fluid through a small filter (filter paper Whatman no.41, 5.5 cmm.). It is possible to obtain just over 2 cc. of filtrate provided the filter is of the size mentioned.

Analysis.

In 3 Folin's tubes (graduated at 12.5 cc. and 25 cc.) A, B, C take

A.	B.	C.
2cc filtrate 2cc Folin-Wu copper Soln.	1cc 0.01% glucose (ie. 0.1mg. glucose 1cc water 2cc Folin-Wu copper soln.	2cc 0.01% glucose 2cc Folin-Wu copper soln.

B and C are the standards. Mix the contents of the tubes and place them in a boiling water bath for 6 minutes. Cool for 1 or 2 minutes without shaking. To each tube add 2 cc. phosphmolybdic acid solution. Dilute to the 12.5 cc. mark $\overline{c}$ water and mix thoroughly. Compare the unknown A in the colorimeter with the standard either B or C, whichever is the closer to it in colour.

Calculation.

In the protein precipitation, 0.2 cc. blood was diluted to a total volume of 4 cc. Of the filtrate 2 cc. were taken, i.e. a volume equivalent to 0.1 cc. blood. Let S be the reading of the standard, U the reading of the un-

known and W be mgm glucose in the 2 cc. filtrate taken. Standard B contains 1 cc. of 0.01% glucose - i.e. 0.1 mgm glucose. Then

$$W \times U = 0.1 \times S$$

$$\therefore \quad W = \frac{0.1 \times S}{U}$$

$\therefore$ 2 cc. filtrate contain $\frac{0.1 \times S}{U}$ mgm glucose.

$\therefore$ 0.1 cc blood contains $\frac{0.1 \times S}{U}$ mgm glucose.

$\therefore$ 100 cc. blood contain $\frac{0.1 \times S \times 100}{0.1 \times U} = \frac{S}{U} \times 100$

Similarly, for the standard C, the blood sugar is given by $\frac{S}{U} \times 200$ mgm. glucose.

Estimation of Sodium in Blood Serum

(Modified from Peters and Van Slyke and from Hawk).

Reagents.

1. Potassium Pyroantimonate Reagent.*
2. 10% alcohol-washed potassium hydrate solution.
3. 95% alcohol (redistilled over caustic potash)
4. 10N hydrochloric acid or concentrated hydrochloric acid of specific gravity 1-182.
5. 20% potassium Iodide solution.
6. N/10 sodium thiosulphate solution (accurately titrated).
7. 1% freshlyp prepared soluble starch solution.

* Potassium pyroantimonate reagent: 500 cc. of distilled water are heated to boiling point in a Pyrex flask and approximately 10 grammes of potassium pyroantimonate are added. The boiling is continued for five minutes after which the flask is cooled under running water; when the contents are cold 15 cc. of 10% potassium hydrate solution (a lcohol-washed) are added. The reagent is filtered through ash-free filter paper into a paraffined bottle; it is usual to find that some undissolved potassium pyroantimonate passes through even the finest filter paper, but if the reagent is allowed to stand for twenty-four hours the undissolved salt will settle to the bottom and the supernatant fluid can be used as the reagent. It usually keeps well at room temperature for one month. 10 cc. of the reagent precipitates about 11 milligrammes of sodium. Before use 2 cc. of distilled water and 3 cc. of 95% alcohol (as above) should be added to 10 cc. of the reagent to ensure that the alcohol (which is in the proportion used in the method) does not precipitate the pyroantimonate.

Method.

Place 1 cc. of serum and 1 drop of 10% potassium hydrate solution in a graduated pyrex centrifuge tube, preferably coated with a thin layer of paraffin. Add 5 cc. of the potassium pyroantimonate reagent and mix thoroughly by rolling the tube between the palms of the hands with the opennend uppermost. Add exactly 1.5 cc. of 95% alcohol and mix in the same manner till a uniform turbidity is obtained. Cover the top of the tube and allow to stand for 30 minutes. Centrifuge for 5 minutes and siphon off all but 2 cc. of supernatant fluid. Add 5 cc. 30% alcohol (30 cc. of 95% alcohol made up to 95 cc. with distilled water), mix and recentrifuge for 5 minutes. Repeat twice. Remove all supernatant fluid with a pipette, the nozzle of which is fine and turned on itself.

Add to the precipitate of sodium pyoantimonate at the foot of the centrifuge tube, 2½ cc. of ⅒N hydrochloric acid, stirring thoroughly with a glass rod to aid solution of the precipitate. Continue till solution is complete. Transfer to a tall beaker aiding transfer with not more than 5 cc. distilled water. Add 2 cc. 20% potassium iodide solution and titrate at once with N/10 sodium thiosulphate solution adding the latter very rapidly with constant stirr-

ing till the yellow colour has almost gone; add 0.5 cc. soluble starch solution and continue adding thiosulphate solution until the blue colour has gone and the contents of the beaker are clear, when the end point is reached.

At the same time controls are carried through with known solutions of sodium chloride of strength equivalent to the solution of sodium in blood serum.

Calculation.

Iodine is produced by the action of antimony (freed from combination in sodium pyroantimonate by the action of hydrochloric acid) on potassium iodide and it is known that one equivalent of iodine is filled by the amount of antimony bound to 0.5 equivalent of sodium. Therefore 1000 cc. N/1 thiosulphate is equivalent to 0.5 equivalent of sodium and 1 cc. n/10 thiosulphate is equivalent to 0.05 milli-equivalent of sodium. If X cc. of N/10 thiosulphate are required the serum sodium in milli-equivalents per litre is,

X x (thiosulphate factor) x 0.05 x 1000

or 50X x (thiosulphate factor)

In actual practice Peters and Van Slyke make a small correction and the figure used is 48.6X x (thiosulphate factor).

Estimation of Chlorine in Blood Serum.

(Peters and Van Slyke.)

Reagents:

1. Van Slyke and Sendwy's reagent: N/200 silver nitrate in concentrated nitric acid of specific gravity 1.4 (Dissolve 8.495 gm. fused silver nitrate in a minimum amount of water and make up to 1 litre with concentrated nitric acid.) The solution is kept in a coloured bottle in the dark and keeps indefinitely.

2. Saturated solution of potassium permanganate.

3. N/50 ammonium sulphocyanide solution (1.5 gm. of salt is dissolved in 900 cc. and the solution is further diluted till 7.5 cc. are required to titrate 3 cc. of the silver nitrate reagent.)

4. 5% ferric alum solution.

5. Concentrated nitric acid solution.

Method.

To 1 cc of serum in a 25 x 200 mm. Pyrex glass tube add slowly and with constant stirring 3 cc. of N/200 silver nitrate in concentrated nitric acid. Add 2 cc. of concentrated nitric acid and bring to the boil over a free flame; add saturated potassium permanganate solution to the boiling mixture, one drop at a time, until the digestion mixture becomes clear. Wash down the sides of the tube with not more than 5 cc. of distilled water and boil again till the mixture is clear: it may be necessary to add several more

drops of saturated potassium permanganate at this stage. The contents of the tube are cooled rapidly to room temperature by placing the tube under running water and 6 cc. of ferric alum solution is added. The excess silver nitrate is titrated with N/50 ammonium, sulphocyanide until a pink colour appears and persists for fifteen seconds.

Calculation.

If x = number of cc. sulphocyanide used, then 20(7.5 - x) = milli-equivalents of chlorine in 1 litre blood serum.

A control experiment using 1 cc of a known strength of solution containing chlorine (as sodium chloride) is carried through at the same time.

Estimation of Potassium in Blood Serum.

(Modified from Peters and Van Slyke)

Reagents:

1. Distilled water.
2. 10% sodium tungstate solution.
3. 2/3N sulphuric acid solution.
4. 5% silver nitrate solution.
5. Sodium cobaltinitrite reagent.
6. 40% silver nitrate solution.
7. N/5 sodium hydrate solution.
8. 0.5% solution of sulphanilic acid in 5% hydrochloric acid.
9. 0.5% solution of naphthylamine in 5% hydrochloric acid.
10. Standard solution of potassium sulphate (0.08 mgm. in 5 cc.).

Sodium cobaltinitrite reagent: (A) Dissolve 25 gm. cobalt nitrate crystals in 50 cc. of water and add 12.5 cc. glacial acetic acid.

(B) Dissolve 120 gm. sodium nitrite (potassium free) in 180 cc. of water.

Add 210 cc. of (B) to the whole of (A) and draw air through the solution until all the nitric oxide gas given off is removed. Place reagent in paraffin-lined bottle in refrigerator. It keeps well for one month but should be filtered before use.

To prepare this reagent for use add 1 cc. of 40% silver nitrate solution to each 10 cc. of sodium cobaltinitrite reagent, mix thoroughly and filter. The filtrate may be called silver cobaltinitrite reagent.

Method.

To 1 cc. serum add 7.5 cc. distilled water, 0.5 cc. sodium tungstate solution, 0.5 cc. 2/3N sulphuric acid, and 0.5 cc. 5% silver nitrate solution. Mix thoroughly and centrifuge till a clear supernatant fluid is present. Take 5 cc. of this fluid and 5 cc. of standard potassium sulphate and place in separate centrifuge tubes. Add 2 cc. of silver cobaltinitrite reagent to each and allow to stand for 2 hours. Centrifuge thoroughly and remove the supernatant fluid with a pipette, the nozzle of which is turned on itself. Wash several times with distilled water, centrifuging and removing the fluid on each occasion. After complete washing add 5 cc. of N/5 sodium hydrate solution to each and heat to the boiling point; centrifuge thoroughly to deposit all the black precipitate and, if the supernatant fluid is not clear, filter. Wash filtrates into 50 cc. volumetric flasks and make up to the volume with distilled water. Mix thoroughly and transfer 8 cc. from each into 100 cc. volumetric flasks; add about 60 cc. of distilled water, 2 cc. of 0.5% sulphanilic acid solution and 1 cc. of 0.5% naphthylamine solution and make up to 100 cc. with distilled water. Mix and allow to stand for 10 minutes when the two may be compared in a colorimeter.

Calculation.

$$\text{Value of potassium in mgm/100 cc} = \frac{\text{Reading of Standard}}{\text{Reading of Unknown}} \times 16$$

$$\therefore \text{Value of potassium in milli-equivalents per litre} = \frac{\text{Reading of Standard}}{\text{Reading of Unknown}} \times \frac{16}{3.91}$$

A P P E N D I X B

"W A T E R T E S T"

CASE XI. A.L., female, 12 years of age.

This patient was admitted on 10.5.33, the sixth day of illness. The severity of the acute stage was that of Group I. She recovered without developing paralysis and was dismissed on 19.6.33.

The Water Test reveals deficiency in the excretion of water by the kidneys on admission.

DATE	11.5.33		18.5.33		25.5.33		1.6.33		8.6.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm-9am.	2½	1020	6	1032	12	1038	12	1034	32	1020
10 am.	2	1032	8	1002	1½	1020	4	1010	18	1000
11 am.	11	1002	12	1000	16	1003	16	1000	12	1000
12 noon	2	1006	7	1001	10	1003	0	-	5	1006
1 pm.	0	-	0	-	1½	1020	1½	1020	2	1018
2 pm.	1)	1024	1)	1032	1)	1030	¾)	1028	1¼)	1022
3 pm.	1)		1)		1½)		1)		½)	
QUANTITY: 2hrs	13		20		17½		20		30	
DEFICIENCY: "	2		0		0		0		0	
% DEFICIENCY	13½		0		0		0		0	
QUANTITY: 5hrs	16		28		30		22¼		38¼	
DEFICIENCY: "	14		2		0		7¾		0	
% DEFICIENCY	46 2/3		6 2/3		0		26%		0	
MINIMUM DEFICIENCY	13⅓		0		0		0		0	

Q. - Quantity in ounces.
S.G. - Specific Gravity.

CASE XII. M.G., female, 9 years of age.

This patient was admitted on 18.5.33, the second day of illness. The severity of the acute stage was that of Group II. She recovered without developing paralysis and was dismissed on 27.6.33.

The Water Test shows deficiency in the excretion of water at the time of admission.

DATE	19.5.33		26.5.33		2.6.33		9.6.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am	32½	1016	16	1022	16	1024	5	1028
10 am.	1)	1028	10	1000	6	1002	2¼	1002
11 am.	1)		10	1000	15	1000	14	1000
12 noon	0	-	4	1012	6	1000	4	1000
1 pm.	4	1012	1½)	1010	2½	1016	1½	1004
2 pm.	1)	1020	1¼)		1)	1018	¾)	1022
3 pm.	1)		1	-	1)		¾)	
QUANTITY: 2hrs	2		20		21		16¼	
DEFICIENCY: "	12		0		0		0	
% DEFICIENCY	86		0		0		0	
QUANTITY: 5hrs	7		26¾		30½		22¼	
DEFICIENCY: "	23		3¼		0		7¾	
% DEFICIENCY	76⅔		10⅝		0		25	
MINIMUM DEFICIENCY	76⅔		0		0		0	

CASE XIII. M.J., female, 32 years of age.

This patient was admitted on 20.5.33, the second day of illness. The severity of the acute stage was that of Group III. The patient's recovery was complicated by the presence of a mild form of muscular paralysis. She was dismissed well on 10.7.33.

The Water Test shows deficiency in the excretion of water at the time of admission.

DATE	21.5.33		28.5.33		4.6.33		11.6.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am	16	1030	10	1034	26	1012	0	-
10 am.	0	-	0	-	22	1020	0	-
11 am.	0	-	0	-	0	-	34	1010
12 noon	0	-	0	-	0	-	0	-
1 pm.	10		0	-	0	-	0	-
2 pm.	0	-	0	-	0	-	13	1010
3 pm.	0	-	32	1010	23	1004	0	-
QUANTITY: 2hrs	0		0		22		34	
DEFICIENCY: "	15		15		0		0	
% DEFICIENCY	100		100		0		0	
QUANTITY: 5hrs	10		about 31*		about 44*		47	
DEFICIENCY: "	20		0		0		0	
% DEFICIENCY	66⅔		0		0		0	
MINIMUM DEFICIENCY	66⅔		0		0		0.	

* On the assumption that 1 oz. is excreted in the 6th hours.

CASE XIV. E.O., female, 12 years of age.

This patient was admitted on 23.5.33, the third day of illness. The severity of the acute stage was that of Group III. She recovered without developing paralysis and was dismissed on 4.7.33.

The Water Test reveals deficiency in the excretion of water at the time of admission.

DATE	24.5.33		31.5.33		7.6.33		14.6.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am	5	1046	10	1040	10	1034	10	1032
10 am.	3	1042	12	1002	12	1002	4	1000
11am.	1)	1024	20	1000	16	1000	26	1000
12 noon	1½)		0	-	2½	1008	0	-
1 pm.	0	-	0	-	0	-	2	1012
2.pm.	1	1024	0	-	2	1008	0	-
3 pm.	0	-	3	1020	0	-	0	-
QUANTITY: 2hrs	4		32		28		30	
DEFICIENCY: "	11		0		0		0	
% DEFICIENCY	73⅓		0		0		0	
QUANTITY: 5hrs	6½		32		32½		32	
DEFICIENCY: "	23½		0		0		0	
% DEFICIENCY	78⅓		0		0		0	
MINIMUM DEFICIENCY	73⅓		0		0		0	

CASE XV. G.B., female, 9 years of age.

This patient was admitted on 23.5.33, the third day of illness. The severity of the acute stage was that of Group III and a throat swab yielded morphologically typical C.diphtheriae. Recovery was normal until 14.6.33 when a fresh attack of typical diphtheria, of Group I severity, took place; the throat swab still yielded C.diphtheriae. Further recovery was accompanied by the occurrence of mild muscular paralysis. The patient was dismissed well on 26.7.33.

The Water Test reveals deficiency in the excretion of water on admission and from the second to the fifth weeks of treatment, inclusive.

DATE	24.5.33		31.5.33		7.6.33		14.6.33		21.6.33		28.6.33		11.7.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	2	1042	8	1028	11	1032	3	1034	8	1036	8	1040	12	1018
10 am.	½		6	1001	2½	1000	2	1004	4	1004	0	-	4	1004
11 am.	1½	1012	12	1000	10	1000	6	1002	8	1000	6	1000	18	1000
12 noon	2½	1010	6	1010	5	1002	1	1016	9	1000	4	1000	2	1000
1 pm.	1		1½	1008	1		1	1016	1		1		1	
2 pm.	1	1020	½		¾	1020	0	-	0	1018	0	1020	1	1016
3 pm.	¾	-	½	1028	1	-	1	-	1½		1½		1	-
QUANTITY: 2hrs	2		18		12½		8		12		6		22	
DEFICIENCY: "	13		0		2½		7		3		9		0	
% DEFICIENCY	86⅔		0		16⅔		46⅔		20		60		0	
QUANTITY: 5hrs	6½		26		19½		10		22½		11½		26	
DEFICIENCY: "	23½		4		10½		20		7½		18½		4	
% DEFICIENCY	78⅓		13⅓		35⅚		66⅔		25		61⅔		13⅓	
MINIMUM DEFICIENCY	78⅓		0		16⅔		46⅔		20		60		0	

CASE XVI. M.D., female, 18 years of age.

This patient was admitted on 24.5.33, the fourth day of illness. The severity of the acute stage was that of Group I. She recovered without developing paralysis and was dismissed on 4.7.33.

The Water Test reveals deficiency in the excretion of water on admission.

DATE	25.5.33		1.6.33		8.6.33		15.6.33		22.6.33	
TIME	Q.	S.G.	Q.	S.P.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm-9am	4	1042	7	1032	28	1028	15	1030	4	1024
10 am.	2	1032	6	1008	25	1002	4	1010	10	1008
11 am.	4	1006	14	1002	12	1002	22	1000	12	1000
12noon	0	-	2	1020	3	1012	2½	1002	1)	1012
1 pm.	0	-	¾)	1022	3	1018	2	1010	1)	
2 pm.	0	-	2½)		1½	1018	1)	1026	1½	1010
3 pm.	4½	1030	¾	-	1½	1020	1½)		1½	1010
QUANTITY: 2hrs	6		20		37		26		22	
DEFICIENCY: "	9		0		0		0		0	
% DEFICIENCY	60		0		0		0		0	
QUANTITY: 5hrs	about 9½		25¼ *		44½		31½		25½	
DEFICIENCY: "	20½		4¾		0		0		4½	
% DEFICIENCY	68⅓		15⅝		0		0		15	
MINIMUM DEFICIENCY	60		0		0		0		0	

* On the assumption that 1 oz. of urine was excreted in the sixth hour.

CASE XVII. J.R., female, 19 years of age.

This patient was admitted on 25.5.33, the third day of illness. The severity of the acute stage was that of Group II. Recovery was interrupted by the presence of mild cardiac paralysis from the end of the second week till the middle of the fourth week of treatment. She was dismissed well on 5.9.33.

The Water Test revealed deficiency in the excretion of water by the kidneys on admission and at the end of the first and second weeks of treatment.

DATE	26.5.33		2.6.33		9.6.33		21.6.33		28.6.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am	12	1040	20	1020	9	1030	12	1036	16	1026
10 am.	0	-	0	-	½	-	0	-	22	1000
11 am.	0	-	0	-	14	1002	23	1000	5	1010
12noon	0	-	8	1018	0	-	15	1000	6	1010
1 pm.	0	-	0	-	0	-	0	-	1	1010
2 pm.	0	-	2	1018	2½	1020	0	-	1	
3 pm.	5	1032	1½	1004	0	-	0	-	1½	1006
QUANTITY: 2hrs	0		0		14½		23		27	
DEFICIENCY: "	15		15		½		0		0	
% DEFICIENCY	100		100		3⅓		0		0	
QUANTITY: 5hrs	4		10		17		38		35	
DEFICIENCY: "	26		20		23		0		0	
% DEFICIENCY	86⅔		66⅔		76⅔		0		0	
MINIMUM DEFICIENCY	86⅔		66⅔		3⅓		0		0	

CASE XVIII. M.J., female, 9 years of age.

This patient was admitted on 25.5.33, the third day of illness. The severity of the acute stage was that of Group II. Recovery took place without the development of paralysis and the patient was dismissed well on 14.7.33.

The Water Test revealed deficiency in the excretion of water by the kidneys on admission and one week later.

DATE	26.5.33		2.6.33		9.6.33		16.6.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am	9	1040	14	1022	6	1024	16	1026
10 am.	1¼)	1030	2	1006	4	1012	14	1002
11 am.	1½)		2½	1004	14	1000	10	1000
12noon	1)	1032	1½	1018	2	1004	5	1006
1 pm.	1)		1¾)	1020	0		0	
2 pm.	¾)	1030	1¼)		1)	1020	2½	1010
3 pm.	1)		1	-	1)		2	1012
QUANTITY: 2hrs	2¾		4½		18		24	
DEFICIENCY: "	12¼		10½		0		0	
% DEFICIENCY	81⅔		70		0		0	
QUANTITY: 5hrs	5½		8		21		31½	
DEFICIENCY: "	24½		22		9		0	
% DEFICIENCY	81⅔		73⅓		30		0	
MINIMUM DEFICIENCY	81⅔		70		0		0	

CASE XIX. M. McK., female, 28 years of age.

This patient was admitted on 16.6.33, the sixth day of illness. The severity of the acute stage was that of Group II. She recovered without developing paralysis and was dismissed on 5.8.33.

The Water Test showed deficiency in the excretion of water on admission and one week later.

DATE	17.6.33		24.6.33		1.7.33		8.7.33		15.7.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am	5	1042	5	1030	9	1030	14	1030	10	1032
10 am.	1½	1010	3	1018	4	1020	4	1020	16	1002
11 am.	3	1006	4	1002	18	1000	20	1002	8	1001
12noon	1)		½)	1008	4	1002	6	1010	3	1012
1 pm.	0)	1016	½)		4	1002	2	1016	4	1012
2 pm.	1)		0	-	1)	1004	1½)	1022	1½	1020
3 pm.	1)		0	-	1)		1½)		2	1020
QUANTITY: 2hrs	4½		7		22		24		24	
DEFICIENCY: "	10½		8		0		0		0	
% DEFICIENCY	70		53⅓		0		0		0	
QUANTITY: 5hrs	6½		8		31		33½		32½	
DEFICIENCY: "	23½		22		0		0		0	
% DEFICIENCY	78⅓		73⅓		0		0		0	
MINIMUM DEFICIENCY	70		53⅓		0		0		0	

CASE XX. A.R., female, 18 years of age.

This patient was admitted on 15.6.33, the second day of illness. The severity of the acute stage was that of Group II. She recovered without developing complications and was dismissed well on 26.7.33.

The Water Test on the day after admission was vitiated by the presence of menstruation; it was attempted again seven days later but was unsatisfactory because of vomiting. Tests at later stages did not reveal deficiency of excretion of water.

DATE	23.6.33		30.6.33		7.7.33		14.7.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am	16	1018	11	1026	18	1020	10	1032
10 am.	½)	1006	14	1002	14	1002	7	1006
11 am.	10)		12	1000	12	1000	16	1002
12noon	0	-	2	1004	3	1002	2½	1004
1 pm	2¼	1002	1¼)	1020	1)	1012	1)	1008
2 pm.	0		1)		1)		1)	
3 pm.	½	-	¾	-	1	-	1	-
QUANTITY: 2hrs	Patient vomited about 1 pt. soon after commencement of test.		26		26		23	
DEFICIENCY: "			0		0		0	
% DEFICIENCY			0		0		0	
QUANTITY: 5hrs			30¼		31		27½	
DEFICIENCY: "			0		0		2½	
% DEFICIENCY			0		0		8⅓	
MINIMUM DEFICIENCY			0		0		0	

CASE XXI. J.W., female, 31 years of age.

This patient was admitted on 20.6.33, the fourth day of illness. The severity of the acute stage was that of Group I. Recovery was uneventful and the patient was dismissed well on 29.7.33.

The Water Test showed deficiency in the excretion of water on admission and up till the end of the second week in hospital.

DATE	21.6.33		28.6.33		5.7.33		12.7.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q..	S.G.
9pm - 9am	10	1042	12	1036	8	1030	10	1032
10 am.	0	-	2	1008	1¾	1006	2	1008
11 am.	4	1028	7	1006	12	1002	14	1002
12noon	0	-	1)	1016	0	-	4	1010
1 pm.	0	-	¾)		0	-	1½	1016
2 pm.	0	-	1	-	2	1020	1)	1022
3 pm.	0	-	0		0	-	½)	
QUANTITY: 2hrs	4		9		13¾		16	
D EFICIENCY: "	11		6		1¼		0	
% DEFICIENCY	73⅓		40		8⅓		0	
QUANTITY: 5hrs	4		11¾		15¾		22½	
DEFICIENCY: "	26		18¼		14¼		7½	
% DEFICIENCY	83⅓		60%		47½		25	
MINIMUM DEFICIENCY	73⅓		40		8⅓		0	

CASE XXII. M.R., female, 9 years of age.

This patient was admitted on 28.6.33, the fourth day of illness. The severity of the acute stage was that of Group I. Recovery was uneventful and the patient was dismissed on 5.8.33.

The Water Test revealed a deficiency in the excretion of water on admission and one week after admission.

DATE	29.6.33		6.7.33		13.7.33		20.7.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	4	1026	3	1034	13	1030	10	1040
10 am.	1½	1006	0	-	7	1010	4	1004
11 am.	9	1002	12	1000	12	1000	14	1001
12noon	2½	1004	7	1000	12	1000	6½	1010
1 pm.	0	-	0	-	0	-	1½	1012
2 pm.	0	-	0	-	2¼	1020	0	-
3 pm.	0	-	3	1024	1	1024	0	-
QUANTITY: 2hrs	10½		12		19		18	
DEFICIENCY: "	4½		3		0		0	
% DEFICIENCY	30		20		0		0	
QUANTITY: 5hrs	13		19		33¼		26	
DEFICIENCY: "	17		11		0		4	
% DEFICIENCY	56⅔		36⅔		0		13⅓	
MINIMUM DEFICIENCY	30		20		0		0	

CASE XXIII. C.D., a female, 9 years of age.

This patient was admitted on 6.7.33, the fifth day of illness. The severity of the acute stage was that of Group I. The patient recovered without developing paralysis and was dismissed well on 16.8.33.

The Water Test showed deficiency of excretion of water on admission and one week later; the test at the end of the second week was normal but there was deficiency again one week later; after that the test was normal.

DATE	6.7.33		13.7.33		20.7.33		27.7.33		3.8.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am	3	1020	26	1015	12	1032	13	1028	4	1032
10 am.	1	1020	0	-	3	1006	3	1004	4	1002
11 am.	0	-	11	1002	14½	1000	7	1000	12	1000
12noon	3	1000	1½)	1010	6	1000	9	1000	7	1000
1 pm.	0	-	1)		2	1016	3¾	1001	5	1002
2 pm.	¾)	1012	1)	1026	0	-	1	1008	2	1004
3 pm.	¾)		1)		1	1020	0	-	1	1016
QUANTITY: 2hrs	1		11		17½		10		16	
DEFICIENCY: "	14		4		0		5		0	
% DEFICIENCY	93⅓		26⅔		0		33⅓		0	
QUANTITY: 5hrs	4¾		14½		25½		23¾		30	
DEFICIENCY: "	25¼		15½		4½		6¼		0	
% DEFICIENCY	84%		51⅓		15		20%		0	
MINIMUM DEFICIENCY	84%		26⅔		0		20%		0	

CASE XXIV. E.G., female, 26 years of age.

This patient was admitted on 1.7.33 and gave a history of having had a sore throat four days previously; the throat swab contained virulent C.diphtheriae. No obvious diphtheritic lesion was present on admission and general symptoms of Diphtheria were absent. She was dismissed on 5.8.33.

The Water Test on admission was abandoned because of menstruation; tests later showed no deficiency in the excretion of water.

DATE	9.7.33		16.7.33		23.7.33		30.7.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	2½	1026	12	1020	18	1022	6	1030
10 am.	5	1003	5	1010	12	1002	13	1002
11 am.	20	1000	19	1000	18	1000	17	1000
12 noon	8	1002	3	1010	2¼	1002	2	1002
1 pm.	0	-	1)	1010	1½	1020	2	1004
2 pm.	1)	1020	1)		1¼	1024	1	1008
3 pm.	1)		1	1015	0	-	0	-
QUANTITY: 2hrs	25		24		30		30	
DEFICIENCY: "	0		0		0		0	
% DEFICIENCY	0		0		0		0	
QUANTITY: 5hrs	34		29		34¾		35	
DEFICIENCY: "	0		1		0		0	
% DEFICIENCY	0		3⅓		0		0	
MINIMUM DEFICIENCY	0		0		0		0	

CASE XXV. J.S., female, 40 years of age.

This patient was admitted on 5.7.33, the fifth day of illness. The severity of the acute stage was that of Group II. Recovery was uneventful and the patient was dismissed well on 17.8.33.

Throat swabs of this patient did not reveal diphtheroid organisms. The clinical appearances were those of a typical faucial diphtheria.

The Water Test did not reveal deficiency in the excretion of water at any stage.

DATE	11.7.33		18.7.33		25.7.33		1.8.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	5	1020	22	1020	18	1020	28	1026
10 am.	5	1010	10	1002	11	1001	1½	,1002
11 am.	12	1000	18	1000	9	1000	15	1000
12 noon	3	1002	3	1002	6	1003	2½	1003
1 pm.	0	-	1½	1016	1¾	1012	2	1006
2 pm.	0	-	1)	1016	1½	1022	1	1008
3 pm.	4	1018	½)		¾	-	0	-
QUANTITY: 2hrs	17		28		20		16½	
DEFICIENCY: "	0		0		0		0	
% DEFICIENCY	0		0		0		0	
QUANTITY: 5hrs	20		33½		29¼		22	
DEFICIENCY: "	10		0		¾		8	
% DEFICIENCY	33⅓		0		2½		26⅔	
MINIMUM DEFICIENCY	0		0		0		0	

CASE XXVI. M.M., female, 12 years of age.

This patient was admitted on 12.7.33, the second day of illness. The severity of the acute stage was that of Group I. Recovery was not complicated by any of the recognized sequelae of Diphtheria but a septic condition of the hands delayed dismissal till 16.9.33.

The Water Test revealed a normal water excretion throughout.

DATE	14.7.33		21.7.33		28.7.33		4.8.33		12.8.33	
TIME	Q.	S.G.	Q.	S.G..	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	0	1024	8	1034	14	1034	11	1028	5	1022
10 am.	0	-	6	1003	6	1006	0	-	4	1010
11 am.	19	1020	13	1002	16	1000	16	1000	14	1000
12 noon	0	-	0	-	13	1000	7	1000	9	1002
1 pm.	0	-	4	1006	2	1004	1)	1020	3	1008
2 pm.	0	-	1¼)		1)		1½)		0	-
3 pm.	½	-	¾)		1¼)		0	-	0	-
QUANTITY: 2hrs	19		19		22		16		18	
DEFICIENCY: "	0		0		0		0		0	
% DEFICIENCY	0		0		0		0		0	
QUANTITY: 5hrs	19½		24¼		38		25½		30	
DEFICIENCY: "	10½		5¾		0		4½		0	
% DEFICIENCY	35		19%		0		15		0	
MINIMUM DEFICIENCY	0		0		0		0		0	

CASE XXVII. J H., female, 13 years of age.

This patient was admitted on 14.7.33, the fifth day of illness. The severity of the acute stage was that of Group IV. Recovery took place without paralysis and the patient was dismissed well on 5.9.33.

The Water Test showed diminished excretion of water only at the end of the second week.

DATE	15.7.33		22.7.33		29.7.33		5.8.33		12.8.33		19.8.33		26.8.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	12	1038	13	1034	5	1040	11	1031	18	1028	12	1030	14	1028
10 am.	5	1006	4	1004	2	1008	4	1006	6	1020	16	1004	6	1010
11 am.	18	1002	17½	1000	4½	1002	13	1000	14	1004	5	1002	7	1010
12 noon	¾	1010	4½	1002	1½		2	1008	0	-	4	1010	1	1012
1 pm.	1	1020	2	1016	½	1024	1½	1008	6	1014	0	-	1½	
2 pm.	1		1	-	¾		1½	1012	1½	1024	5	1018	1	1024
3 pm.	1½	1024	0	-	0	-	1	-	3	1026	1½	1018	2	
QUANTITY: 2hrs.	23		21½		6½		17		20		21		13	
DEFICIENCY: "	0		0		8½		0		0		0		0	
% DEFICIENCY	0		0		56⅓		0		0		0		0	
QUANTITY: 5hrs.	28½		29		9		21¼		27½		30		16½	
DEFICIENCY: "	1½		1		21		8¼		2½		0		13½	
% DEFICIENCY	5		3⅓		70		27½		8⅓		0		45	
MINIMUM DEFICIENCY	0		0		56⅔		0		0		0		0	

CASE XXVIII. J. McL., female, 44 years of age.

This patient was admitted on 19.7.33, on the fifth day of illness. The severity of the acute stage was that of Group II. Recovery took place normally and the patient was dismissed on 19.8.33.

The Water Test revealed deficiency in the excretion of water on admission and one week later.

DATE	20.7.33		27.7.33		3.8.33		10.8.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	1½	1028	18	1020	10	1024	18	1020
10 am.	0	-	3½	1006	4½	1010	6	1008
11 am.	7½	1012	3¼	1002	18	1000	18	1000
12 noon	0	-	2¼	1012	10	1000	1½	1002
1 pm.	2½	1006	2	1012	1½	1004	2	1010
2 pm.	0	-	1½	1014	0	-	1	1012
3 pm.	0	-	0	-	1	-	1¾	-
QUANTITY: 2hrs	7½		6¾		22½		24	
DEFICIENCY: "	7½		8¼		0		0	
% DEFICIENCY	50		55		0		0	
QUANTITY: 5hrs	10		12½		34		28½	
DEFICIENCY: "	20		17½		0		1½	
% DEFICIENCY	66⅔		58⅓		0		5	
MINIMUM DEFICIENCY	50		55		0		0	

CASE XXIX. A.C., female, 12 years of age.

This patient was admitted on 20.7.33, the fourth day of illness. The severity of the acute stage was that of Group II. The patient recovered without developing paralysis and was dismissed on 31.8.33.

The Water Test revealed deficiency in the excretion of water on admission and again at the end of the second week, but not at the end of the first week or later than the second week.

DATE	21.7.33		28.7.33		4.8.33		11.8.33		18.8.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	14	1026	15	1032	12	1032	10	1040	20	1028
10 am.	0	-	10	1020	0	-	4	1000	4	1012
11 am.	1¾	1020	8	1002	4	1000	12	1000	16	1002
12 noon	0	-	0	-	0	-	10	1008	12	1008
1 pm.	2	1024	4	1010	3	1010	3	1006	3½	1010
2 pm.	1)		0	-	1½	1020	0	-	2½	1024
3 pm.	¾)	1028	2¼	1012	1½	1030	0	-	2½	1020
QUANTITY: 2hrs	1¾		18		4		16		20	
DEFICIENCY: "	13¼		0		11		0		0	
% DEFICIENCY	88⅓		0		73⅓		0		0	
QUANTITY: 5hrs	4¾		22		8½		29		38	
DEFICIENCY: "	24¼		8		21½		1		0	
% DEFICIENCY	84⅙		26⅔		71⅔		3⅓		0	
MINIMUM DEFICIENCY	84⅙		0		71⅔		0		0	

CASE XXX. J. McM., female, 12 years of age.

This patient was admitted on 27.7.33, the fifth day of illness. The severity of the acute stage was that of Group II. Recovery was uncomplicated and the patient was dismissed on 5.9.33.

The Water Test revealed deficiency in the excretion of water on admission and one week later.

DATE	28.7.33		4.8.33		11.8.33		18.8.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	8	1038	10	1030	14	1028	20	1036
10 am.	2	1012	1½	1022	6	1012	4	1014
11 am.	12	1000	13	1002	18	1000	12	1008
12 noon	6	1002	6	1002	6	1002	4	1020
1 pm.	1½	1018	0	-	2	1010	1½	1020
2 pm.	½	1020	3	1020	0	-	½	1028
3 pm.	¼		0	-	0	-	1½	1028
QUANTITY: 2hrs	14		14½		24		16	
DEFICIENCY: "	1		½		0		0	
% DEFICIENCY	6⅔ ⅔		3⅓ ⅓		0		0	
QUANTITY: 5hrs	22		23½		32		23	
DEFICIENCY: "	8		6½		0		7	
% DEFICIENCY	26⅔ ⅔		21⅔ ⅔		0		23⅓ ⅓	
MINIMUM DEFICIENCY	6⅔ ⅔		3⅓ ⅓		0		0	

CASE XXXI. W.P., female, 12 years of age.

This patient was admitted on 8.11.33, the third day of illness. The severity of the acute stage was that of Group II. Albuminuria was present on the first two days in hospital. The patient's recovery was uninterrupted by paralysis and she was dismissed on 27.12.33.

The Water Test revealed deficiency in the excretion of water on admission and one week later.

DATE	10.11.33		17.11.33		24.11.33		1.12.33		8.12.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	11	1022	6	1035	9½	1028	14½	1024	8	1030
10 am.	2¼	1022	3½	1010	11	1006	8	1014	10	1001
11 am.	2¾	1012	5½	1002	12	1002	15	1003	11	1000
12 noon	2½	1011	1¾	1022	3½	1010	3½	1014	7	1000
1 pm.	2	1016	0	-	2¾	1020	2½	1020	4	1002
2 pm.	2	1020	0	-	2	1022	1¼	1022	1	1018
3 pm.	1½	1022	2¾	1026	2⅝	1024	1¾	1024	1	1020
QUANTITY: 2hrs	5		9		23		23		21	
DEFICIENCY: "	10		6		0		0		0	
% DEFICIENCY	66⅔		40		0		0		0	
QUANTITY: 5hrs	11½		10¾		31¼		30¾		33	
DEFICIENCY: "	18½		19¼		0		0		0	
% DEFICIENCY	61⅔		64⅙		0		0		0	
MINIMUM DEFICIENCY	61⅔		40		0		0		0	

CASE XXXII. E.S., female, 10 years of age.

This patient was admitted on 11.11.33, the fifth day of illness. The severity of the acute stage was that of Group II. She made an uneventful recovery and was dismissed on 27.12.33.

The Water Test revealed diminished excretion of water on the third day after admission.

DATE	14.11.33		21.11.33		28.11.33		5.12.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	0	-	5	1021	8	1022	7	1032
10 am.	0	-	12	1002	8½	1010	8½	1028
11 am.	10	1012	4	1010	16	1003	14	1004
12 noon	0	-	1)		0	-	1½	1016
1 pm.	9	1019	1½)	1020	2	1020	1½	1028
2 pm.	0	-	1½	1024	¾)		1)	
3 pm.	0	-	1⅛	1040	1)	1022	1)	1024
QUANTITY: 2hrs	10		16		24½		22½	
DEFICIENCY: "	5		0		0		0	
% DEFICIENCY	33⅓ ⅓		0		0		0	
QUANTITY: 5hrs	19		20		27¼		26½	
DEFICIENCY: "	11		10		2¾		3½	
% DEFICIENCY	36⅔ ⅔		33⅓ ⅓		9⅙		11⅔ ⅔	
MINIMUM DEFICIENCY	33⅓ ⅓		0		0		0	

CASE XXXIII.

A.G., female, 28 years of age.

This patient was admitted on 12.11.33, the fourth day of illness. The severity of the acute stage was that of Group I. The patient recovered without developing paralysis and was dismissed on 23.12.33.

The Water Test showed deficiency in the excretion of water on admission and one week later.

DATE										
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	4	1040	8	1030	10	1032	19	1028	8	1032
10 am.	2	1032	7	1001	7¼	1008	8¾	1009	11	1003
11 am.	5	1020	4½	1002	11	1002	13	1003	9	1001
12 noon	2½	1020	2½	1002	1½	1002	5	1003	5	1002
1 pm.	1½	1026	1½	1024	1½	1022	1)	1030	3	1004
2 pm.	¾)		1½	1028	¾)		1)		2½	1008
3 pm.	1)	1040	1¼	1024	¾)	1036	½	-	¾	-
QUANTITY: 2hrs	7		11½		18¼		21¾		20	
DEFICIENCY: "	8		3½		0		0		0	
% DEFICIENCY	53⅓ ⅓		23⅓ ⅓		0		0		0	
QUANTITY: 5hrs	11¾		17		22		28¾		30½	
DEFICIENCY: "	18¼		13		8		1¼		0	
% DEFICIENCY	60⅝ ⅝		43⅓ ⅓		26⅔ ⅔		4⅙ ⅙		0	
MINIMUM DEFICIENCY	53⅓ ⅓		23⅓ ⅓		0		0		0	

CASE XXXIV. M.B., female, 20 years of age.

This patient was admitted on 5.12.33, the fourth day of illness. The severity of the acute stage was that of Group II. Recovery was uncomplicated and the patient was dismissed well on 5.12.33.

The Water Test revealed deficient excretion of water on admission and one week after admission.

DATE	6.12.33		14.12.33		21.12.33		28.12.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	7	1044	10	1030	11½	1032	9	1026
10 am.	0	-	¼	1012	6	1012	4	1012
11 am.	0	-	12		14	1010	18	1003
12 noon	0	-	1	1024	1	1016	5	1010
1 pm.	4	1040	3		3½		1⅛	1020
2 pm.	0	-	¼	-	1¾	1018	⅞	1025
3 pm.	0	-	¼	-	1¼		⅝	
QUANTITY: 2hrs	0		12¼		20		22	
DEFICIENCY: "	15		2¾		0		0	
% DEFICIENCY	100		18⅓ ⅓		0		0	
QUANTITY: 5hrs	4		16½		26¼		29⅜	
DEFICIENCY: "	26		13½		3¾		⅝	
% DEFICIENCY	86⅔ ⅔		45		12½		2 1/12	
MINIMUM DEFICIENCY	86⅔ ⅔		18⅓ ⅓		0		0	

CASE XXXV. A.J., female, 10 years of age.

This patient was admitted on 6.12.33, the fifth day of illness. The severity of the acute stage was that of Group I. She made an uninterrupted recovery and was dismissed on 16.1.34.

The Water Test revealed deficiency in the excretion of water three days after admission.

DATE	9.12.33		16.12.33		23.12.33		30.12.33	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	6	1032	2½	1044	13	1038	5	1035
10 am.	0		6	1012	11	1014	14	1012
11 am.	2¾	1024	17¾	1006	8	1006	11½	1004
12 noon	1½	1026	9¼	1006	7	1009	3½	1010
1 pm.	1		3¾	1012	4	1010	2	1020
2 pm.	1)	1028	1½)	1020	1¾	1020	1½	1024
3pm.	1)		¾)		1	-	2	1016
QUANTITY: 2hrs	2¾		23¾		19		25½	
DEFICIENCY: "	12¼		0		0		0	
% DEFICIENCY	81⅓		0		0		0	
QUANTITY: 5hrs	6¼		38		31¾		32½	
DEFICIENCY: "	23¾		0		0		0	
% DEFICIENCY	79 1/6		0		0		0	
MINIMUM DEFICIENCY	79 1/6		0		0		0	

CASE XXXVI. G.T., female, 11 years of age.

This patient was admitted on 27.12.33, the fourth day of illness. The severity of the acute stage was that of Group I. She recovered without developing complications and was dismissed well on 5.2.34.

The Water Test revealed diminished excretion of water on admission.

DATE	29.1.33		5.1.34		12.1.34		19.1.34		26.1.34	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	4	1034	6½	1032	8	1030	9½	1030	12	1020
10 am.	1½	1024	6	1014	16	1003	17	1018	9	1008
11 am.	5	1010	19¾	1003	14½	1002	6	1002	12	1003
12 noon	2½	1020	3½	1009	1½	1018	¾)		2	1020
1 pm.	1½	1015	1¼)		1)		6)	1028	½)	
2 pm.	¾)		1)	1036	2)	1024	½	-	1)	1024
3 pm.	1)		1	-	1	-	½	-	½)	
QUANTITY: 2hrs	6½		25¾		30½		23		21	
DEFICIENCY: "	8½		0		0		0		0	
% DEFICIENCY	56⅔		0		0		0		0	
QUANTITY: 5hrs	11¼		31½		35		30¼		24½	
DEFICIENCY: "	18¾		0		0		0		5½	
% DEFICIENCY	62½		0		0		0		18⅓	
MINIMUM DEFICIENCY	56⅔		0		0		0		0	

CASE XXXVII. J.P., female, 15 years of age.

This patient was admitted on 28.12.33, the eighth day of illness. The severity of the acute stage was that of Group II. Albuminuria was present from 29.12.33 to 2.1.34 inclusive and was not associated with menstruation. Recovery was retarded by the development of cardiac paralysis of a mild type on 9.2.34. The patient was dismissed well on 28.2.34.

The Water Test revealed deficiency of excretion of water on admission and a fortnight later but not at the end of the first week in hospital.

DATE	30.12.33		8.1.34		15.1.34		22.1.34		29.1.34	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	1	1026	17	1014	6	1028	8	1040	12	1028
10 am.	11	1025	14	1010	10	1012	6	1014	12	1008
11 am.	1½	1020	3½	1008	0	-	10	1004	10	1002
12 noon	1½	1030	11¼	1020	8	1010	4¼	1010	6	1004
1 pm.	1½	1028	3	1018	0	-	2	1020	1½	1016
2 pm.	1½	1022	1)	1022	4½	1020	1½	1022	1½	1018
3 pm.	1¼	1018	2)		0	-	1	-	1	-
QUANTITY: 2hrs	12½		17½		10		16		22	
DEFICIENCY: "	2½		0		5		0		0	
% DEFICIENCY	16⅔		0		33⅓		0		0	
QUANTITY: 5hrs	17		32¾		22½		23¾		31	
DEFICIENCY: "	13		0		7½		6¼		0	
% DEFICIENCY	43⅓		0		25		20⅚		0	
MINIMUM DEFICIENCY	16⅔		0		25		0		0	

CASE XXXVIII. M. McC., female, 34 years of age.

This patient was admitted on 5.1.34, the second day of illness. The severity of the acute stage was that of Group II. She developed arthritis and colitis in the fourth week of residence but no evidence of paralysis was found. The patient was dismissed on 22.2.34.

The Water Test showed deficiency in the excretion of water on admission and one week later.

DATE	8.1.34		15.1.34		22.1.34		29.1.34		5.2.34	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	11	1026	8	1030	7	1034	3	1034	2½	1034
10 am.	4	1012	4½	1022	4	1024	4	1011	6	1004
11 am.	3¼	1012	2	1022	13	1003	19	1006	15	1000
12 noon	1½)		2	1010	2	1016	3¾	1010	5	1002
1 pm.	1½)	1020	1½	1028	1½	1024	1¼)		2½	1008
2 pm.	2	1020	1½)	1028	1)	1036	½		1¾)	1020
3 pm.	1½	1012	1)		¾)		½	-	¾)	
QUANTITY: 2hrs	7¼		6½		17		23		21	
DEFICIENCY: "	7¾		8½		0		0		0	
% DEFICIENCY	51⅓ ⅔		56 ⅓ ⅓		0		0		0	
QUANTITY: 5hrs	12¼		11½		21½		28½		30¼	
DEFICIENCY: "	17¾		18½		8½		1½		0	
% DEFICIENCY	59 ⅙ ⅙		61⅔ ⅔		28 ⅓ ⅓		5		0	
MINIMUM DEFICIENCY	51 ⅔ ⅔		56 ⅔ ⅔		0		0		0	

CASE XXXIX. M.G., female, 20 years of age.

This patient was admitted on 18.1.34, the fourth day of illness. The severity of the acute stage was that of Group I. Recovery was free from the accompaniment of paralysis and the patient was dismissed on 2.3.34.

The Water Test revealed deficiency in the excretion of water at the end of the first and third weeks in hospital but not on admission or at the end of the second week.

DATE	20.1.34		27.1.34		3.2.34		10.2.34		17.2.34		24.2.34	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	12	1036	7	1030	8	1032	1½	1032	12	1030	11	1032
10 am.	12¼	1024	0	-	4	1022	2	1016	10	1002	12	1002
11 am.	9½	1004	12	1012	13	1003	5½	1010	8	1002	10	1001
12 noon	0	-	0	-	3½	1010	2	1011	6	1003	6	1004
1 pm.	0	-	0	-	3¼	1011	6	1010	1½	1010	1¼	1016
2 pm.	0	-	4	1022	2	1028	2½	1020	1½	1018	1½	1022
3 pm.	0	-	0	-	1	-	1¼	-	1	-	0	-
QUANTITY: 2hrs.	21¾		12		17		7½		18		22	
DEFICIENCY: "	0		3		0		7½		0		0	
% DEFICIENCY	0		20		0		50		0		0	
QUANTITY: 5hrs.	21¾		16		25¾		18		27		30¾	
DEFICIENCY: "	8¼		14		4¼		12		3		0	
% DEFICIENCY	27½		46⅔		14⅙		40		10		0	
MINIMUM DEFICIENCY	0		20		0		40		0		0	

CASE XL. M.M., female, 11 years of age.

This patient was admitted on 22.1.34, the second day of illness. The severity of the acute stage was that of Group II. Recovery was uninterrupted and the patient was dismissed on 23.2.34.

The Water Test revealed deficiency in the excretion of water on admission.

DATE	25.1.34		2.2.34		10.2.34		17.2.34	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	4	1040	8	1025	6	1038	9	1026
10 am.	1½	1040	12	1010	2½	1012	8	1010
11 am.	4	1010	13	1002	13½	1003	20	1010
12 noon	1¾	1018	8½	1010	9½	1004	4	1000
1 pm.	1	1020	1)	1028	1)	1028	½)	
2 pm.	1¼)	1024	½)		1)		0)	1022
3 pm.	1)		1	-	¾	-	1½)	
QUANTITY: 2hrs		5½		25		16		28
DEFICIENCY: "		9½		0		0		0
% DEFICIENCY		63⅓		0		0		0
QUANTITY: 5hrs		9½		35		27½		32½
DERICIENCY: "		20½		0		2½		0
% DEFICIENCY		68⅓		0		8⅓		0
MINIMUM DEFICIENCY		63⅓		0		0		0

CASE XLI. I.S., female, 13 years of age.

This patient was admitted on 13.2.34, the fourteenth day of illness. The severity of the acute stage was that of Group II. The patient's recovery from her attack of Diphtheria was free from incident but she developed Scarlet Fever two days before she would have been dismissed.

The Water Test showed deficiency in water excretion on admission.

DATE	17.2.34		24.2.34		3.3.34		10.3.34	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	6	1028	10	1034	10	1032	2½	1024
10 am.	1½	1036	5	1034	4½	1011	14	1011
11 am.	9	1012	16	1000	13	1006	15	1003
12 noon	0		10	1004	4½	1004	11	1003
1 pm.	0		0	-	0	-	3¾	1012
2 pm.	0		0	-	0	-	1½)	1020
3 pm.	0		3¼	1018	2½	1030	1)	
QUANTITY: 2hrs	10½		21		17½		29	
DEFICIENCY: "	4½		0		0		0	
% DEFICIENCY	30		0		0		0	
QUANTITY: 5hrs	10½		about 33¼ *		about 23½*		45¼	
DEFICIENCY: "	19½		0		6½		0	
% DEFICIENCY	65		0		21⅓		0	
MINIMUM DEFICIENCY	30		0		0		0	

* On the assumption that 1 oz. is excreted in the sixth hour.

CASE XLII. J.G., female, 24 years of age.

This patient was admitted on 24.2.34, the third day of illness. The severity of the acute stage was that of Group II. Recovery was free from incident and the patient was dismissed on 30.3.34.

The Water Test showed deficiency of excretion of water on admission and one week later.

DATE	26.2.34		5.3.34		12.3.34		19.3.34	
TIME	Q.	S.G.	Q.	S.G.	Q.	S.G.	Q.	S.G.
9pm - 9am.	2½	1024	14	1028	8½	1030	8	1026
10 am.	1¼	1026	8	1002	13	1002	16	1004
11 am.	1½	1028	4	1008	19	1000	14	1000
12 noon	1)	1036	1½	1006	2½	1012	4	1010
1 pm.	1)		2	1016	3½	1026	4	1024
2 pm.	1)	1038	1)	1026	1½	1020	2	1020
3 pm.	¾)		1½)		¼	-	0	-
QUANTITY: 2hrs.	2¾		12		32		30	
DEFICIENCY: "	12¼		3		0		0	
% DEFICIENCY	81⅓		20		0		0	
QUANTITY: 5hrs.	5¾		16½		39½		40	
DEFICIENCY: "	24¼		13½		0		0	
% DEFICIENCY	80%		45		0		0	
MINIMUM DEFICIENCY	80%		20		0		0	

"B L O O D S U G A R C U R V E S"

The term "amount of reducing substance excreted in the urine" in the following case-records refers to the period of five hours following the ingestion of the sugar. It has been calculated by diluting the urine a hundred times, or more, with water, and employing Herbert and Bourne's method as used for blood sugar for the test. The term "reducing substance" is used in preference to "sugar" because other substances in the urine, other than sugar, are capable of reducing cupric solutions. The figures have been included only because the excretion of galactose and laevulose in amounts of more than 3 grammes in five hours have been regarded as evidence of hepatic insufficiency by certain clinicians, notably the physicians of the Mayo Clinic. It is seen that there is no significance in the excretions in the present investigation.

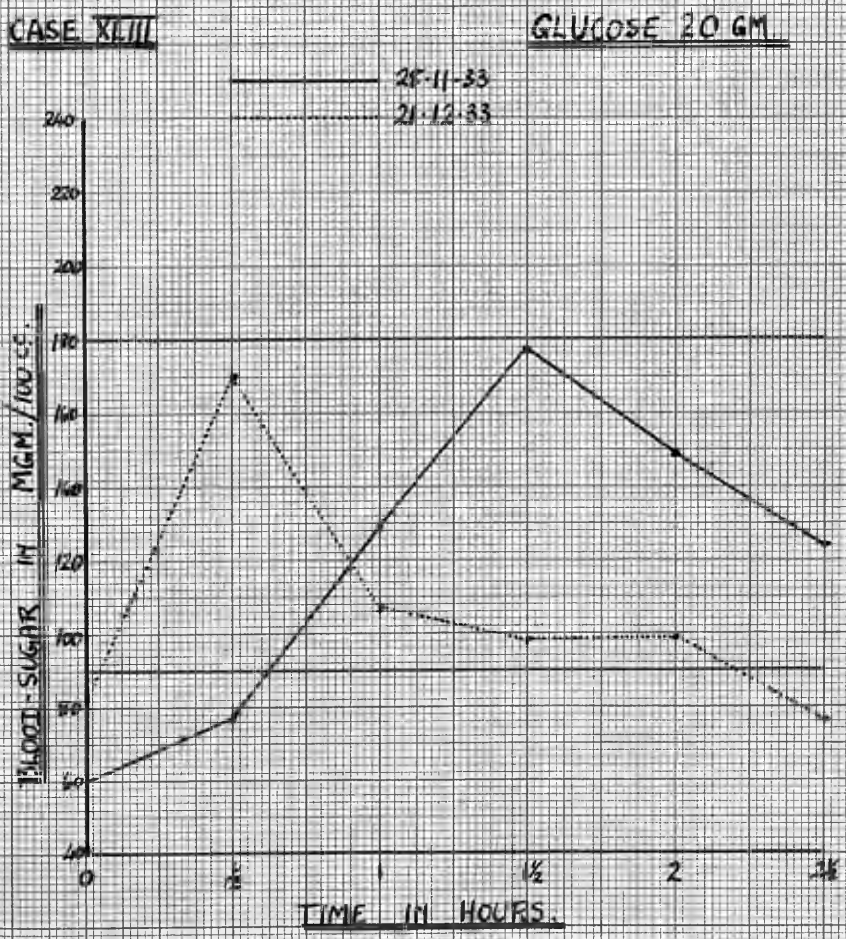
CASE XLIII.

T.R., male, 13 years of age. GLUCOSE.

This patient was admitted on 26.11.33, the fifth day of illness. The severity of the acute stage was that of Group I. He recovered without developing paralysis and was dismissed on 5.1.34.

A blood sugar curve on 28.11.33 showed initial hypoglycaemia and had the form of a lag curve. On 21.12.33 the fasting blood sugar was within normal limits and the curve was of normal form.

The amount of reducing substance excreted in the urine on 28.11.33 was 0.085 gm. and on 21.12.33, 0.116 gm.


CASE XLIII
GLUCOSE 20 GM
25-11-33
21-12-33
240
220
200
180
160
140
120
100
80
60
40
BLOOD-SUGAR IN MGM./100 C.C.
0
½
1
1½
2
2½
TIME IN HOURS.

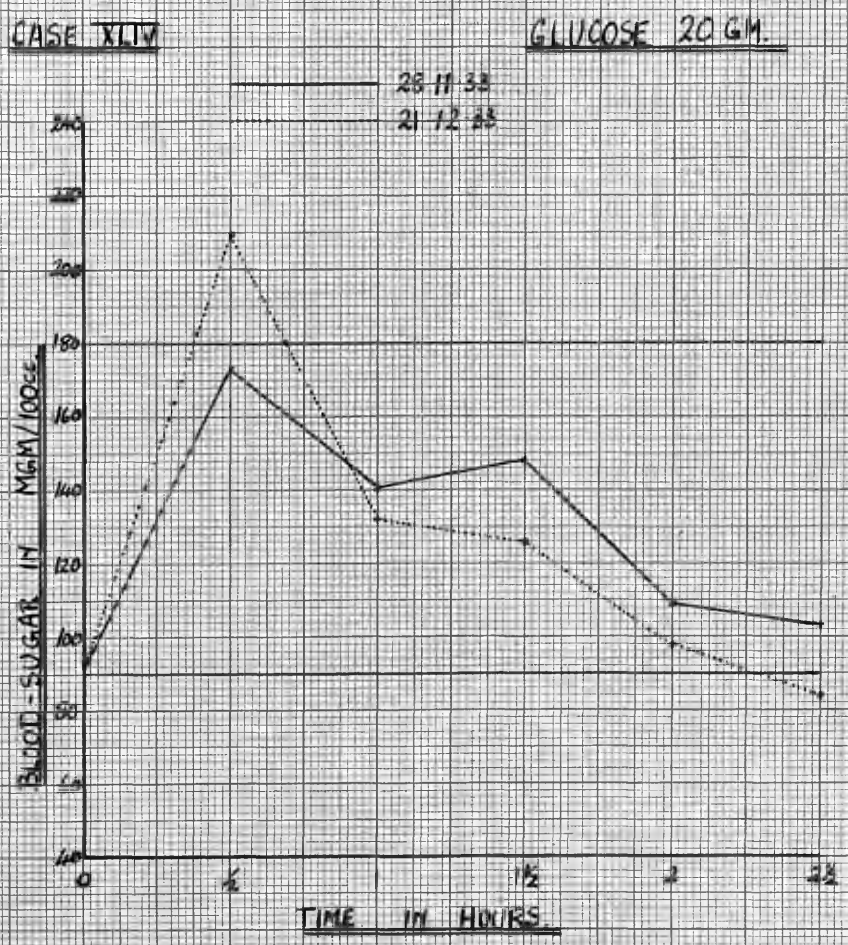
CASE XLIV.

S.B., male, 22 years of age. GLUCOSE.

This patient was admitted on 26.11.33, the third day of illness. The severity of the acute stage was that of Group I. Recovery took place without complications and the patient was dismissed on 29.12.33.

A blood sugar curve on 28.11.33 showed a normal fasting blood sugar and a normal rise after half-an-hour; the fall of the blood sugar was delayed however. On 21.12.33, the fasting blood sugar was normal and the form of the curve was approaching normal; the amount of the blood sugar after half-an-hour was unduly high.

The amount of reducing substance excreted in the urine on 28.11.33 was 0.080 gm. and on 21.12.33, 0.163 gm.


CASE XLV
GLUCOSE 20 GM.
28 11 33
21 12 28
240
220
200
180
160
140
120
100
80
60
40
BLOOD-SUGAR IN MGM/100CC
0
½
1½
2
2½
TIME IN HOURS

CASE XLV.

A.N., male, 11 years of age. GLUCOSE.

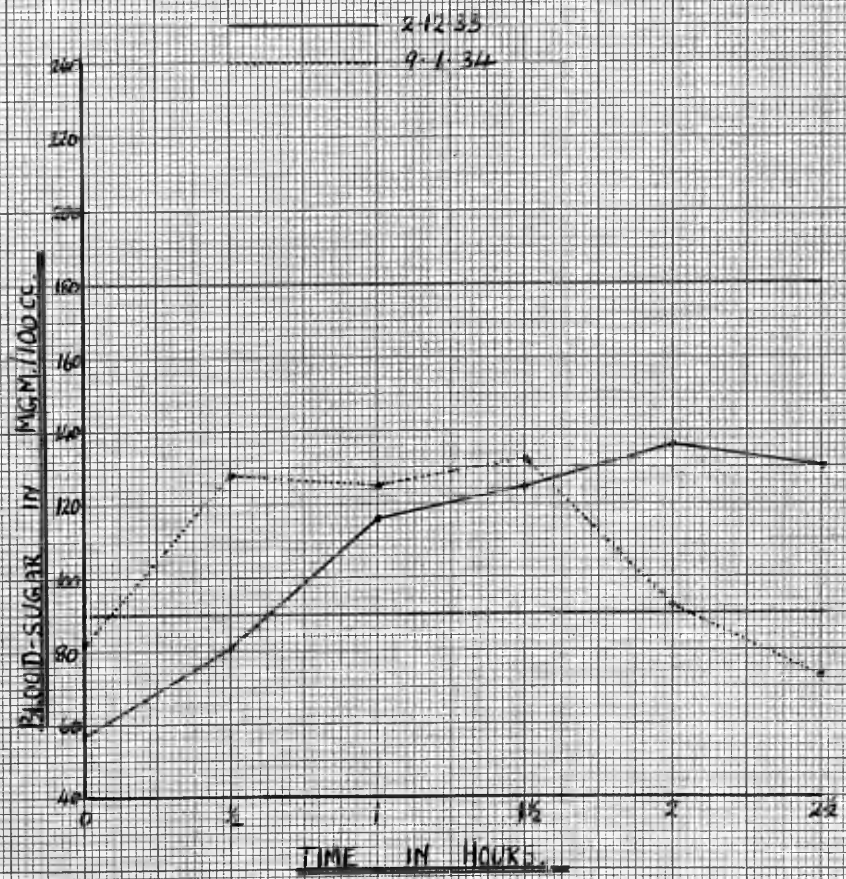
This patient was admitted on 1.12.33, the eighth day of illness. The severity of the acute stage was that of Group II. Recovery was unaccompanied by paralysis but dismissal was delayed till 2.2.34 because of the persistence of positive throat cultures.

A blood sugar curve on 2.12.33 showed fasting hypoglycaemia and had a lag; on 9.1.34 the fasting blood sugar was within normal limits, but the curve still had a lag form although the blood sugar was back to normal within two hours.

The amount of reducing substance excreted in the urine on 2.12.33 was 0.097 gm., and on 9.1.34, 0.044 gm.

CASE XLV

GLUCOSE 20 GM.



CASE XLVI.

D.D., male, 12 years of age. GLUCOSE.

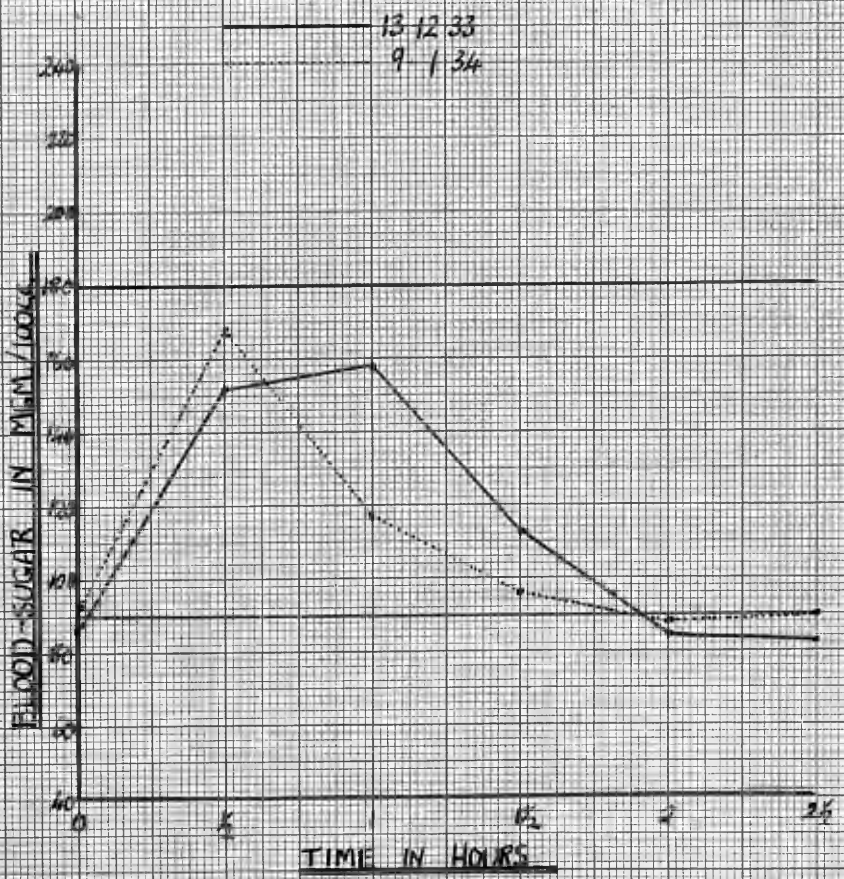
This patient was admitted on 12.12.33, the fifth day of illness. The severity of the acute stage was that of Group I. Recovery took place without incident and the patient was dismissed on 20.1.34.

A blood sugar curve on 13.12.33 had its peak after one hour and the return to normal was slightly delayed - the fasting blood sugar was within normal limits. On 9.1.34 the curve was normal.

The amount of reducing substance in the urine was 0.079 gm. on 13.12.33 and 0.058 gm. on 9.1.34.

CASE XLVI

GLUCOSE 20 GM



CASE XLVII.

W.H., male, 18 years of age. GLUCOSE.

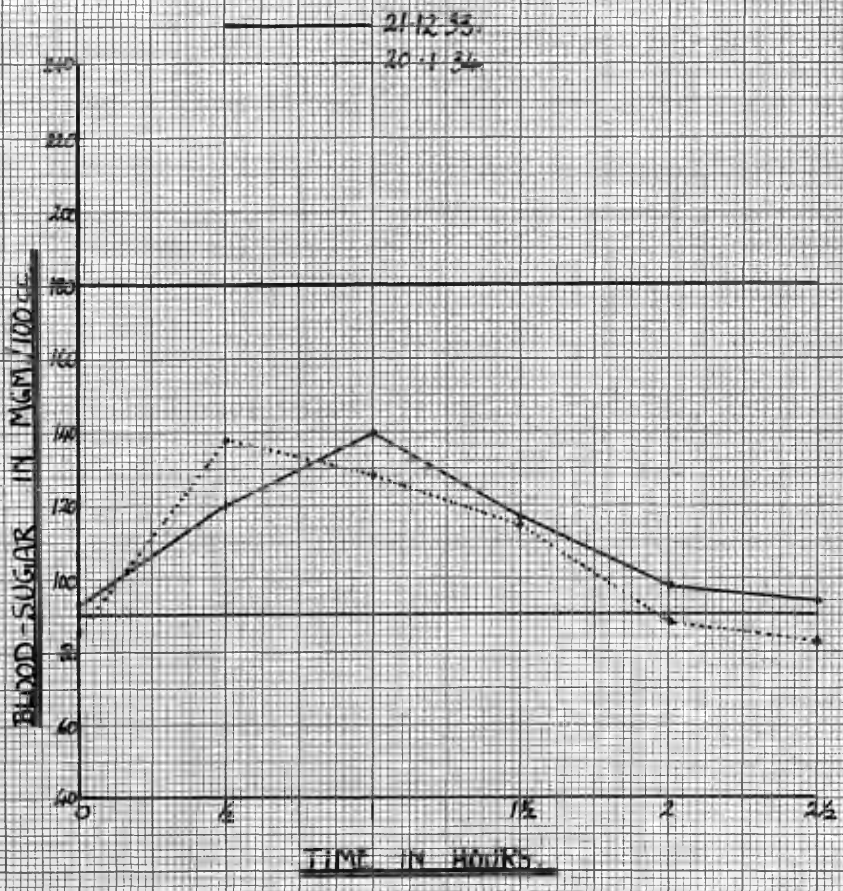
This patient was admitted on 19.12.33, the second day of illness. The severity of the acute stage was that of Group II. The patient's recovery was normal and he was dismissed on 29.1.34.

A blood sugar curve on 21.12.33 showed a normal fasting blood sugar; the peak of the curve was at one hour and the return to normal was delayed. On 20.1.34 the fasting blood sugar was lower but still within normal limits, the peak of the curve was at half-an-hour and the return to normal was slightly delayed.

The amount of reducing substance in the urine on 21.12.33 was 0.287 gm. and on 20.1.34 was 0.144 gm.

CASE XLVII

GLUCOSE 20 GM.



CASE XLVIII.

A.F., male, 16 years of age. GLUCOSE.

This patient was admitted on 25.12.33, the third day of illness. He had previously been under treatment for Chronic Nephritis of azotaemic nature and was dismissed to a general hospital on 22.1.34. The severity of the acute stage of his Diphtheria was that of Group I.

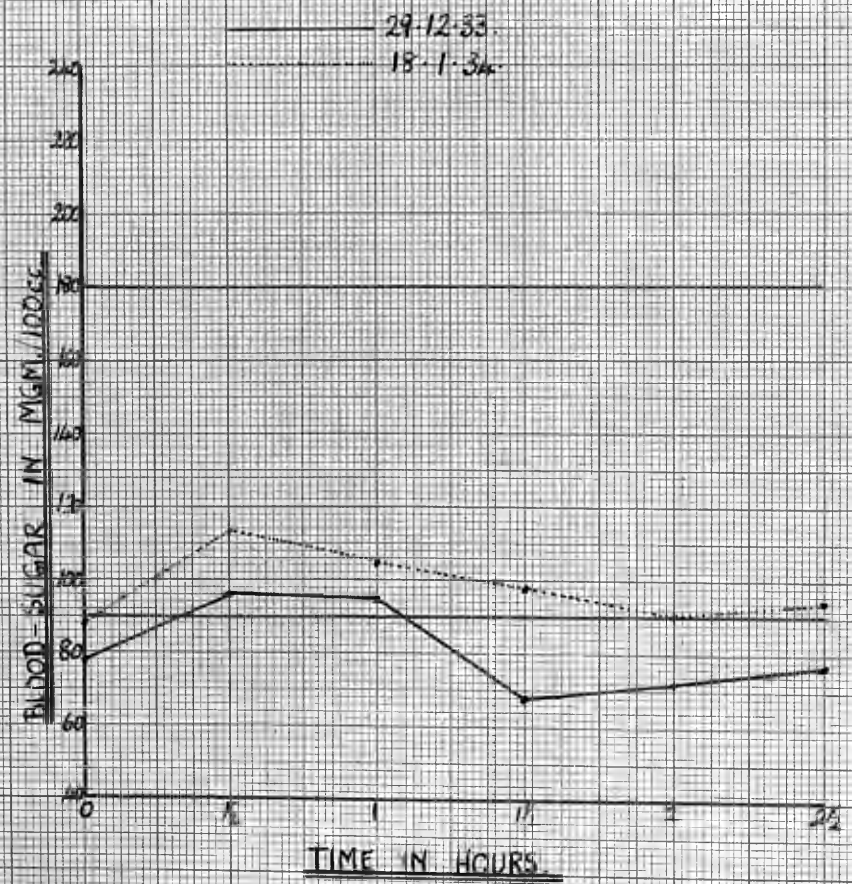
A blood sugar curve on 29.12.33 showed a fasting blood sugar which was slightly below normal; there was a very small rise in the ensuing hour. On 18.1.34, the fasting blood sugar had returned to normal and the peak of the curve, although not high, was after half-an-hour.

The amount of reducing substance excreted in the urine on 29.12.33 was 0.187 gm. and, on 18.1.34 it was 0.127 gm.

The blood urea, which was tested in samples of blood from the pricked finger, was 127 and 84 mgm. per 100 cc. on 25.12.33 and 18.1.34, respectively.

CASE XLVIII

GLUCOSE 20GM.



CASE XLIX.

H.J., male, 22 years of age. GLUCOSE.

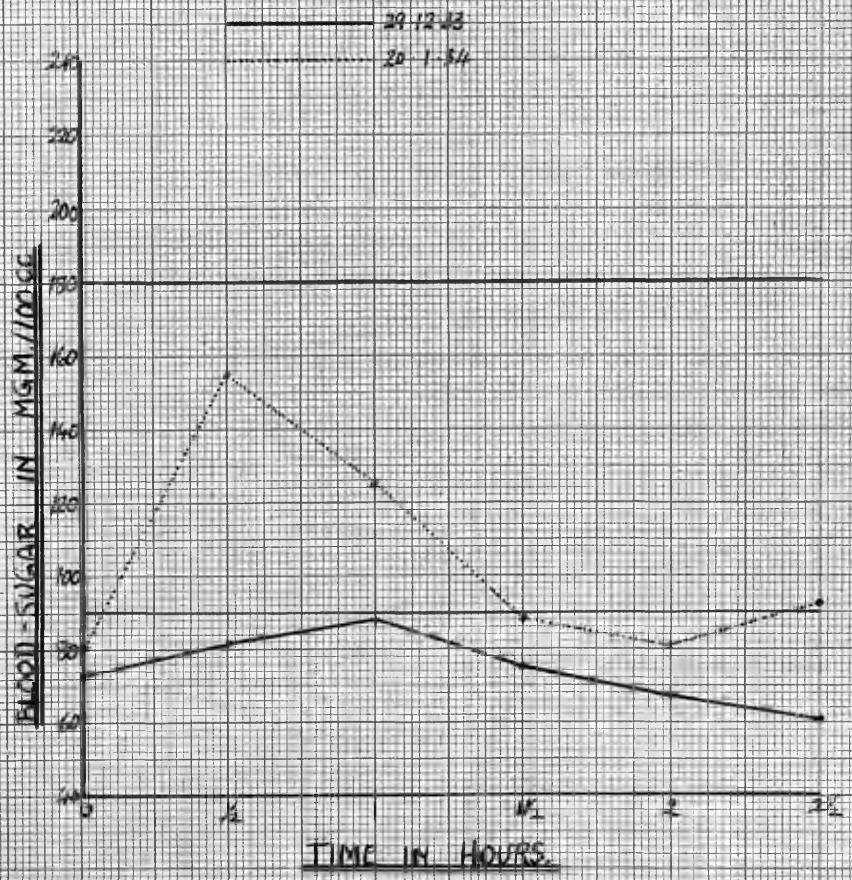
This patient was admitted on 24.12.33, the second day of illness. The severity of the acute stage was that of Group II. He made an uncomplicated recovery and was dismissed on 6.2.34.

A blood sugar curve on 29.12.33 showed a hypoglycaemic fasting state and a very flat shape which failed to rise above normal; on 20.1.34 the fasting blood sugar had risen within normal limits and the form of the curve was practically normal.

The amount of reducing substance excreted in the urine on 29.12.33 was 0.261 gm. and on 20.1.34 was 0.112gm.

CASE XLIX

GLUCOSE 20GM



CASE L.

R.B., male, 32 years of age.

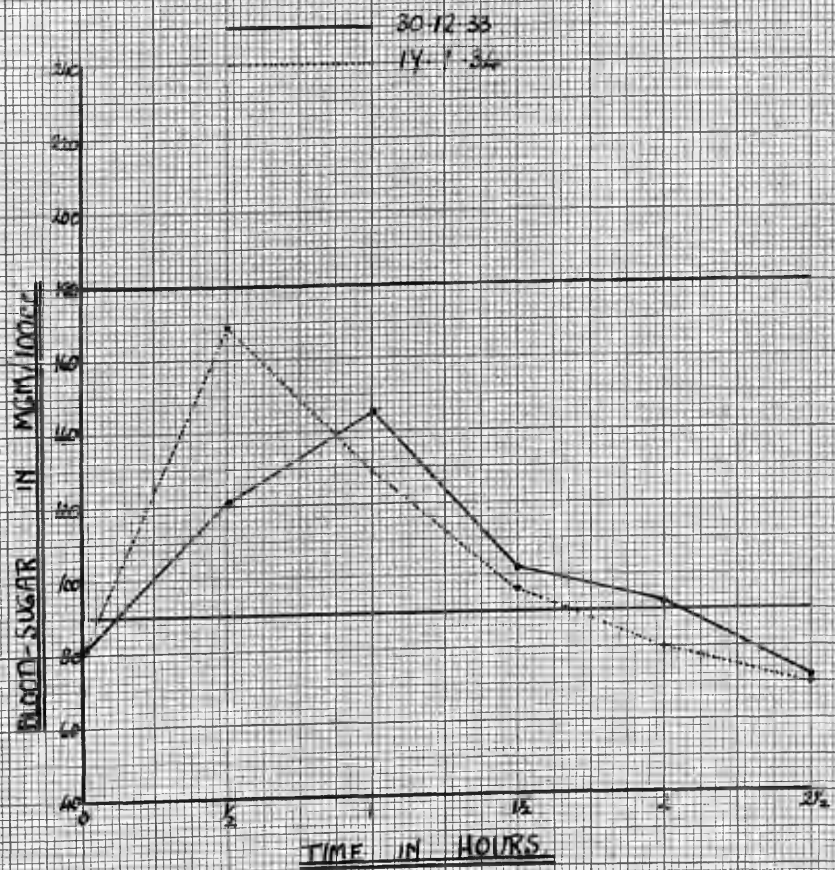
This patient was admitted on 26.12.33. He appeared to be in good health, but a culture made from a swab of his throat had been found to contain C.diphtheriae. He was dismissed, no longer a diphtheria carrier, on 18.1.34.

A blood sugar curve on 30.12.33 was abnormal only in that the peak was delayed to one hour; on 17.1.34, the curve was normal.

The amount of reducing substance excreted in the urine on 30.12.33 was 0.096 gm. and, on 17.1.34, it was 0.106 gm.

CASE I.

GLUCOSE 20GM.



CASE LI.

J.G., male, 50 years of age. GLUCOSE.

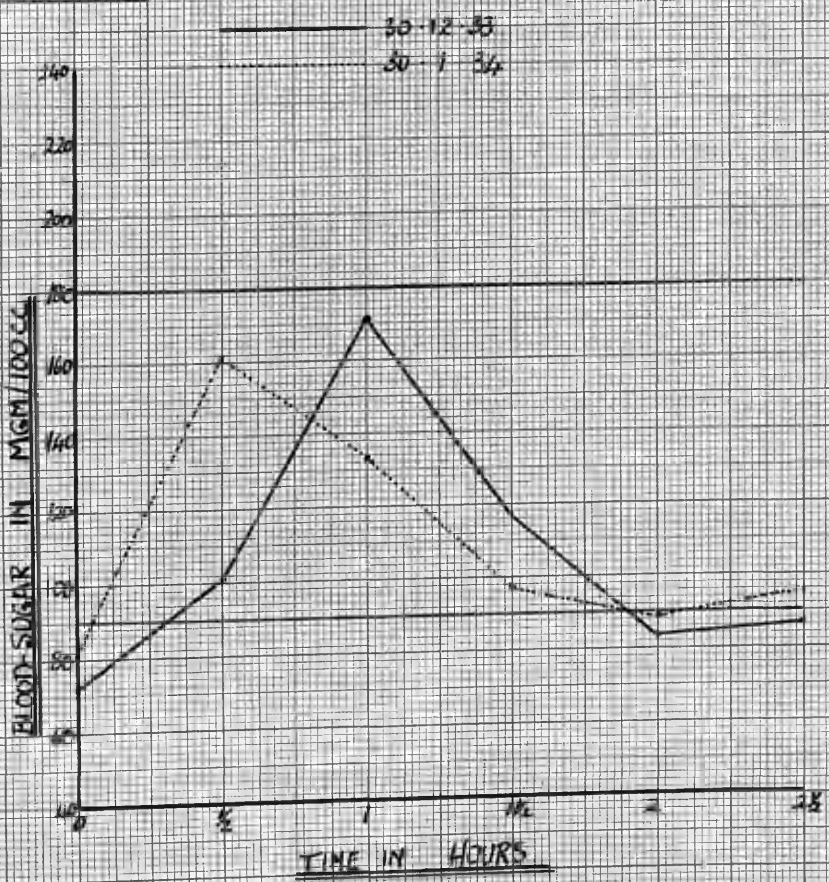
This patient was admitted on 26.12.33, the fifth day of illness. The severity of the acute stage was that of Group I. No complications occurred and the patient was dismissed on 6.2.34.

A blood sugar curve on 30.12.33 showed a fasting hypoglycaemia and delay of the peak till one hour; on 30.1.34 the fasting blood sugar was normal and the shape of the curve was practically normal.

The amount of reducing substance in the urine on 30.12.33 was 0.136 gm. and on 30.1.34 it was 0.296 gm.

CASE II.

GLUCOSE 20GM.



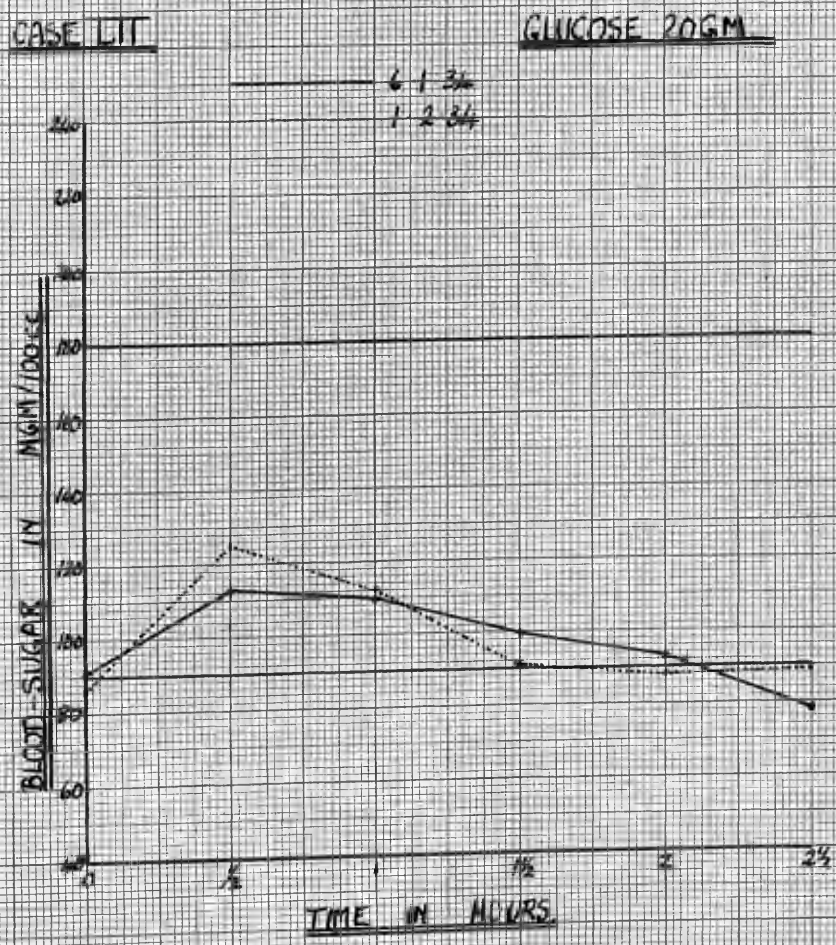
CASE LII.

F. McK., male, 11 years of age. GLUCOSE.

This patient was admitted to hospital with a condition which was later diagnosed as Influenza, on 29.12.33. While convalescent from this, he developed Diphtheria on 4.1.34 and came under treatment on 5.1.34, the second day of illness. The severity of the acute stage was that of Group II. Recovery was uncomplicated but dismissal was delayed by the continuation of positive cultures.

A blood sugar curve on 6.1.34 showed a normal fasting blood sugar and a form of low amplitude; on 1.2.34, the curve was similar but the blood sugar at half-an-hour reached a slightly higher level.

The amount of reducing substance excreted in the urine on 6.1.34 was 0.050 gm. and, on 1.2.34, it was 0.081 gm.


CASE LTT
GLUCOSE 20GM
4 1 3½
1 2 84
BLOOD-SUGAR IN MGM%/100CC
TIME IN HOURS

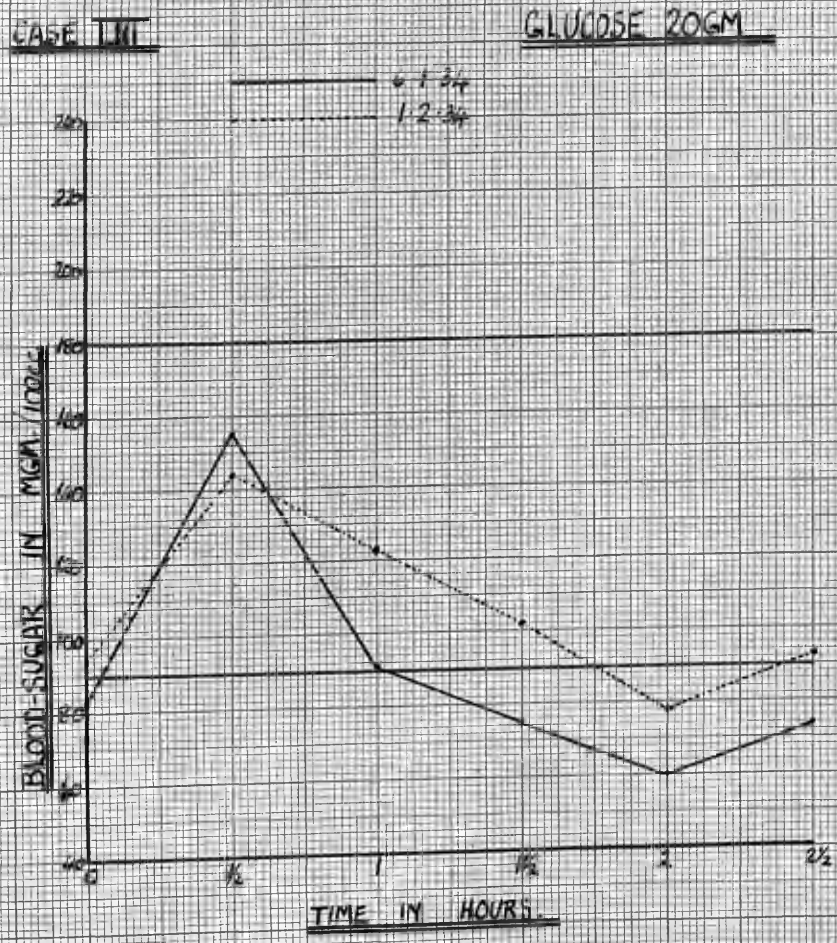
CASE LIII.

D.L., male, 34 years of age. GLUCOSE.

This patient was admitted on 3.1.34, the third day of illness. The severity of the acute stage was that of Group I. He recovered without developing paralysis and was dismissed on 11.2.34.

The blood sugar curve was normal on admission. On 1.2.34 the return of the blood sugar level to normal was somewhat delayed.

The amount of reducing substance excreted in the urine on 6.1.34 was 0.090 gm. and on 1.2.34 was 0.261 gm.


CASE I.M.
GLUCOSE 20GM
BLOOD-SUGAR IN MGM./100cc.
TIME IN HOURS.

CASE LIV.

J.M., male, 30 years of age. GLUCOSE.

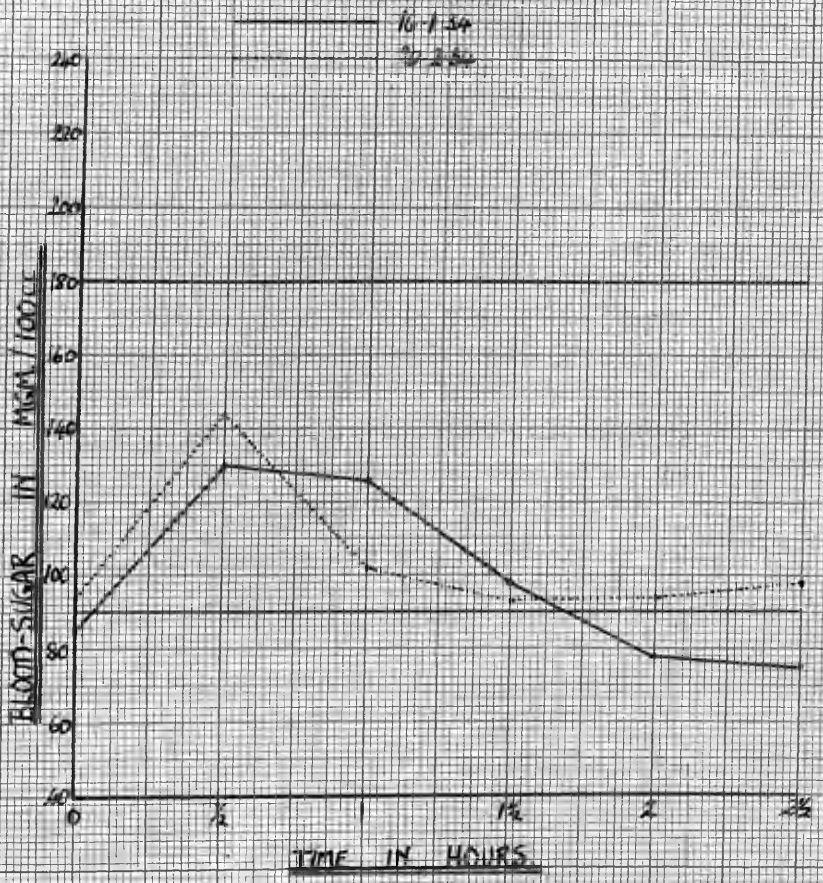
This patient was admitted on 14.1.34, the fourth day of illness. The severity of the acute stage was that of Group II. Recovery was normal and the patient was dismissed on 24.2.34.

A blood sugar curve on 16.1.34 showed a fasting blood sugar which was within normal limits but a slight tendency to delay in the fall of the sugar level to normal. The curve was normal on 20.2.34.

The amount of reducing substance excreted in the urine on 16.1.34 was 0.340 gm. and, on 20.2.34, it was 0.655 gm.

CASE IV

GLUCOSE 20GM.



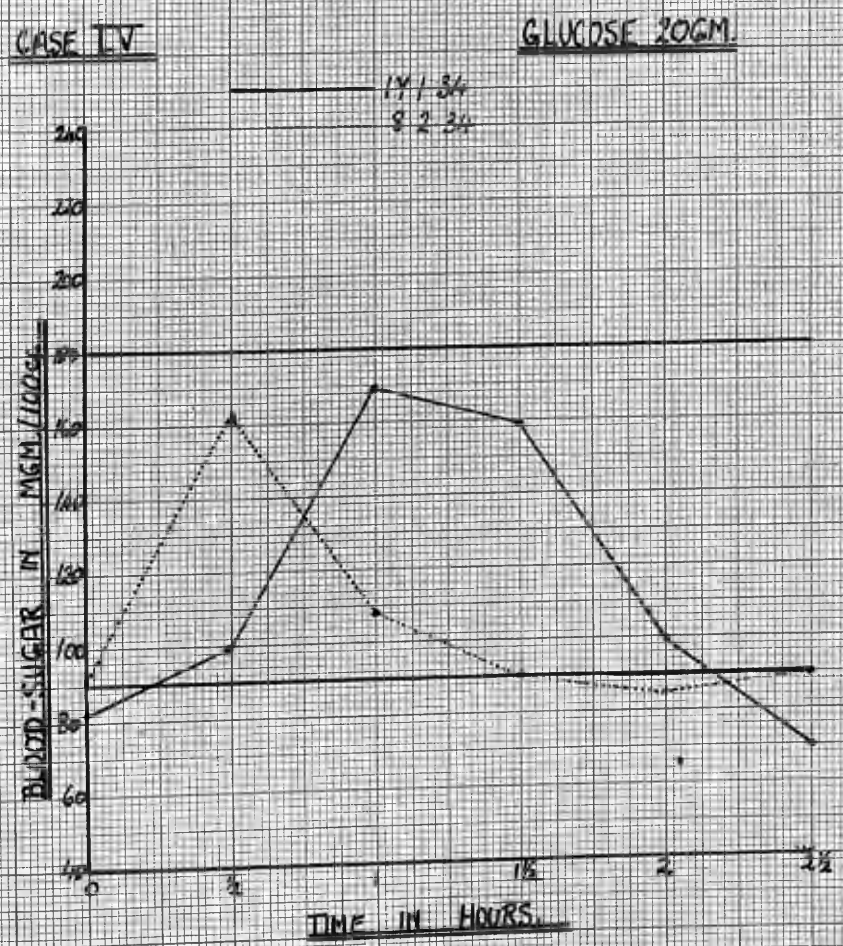
CASE LV.

J.L., male, 14 years of age. GLUCOSE.

This patient was admitted on 16.1.34, the fourth day of illness. The severity of the acute stage was that of Group II. Slight albuminuria was present from admission till 1.2.34. Recovery took place without the development of paralysis. The patient was dismissed well on 2.3.34.

A blood sugar curve on 17.1.34 showed a normal fasting blood sugar but there was a tendency in the shape of the curve to approximate to a lag curve; on 8.2.34 the blood sugar curve was normal.

The amount of reducing substance excreted in the urine on 17.1.34 was 0.069 gm. and on 8.2.34 it was 0.207 gm.


CASE IV
GLUCOSE 20GM.
1¼ 1 3/4
2 2 3/4
BLOOD-SUGAR IN MGM./100c.c.
200
180
160
140
120
100
80
60
40
0
½
1
1½
2
2½
TIME IN HOURS.

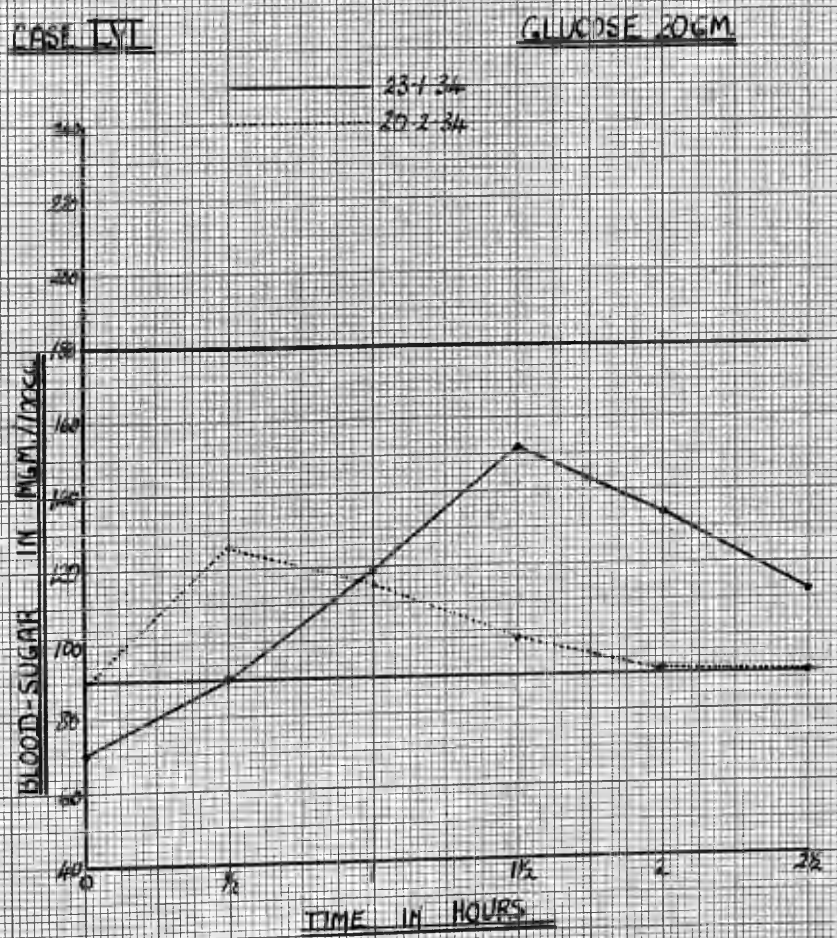
CASE LVI.

A.H., male, 13 years of age. GLUCOSE.

This patient was admitted on 20.1.34, the second day of illnesss. The severity of the acute stage was that of Group II. The patient's recovery was interrupted by the development of acute tonsillitis on 1.2.34 but otherwise there were no complications.

A blood sugar curve on 23.1.34 showed a fasting hypoglycaemia and was definitely of the lag type. The blood sugar curve was approaching normal on 20.2.34.

The amount of reducing substance excreted in the urine was 0.102 gm. on 23.1.34 and 0.274 gm. on 20.2.34.


CASE TST
GLUCOSE 20GM.
23-1-34
20-2-34
BLOOD-SUGAR IN MGM.//100CC.
TIME IN HOURS
0
½
1½
2
2½

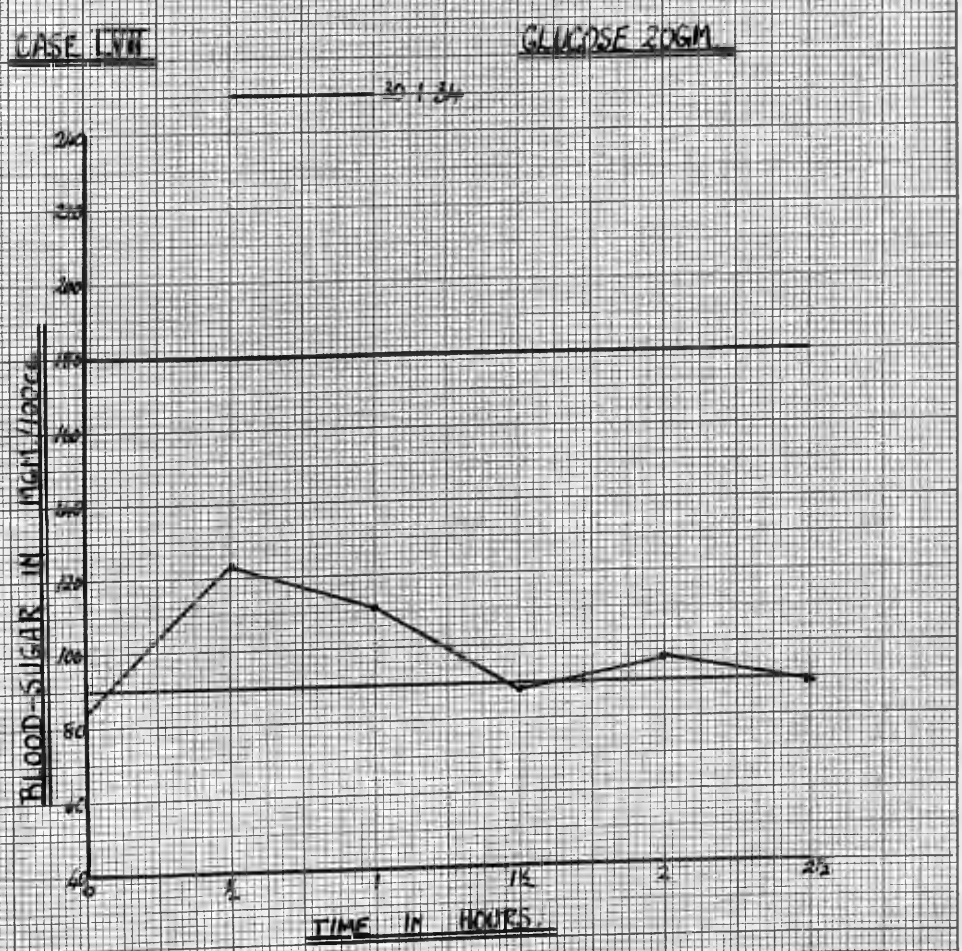
CASE LVII.

J.N., male, 30 years of age. GLUCOSE.

This patient was admitted on 27.1.34 and was dismissed on 7.2.34. He was a contact of several cases of severe Diphtheria and was found to have a positive throat swab. He gave a history of having had a sore throat one week before admission. No clinical evidence of Diphtheria was present.

The blood sugar curve on 30.1.34 showed a slight tendency to delay in the return to normal of the sugar level.

The amount of reducing substance excreted in the urine on 30.1.34 was 0.294 gm.


CASE LNM
GLUCOSE 20GM
30 1 34
BLOOD-SUGAR IN MGM/100CC
TIME IN HOURS

CASE LVIII.

J. McM., male, 13 years of age. GLUCOSE.

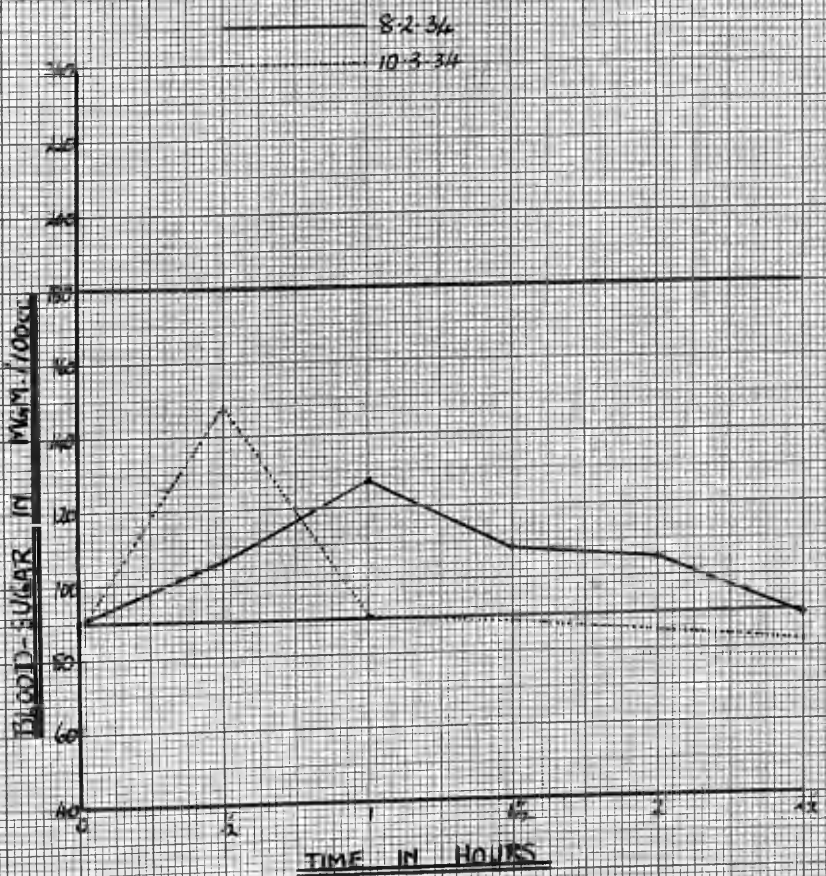
This patient was admitted on 7.2.34, the fourth day of illness. The severity of the acute stage was that of Group II. He made a normal recovery and was dismissed on 19.3.34.

A blood sugar curve on 8.2.34 showed a normal fasting blood sugar but the form of the curve was definitely suggestive of the lag type. On 10.3.34 the blood sugar curve was normal.

The amount of reducing substance excreted in the urine was 0.062 gm. on 8.2.34 and 0.185 gm. on 10.3.34.

CASE LVIII

GLUCOSE 20 GM.



CASE LIX.

J.P., female, 10 years of age. GALACTOSE.

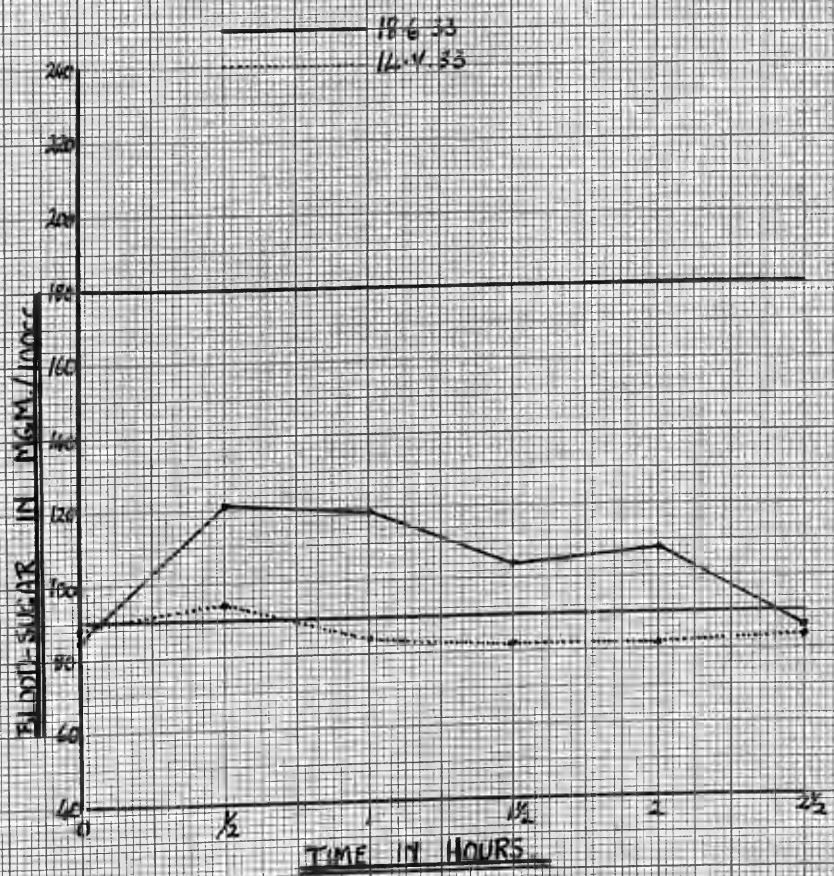
This patient was admitted on 17.6.33, the third day of illness. The severity of the acute stage was that of Group I. Recovery was normal and dismissal took place on 29.7.33.

A blood sugar curve on 18.6.33 showed a normal fasting blood sugar; the range of the blood sugar in the subsequent curve was similar to the lag curve in several cases in which glucose was the test sugar. On 14.7.33, the blood sugar curve was normal.

The amount of reducing substance in the urine was not estimated. Fehling's solution for the testing of the qualitative presence of sugar in urine was not reduced on either occasion.

CASE LIX

GALACTOSE 20GM



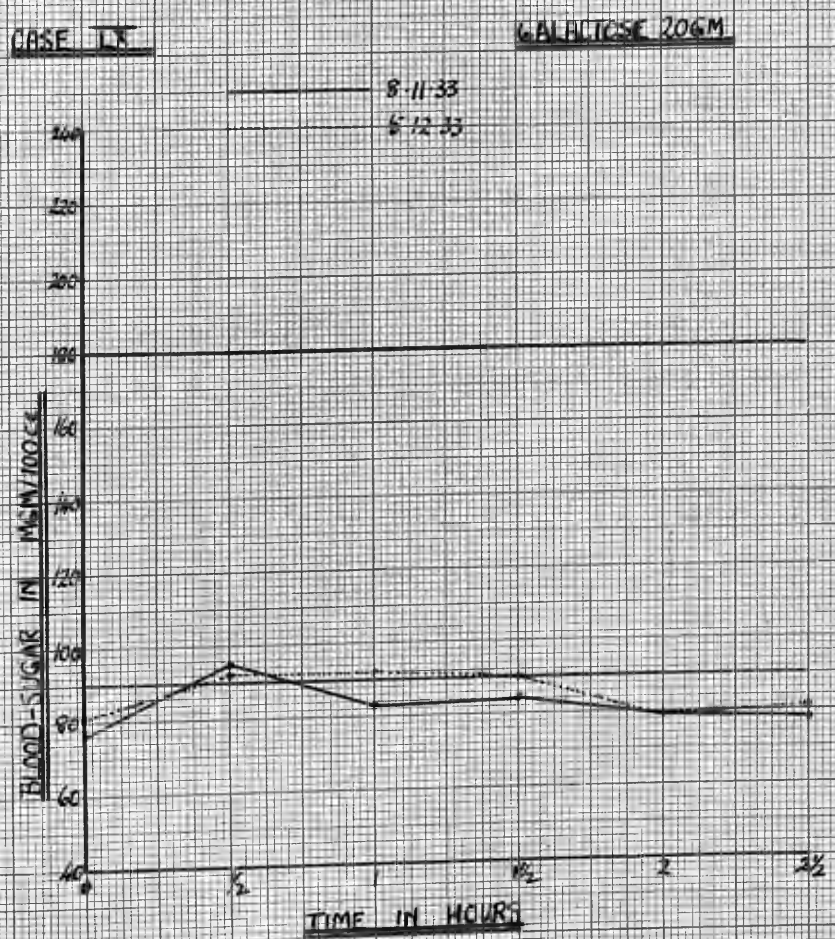
CASE LX.

J.B., female, 12 years of age. GALACTOSE.

This patient was admitted on 7.11.33, the fourth day of illness. The severity of the acute stage was that of Group I. Between 23.12.33 and 27.12.33 the patient had numerous extra-systoles. She was dismissed well on 2.1.34.

Blood sugar curves on 8.11.33 and 5.12.33 were normal with the exception that on the former occasion the fasting blood sugar was subnormal.

The amount of reducing substance excreted in the urine was 0.289 gm. on 8.11.33 and 0.273 gm. on 5.12.33.


CASE IX
GALACTOSE 20GM
8-11-33
6-12-33
BLOOD-SUGAR IN MGM/100 C.C.
TIME IN HOURS

CASE LXI.

E.R., female, 14 years of age. GALACTOSE.

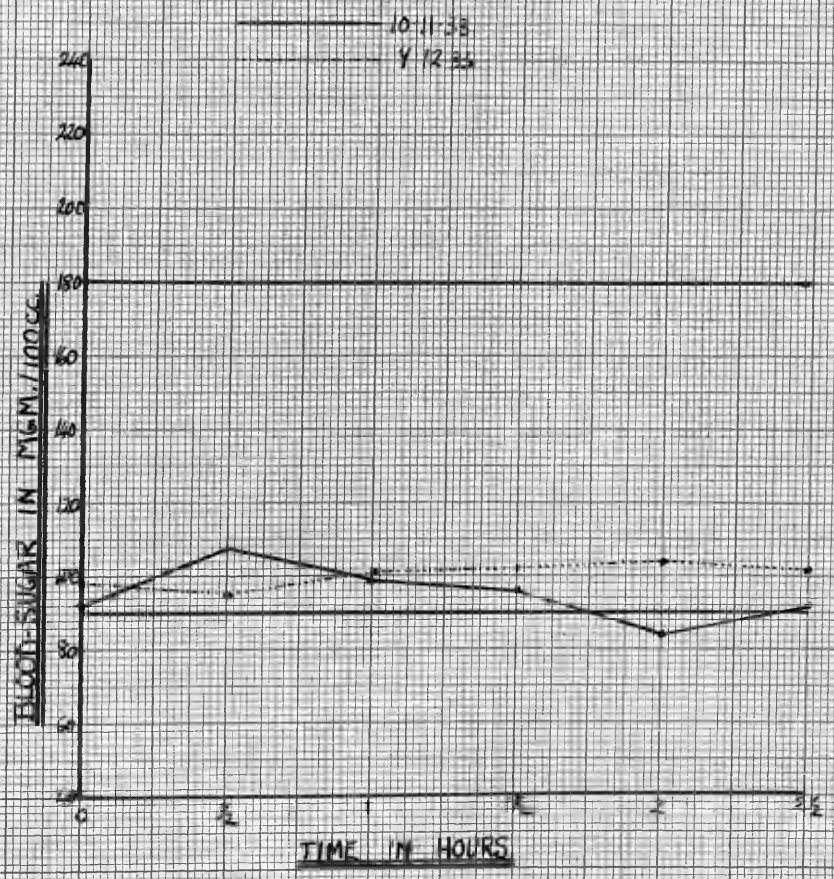
This patient was admitted on 9.11.33, the second day of illness. The severity of the acute stage was that of Group II. Mild albuminuria was present from 27.11.33 to 5.12.33. Recovery was normal. The patient was dismissed on 21.12.33.

Blood sugar curves on 10.11.33 and 7.12.33 were normal.

The amount of reducing substance excreted in the urine was 0.242 gm. on 10.11.33 and 0.264 gm. on 7.12.33.

CASE TEST

SALBUTOSE 20GM



CASE LXII.

M.D., female, 11 years of age. GALACTOSE.

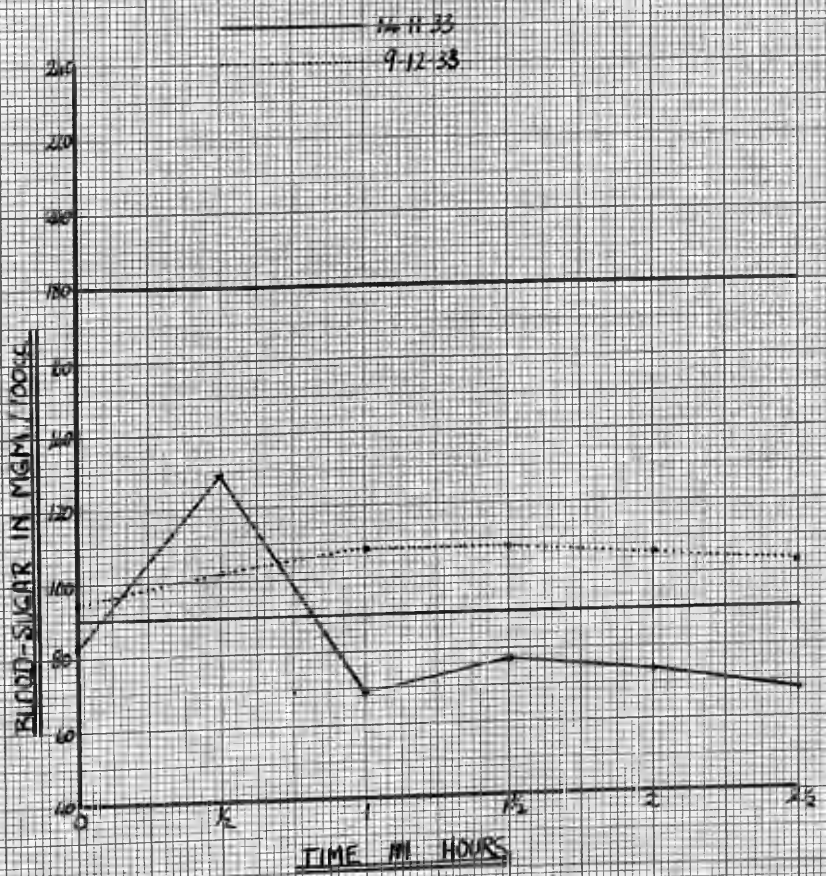
This patient was admitted on 11.11.33, the fifth day of illness. The severity of the acute stage was that of Group I. Recovery was normal and dismissal took place on 27.12.33.

A blood sugar curve on 14.11.33 was similar in shape to that of a normal curve following glucose. On 9.12.33 the curve was normal in shape but the level of the blood sugar was rather high.

The amount of reducing substance excreted in the urine was 0.168 gm. on 14.11.33 and 0.094 gm. on 9.12.33.

CASE EXIT

GALACTOSE 20GM



CASE LXIII.

M.A., female, 19 years of age. GALACTOSE.

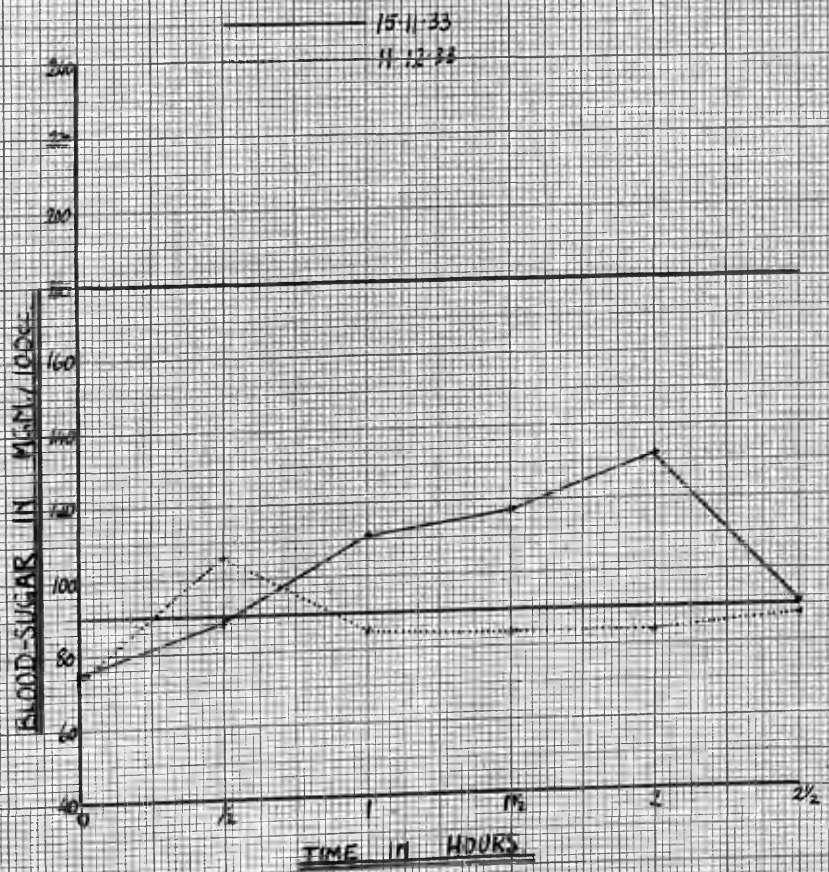
This patient was admitted on 14.11.33, the third day of illness. The severity of the acute stage was that of Group I. The patient developed acute tonsillitis on 24.12.33. She was dismissed on 11.1.34, having recovered without paralysis.

The blood sugar curve on admission showed a fasting hypoglycaemia and the shape of the curve was that of a lag curve. On 11.12.33 hypoglycaemia was still present in the fasting state but the curve was practically normal in shape.

The amount of reducing substance excreted in the urine was 0.747 gm. on 15.11.33 and 0.692 gm. on 11.12.33.

CASE LXII

GALACTOSE 20GM.



CASE LXIV.

M. McK., female, 12 years of age. LAEVULOSE.

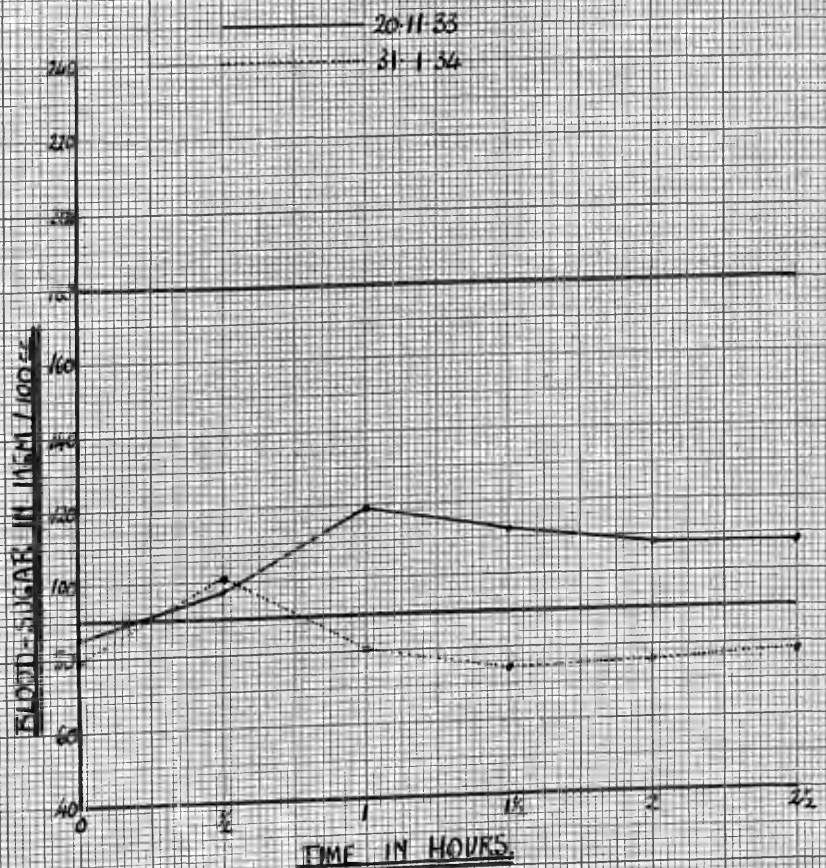
This patient was admitted on 17.11.33, the second day of illness. The severity of the acute stage was that of Group IV. There was mild cardiac paralysis from 22.11.33 till 18.1.34, and the legs at the walking stage were very stiff. She was dismissed on 3.3.34.

A blood sugar curve on 20.11.33 showed a normal fasting blood sugar; the curve had a lag shape. On 31.1.34 the curve was normal.

The amount of reducing substance present in the urine was 0.566 gm. on 20.11.33 and 0.688 gm. on 31.1.34.

CASE LXIV

LAEVULOSE 20 GM



CASE LXV.

E.K., female, 11 years of age. LAEVULOSE.

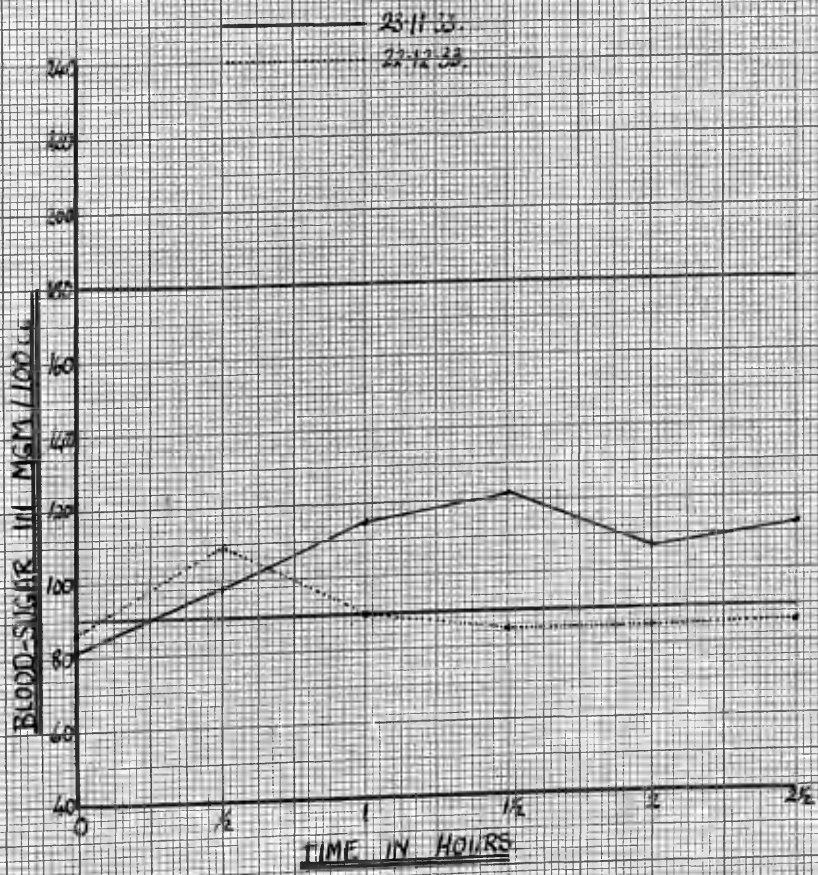
This patient was admitted on 22.11.33, the third day of illness. The severity of the acute stage was that of Group I. Dismissal was delayed till 3.2.34 by the presence of C.diphtheriae in nasal swabs.

A blood sugar curve on 23.11.33 had a lag shape; the fasting blood sugar was normal. On 22.12.33, the blood sugar curve was practically normal.

The amount of reducing substance excreted in the urine was 0.094 gm. on 23.11.33 and 0.054 gm. on 22.12.33.

CASE LXV.

LAEVULOSE 20GM.



CASE LXVI.

M.N., female, 14 years of age. LAEVULOSE.

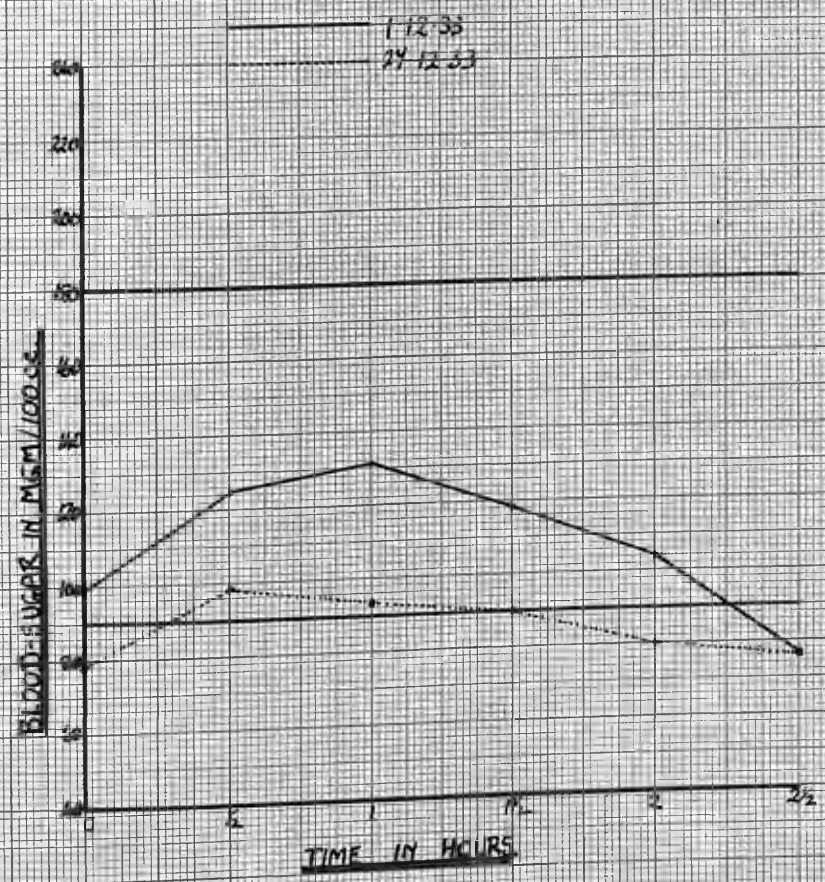
This patient was admitted on 30.11.33, the ninth day of illness. The severity of the acute stage was that of Group I. Recovery was normal and dismissal took place on 16.1.34.

A blood sugar curve on 1.12.33 was definitely of lag type and the fasting blood sugar, although within normal limits was rather high. On 27.12.33 the curve was normal.

The amount of reducing substance excreted in the urine was 0.085 gm. on 1.12.33 and 0.122 gm. on 27.12.33.

CASE LXVI

LAEVULOSE 20GM



CASE LXVII.

N.M., female, 25 years of age. LAEVULOSE.

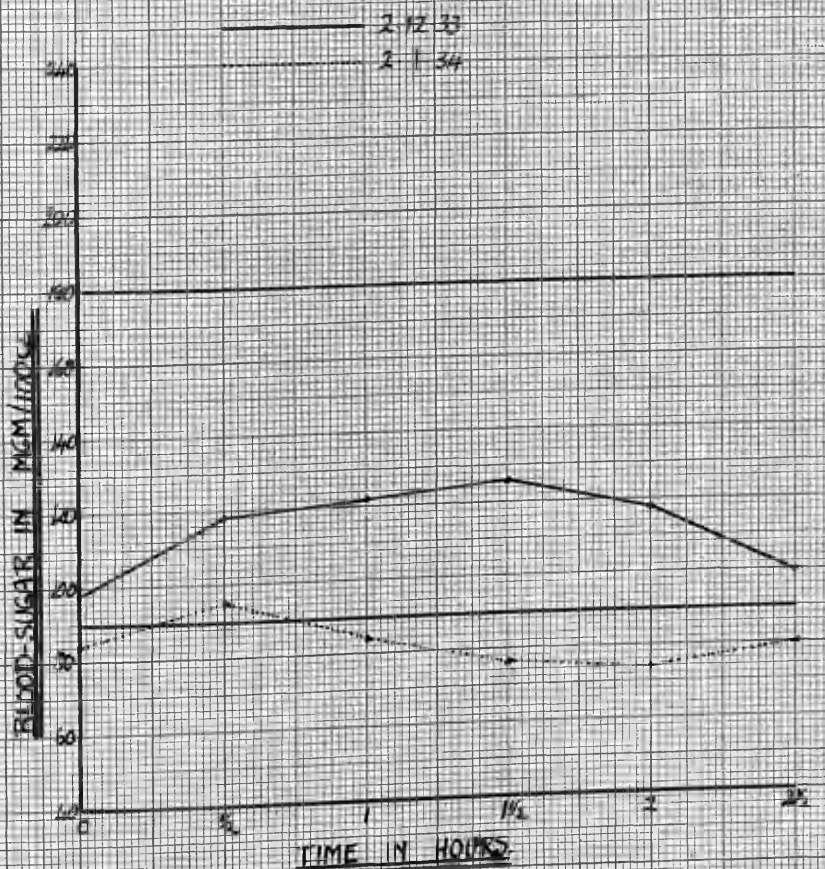
This patient was admitted on 1.12.33, the seventh day of illness. The severity of the acute stage was that of Group I. Recovery was normal and dismissal took place on 16.1.34.

A blood sugar curve on 2.12.33 was of the lag type; the fasting blood sugar was within normal limits but near the upper limit of normal. The curve was normal on 2.1.34.

The amount of reducing substance excreted in the urine was 0.295 gm. on 2.12.33 and 0.069 gm. on 2.1.34.

CASE LXVII

LAEVULOSE 20G.M.



CASE LXVIII.

N. McG., female, 12 years of age. LAEVULOSE.

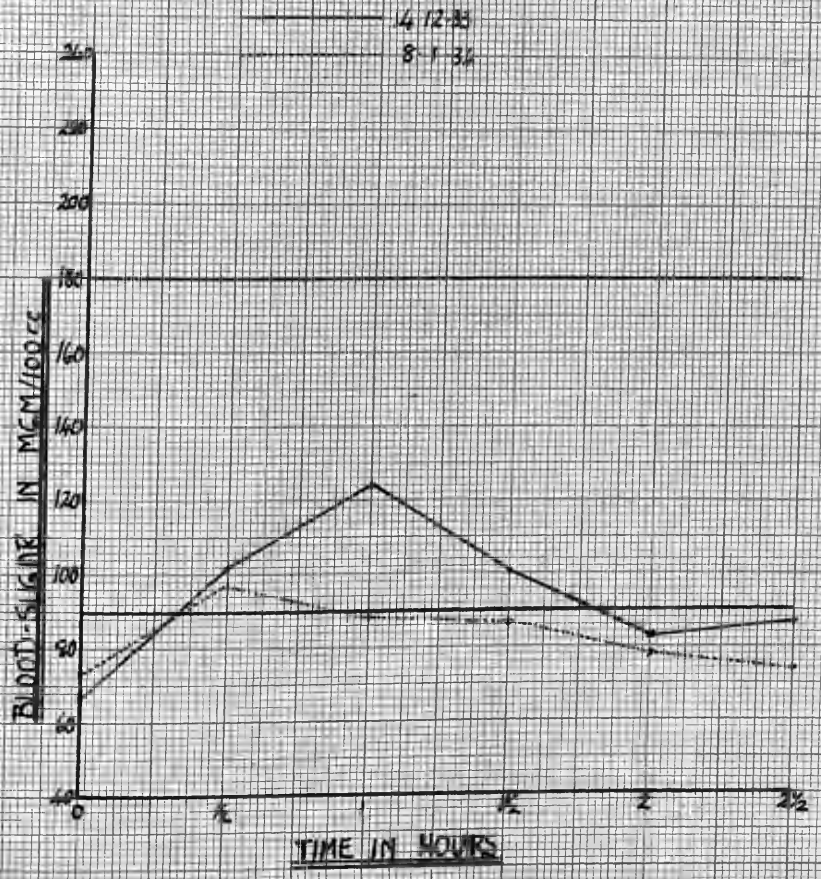
This patient was admitted on 12.12.33, the third day of illness. The severity of the acute stage was that of Group I. The patient made a normal recovery and was dismissed well on 24.1.34.

The fasting blood sugar on 14.12.33 was subnormal and the form of the curve approached that of a lag curve. On 8.1.34 the fasting blood sugar was still subnormal although higher than on admission and the curve was normal.

The amount of reducing substance excreted in the urine on 14.12.33 was 0.082 gm. and, on 8.1.34 it was 0.136 gm.

CASE LXVIII

LAEVULOSE 20GM.



CASE LXIX.

R.M., female, 14 years of age. LAEVULOSE.

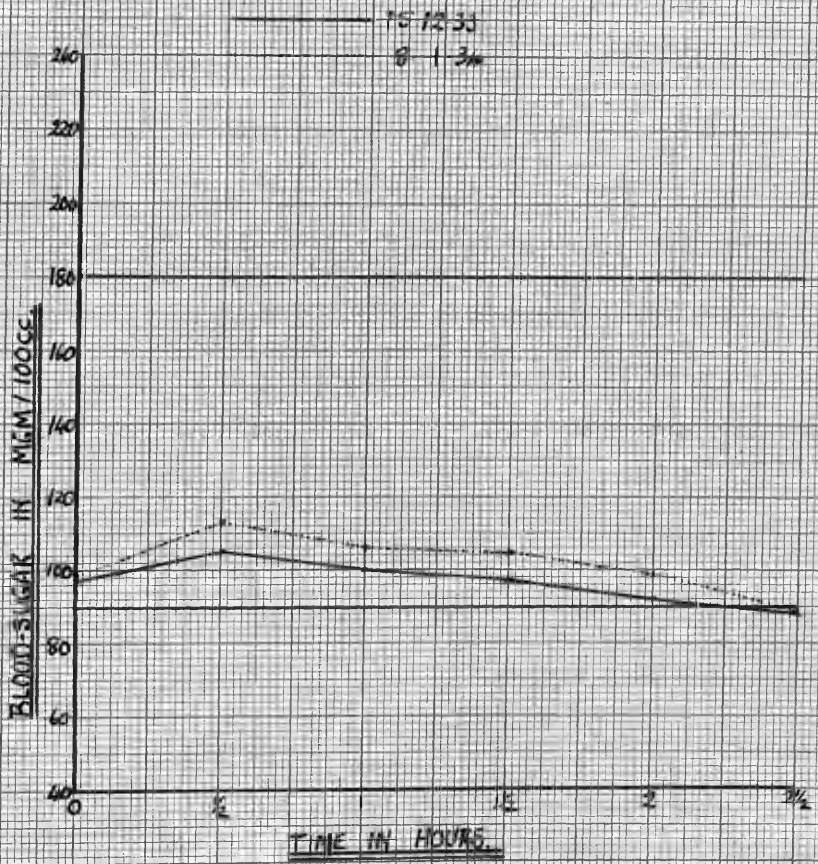
This patient was admitted on 13.12.33, the third day of illness. The severity of the acute stage was that of Group I. The patient's recovery was normal and she was dismissed on 22.1.34.

Blood sugar curves on 15.12.33 and 8.1.34 were similar; the shape in both was normal but the level was rather high.

The amount of reducing substance excreted in the urine was 0.172 gm. on 15.12.33 and 0.247 gm. on 8.1.34.

CASE LXIX

LAEVULOSE 20GM



CASE LXX.

H.V., female, 11 years of age. LAEVULOSE.

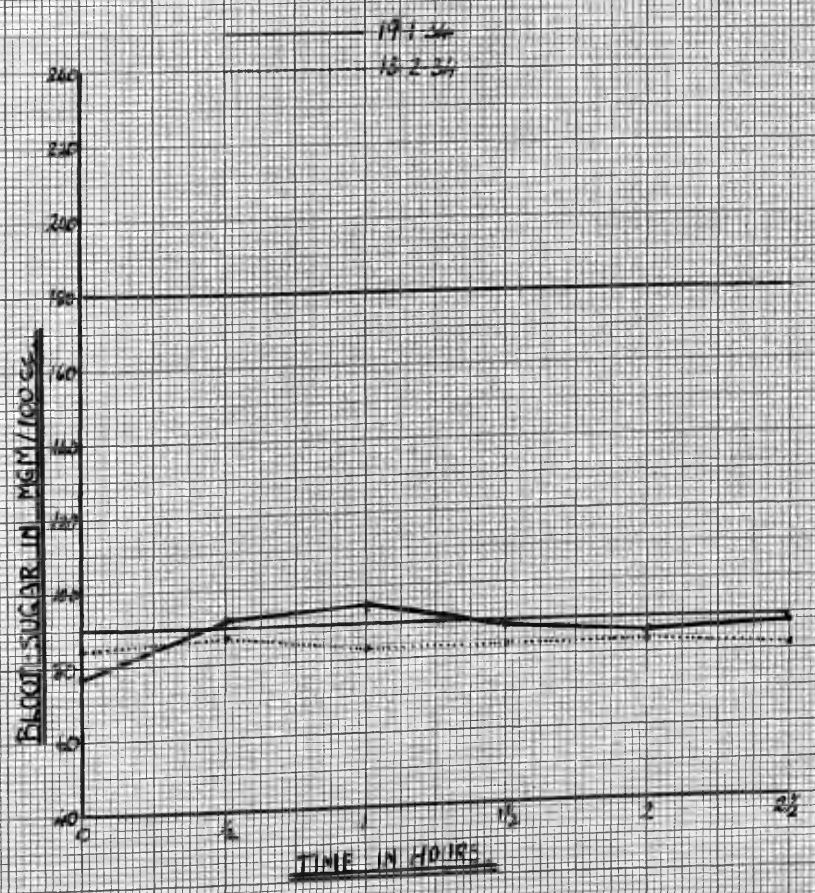
This patient was admitted on 15.1.34, the fourth day of illness. The severity of the acute stage was that of Group IV. Palatal paralysis was present from 17.1.34 till 18.2.34; cardiac paralysis of severe type from 28.1.34 till 26.2.34 and pharyngeal paralysis from 18.2.34 till 28.2.34. Positive cultures and a succession of attacks of acute tonsillitis further delayed dismissal till 28.5.34. Albuminuria was present in the first four weeks of treatment.

Blood sugar curves on 19.1.34 and 13.2.34 were both normal; the fasting blood sugar on the former date was very slightly subnormal.

The amount of reducing substance excreted in the urine was 0.128 gm. on 19.1.34 and 0.154 gm. on 13.2.34.

CASE [illegible]

LAEVULOSE 20GM.



CASE LXXI.

F. McI., female, 15 years of age. LAEVULOSE.

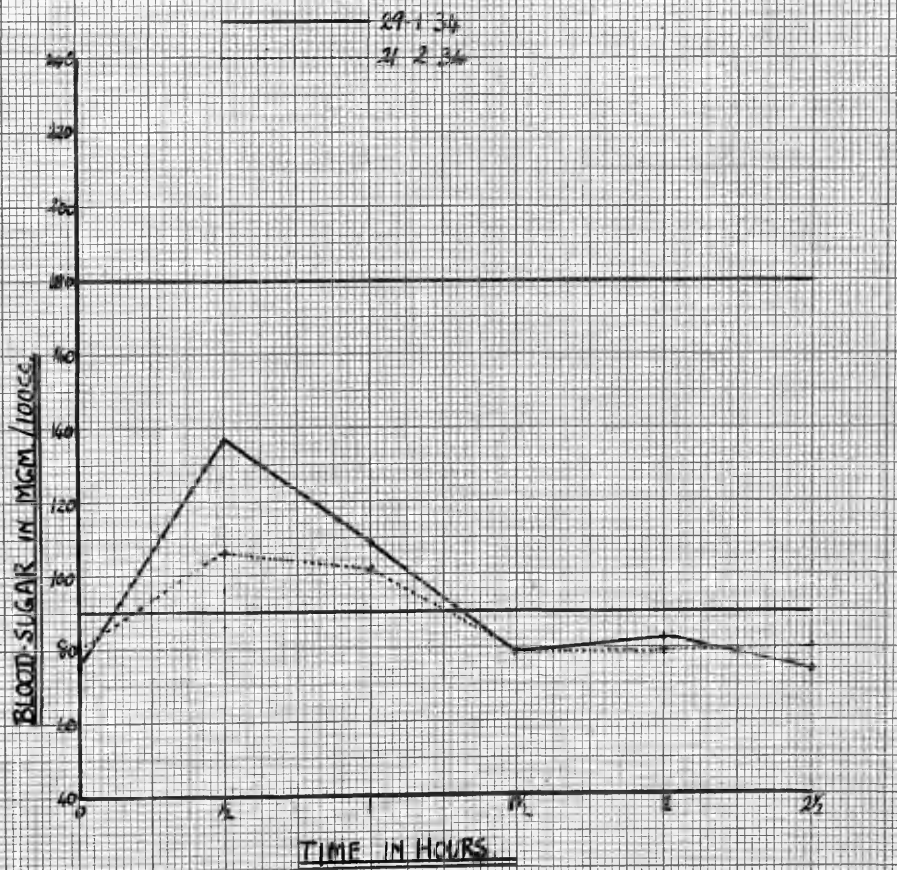
This patient was admitted on 28.1.34 the third day of treatment. The severity of the acute stage was that of Group II. Recovery was uncomplicated and dismissal took place on 14.3.34.

A blood sugar curve on 29.1.34 was similar to a normal curve following glucose with the exception that the fasting blood sugar was slightly subnormal. The curve was more nearly normal on 21.2.34.

The amount of reducing substance excreted in the urine was 0.306 gm. on 29.1.34 and 0.187 gm. on 21.2.34.

CASE LXXI

LAEVULOSE 20GM



CASE LXXII.

M.H., female, 20 years of age. LAEVULOSE.

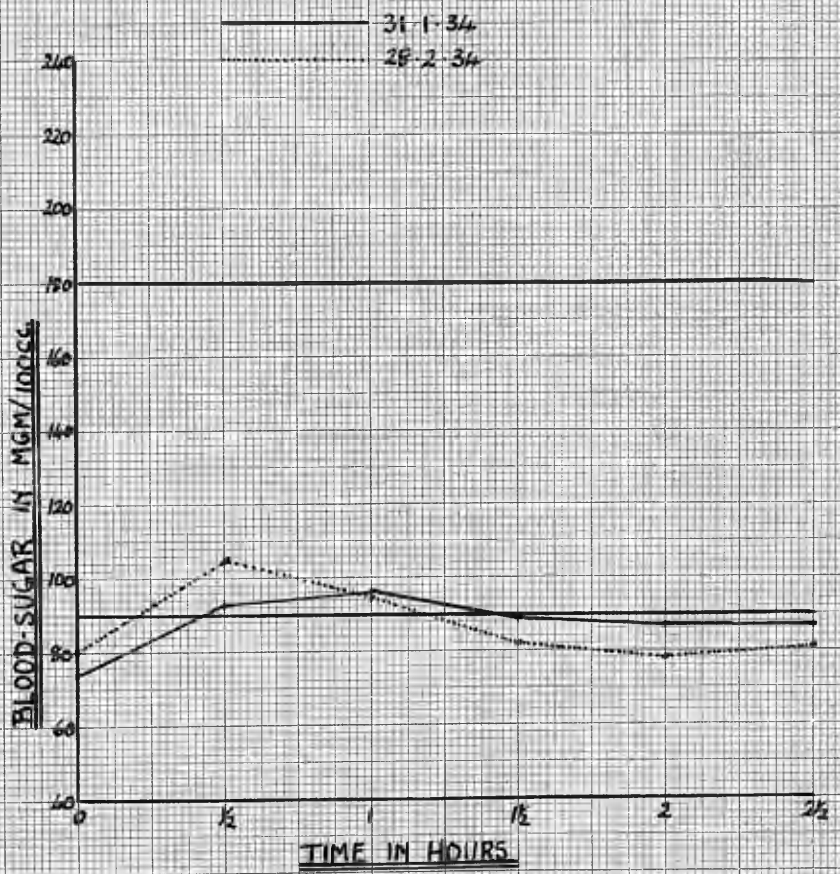
This patient was admitted on 30.1.34, the fourth day of illness. The severity of the acute stage was that of Group II. Recovery was uncomplicated but dismissal was delayed till 23.3.34 by the persistance of positive cultures.

A blood sugar curve on 31.1.34 was normal in shape but the fasting blood sugar was subnormal. On 28.2.34 the curve was practically normal.

The amount of reducing substance excreted in the urine was 0.249 gm. on 31.1.34 and 0.685 gm. on 28.2.34.

CASE LXXII

LAEVULOSE 20GM



CASE LXXIII.

J.N., female, 11 years of age. LAEVULOSE.

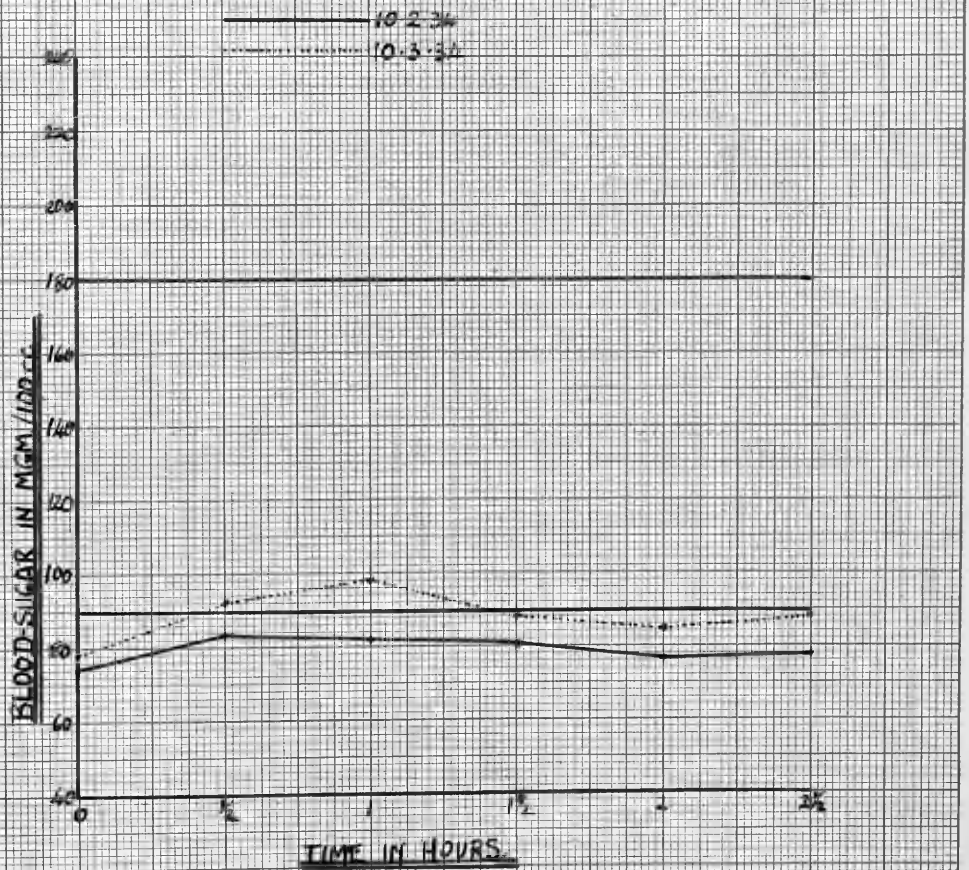
The patient was admitted on 8.2.34, the seventh day of illness. The severity of the acute stage was that of Group I. The patient had had epileptiform convulsions before admission and she had convulsions during treatment on 27.2.34, 6.3.34 and 7.3.34. She was dismissed well on 14.3.34.

The fasting blood sugar was subnormal on 10.2.34, and somewhat higher but still subnormal on 10.3.34. The curves were normal on each occasion.

The amount of reducing substance excreted in the urine was 0.245 gm. on 10.2.34 and 0.322 gm. on 10.3.34.

CASE LXXIII

LAEVULOSE 20GM.



CASE LXXIV.

G.S., female, 37 years of age. LAEVULOSE.

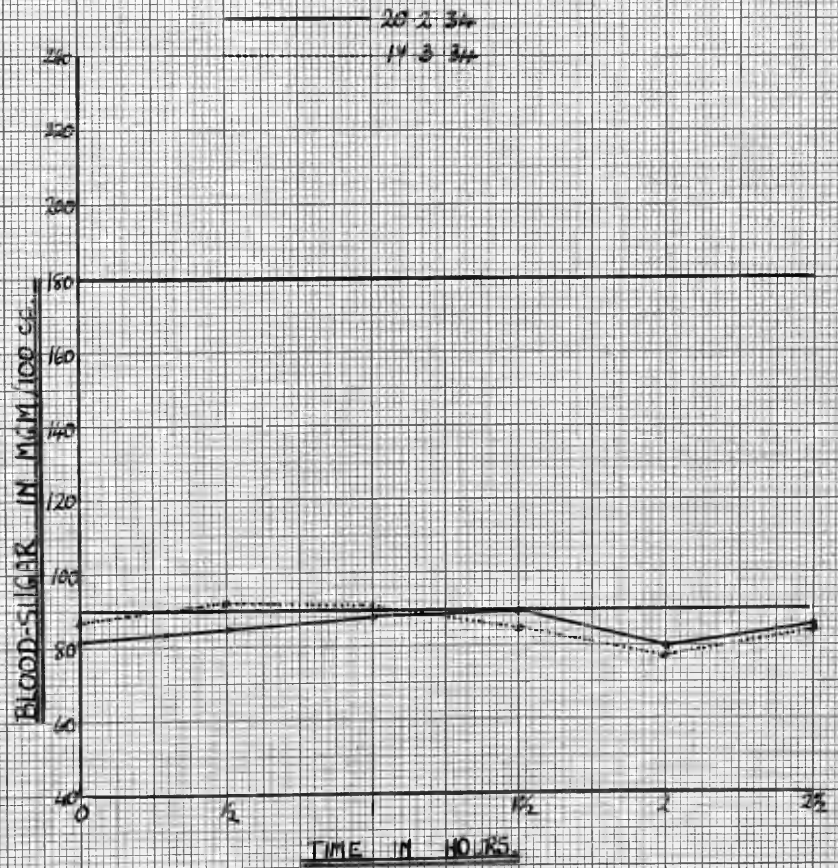
The patient was admitted on 19.2.34, the fourth day of illness. The severity of the acute stage was that of Group II. She recovered without developing paralysis and was dismissed well on 27.3.34.

Blood sugar curves were normal on 20.2.34 and on 17.3.34.

The amount of reducing substance excreted in the urine was 0.282 gms. on20.2.34 and 0.359 gm. on 17.3.34.

CASE LXXIV

LAEVULOSE 20GM.



TABLES OF RESULTS

SERUM SODIUM

SERUM CHLORINE

SERUM POTASSIUM.

TABLE 3.

GROUP I.

A. Cases Receiving Extra Sodium Chloride.

Patient	Type of recovery	Serum Sodium in Milli-Equivalents per Litre				Serum Chlorine in Milli-Equivalents per Litre.				Serum Potassium in Milli-Equivalents per Litre.			
		Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal
MAS	Normal	128.73	132.48	134.00	129.95	104.39	98.82	101.31	106.28	12.69	12.60	10.23	5.25
MDY	"	126.85	126.35	129.20	132.65	101.04	102.48	105.12	99.51	5.98	6.82	13.36	10.40
MBS	"	120.71	118.99	122.29	133.73	100.71	104.48	105.96	108.29	6.55	5.64	4.69	5.57
MMY	"	119.10	125.89	107.64	125.59	101.79	98.99	103.13	102.92	6.57	5.24	9.91	8.46
RDH	"	118.26	128.24	122.29	121.20	103.40	107.02	106.59	95.79	5.18	3.02	2.06	4.04
RCR	"	119.38	119.90	117.19	134.68	99. 97	107.58	104.40	106.82	9.35	9.82	8.46	4.42
JBT	"	115.10	125.27	121.23	118.73	105.38	95.36	98.76	103.35	7.04	6.31	12.22	7.57
JHD	Mild Paralysis.	115.59	114.32	118.75	135.08	105.05	101.68	99.49	107.04	3.99	9.73	9.80	6.20
JYG	Mild Paralysis.	130.80	118.30	121.81	138.01	105.54	98.99	98.00	101.86	3.27	6.65	5.70	6.82
EJN	Delay in Dismissal.	127.26	132.01	125.39	126.50	99.77	104.98	98.98	101.70	11.15	15.27	8.02	4.38
EKS	Normal	128.25	129.49	135.08	127.90	105.44	105.98	103.68	99.51	11.98	7.72	8.80	4.56
JHY	"	122.55	120.65	130.99	140.59	105.82	104.42	108.70	107.99	3.47	6.88	5.24	7.44
JME	"	136.23	129.20	135.76	132.23	106.95	106.09	103.97	108.83	5.16	9.23	6.82	7.38
MSN	"	114.30	119.61	106.89	133.12	105.38	92.13	103.91	105.09	4.99	4.22	3.91	3.91
ECD	"	118.15	136.25	123.72	124.04	105.68	107.33	106.87	108.36	8.14	6.98	4.39	4.55
MAR	"	128.22	131.83	124.99	129.79	105.63	101.19	101.05	101.27	6.74	4.86	4.93	5.12
FCW	"	126.44	115.10	104.31	127.64	101.72	100.66	101.32	102.93	6.67	4.20	7.98	8.06
	Average	123.29	124.93	122.44	130.08	103.74	102.25	103.01	103.97	6.76	7.38	7.32	6.11

T A B L E 3 (Contd.)

GROUP I.

B. Cases not receiving Extra Sodium Chloride.

Patient.	Type of Recovery	Serium Sodium in Milli-Equivalents per Litre.				Serum Chlorine in Milli-Equivalents per Litre				Serum Potassium in Milli-Equivalents per Litre			
		Admission	8th. Day	15th. Day	Dismissal	Admission	8th. Day	15th. Day	Dismissal	Admission	8th. day	15th. Day	Dismissal.
JNL	Normal	117.72	120.29	121.87	122.21	93.94	94.94	99.64	102.94	8.87	9.38	7.24	6.07
MDY	"	126.46	129.29	129.14	130.19	104.47	102.03	101.14	106.79	6.50	6.30	5.12	5.49
AMR	"	125.94	124.55	127.48	135.52	102.07	106.07	109.81	106.42	7.04	6.11	6.50	4.87
RHY	"	125.56	127.23	125.33	126.96	98.29	99.10	102.91	98.89	7.21	5.05	5.75	5.83
HOL	"	122.46	138.66	123.34	123.58	104.94	94.03	99.71	103.71	8.49	9.23	7.85	6.01
SZO	"	130.91	132.38	134.42	132.16	102.78	99.85	104.61	103.95	6.54	6.07	5.16	7.21
	Average	124.84	125.40	126.93	128.44	101.08	99.34	102.97	103.78	7.44	7.02	6.27	5.91

TABLE 4.

GROUP II.

A. Cases receiving extra Sodium Chloride

Patient.	Type of Recovery.	Serum Sodium in Milli-Equivalents per Litre.				Serum Chlorine in Milli-Equivalents per Litre				Serum Potassium in Milli-Equivalents per Litre			
		Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal.	Admission	8th Day	15th Day	Dismissal.
SNT	Normal	130.15	110.20	135.74	135.08	103.01	106.67	105.50	105.89	4.52	3.81	9.82	12.03
MPS	"	124.45	121.60	137.22	132.99	100.77	104.98	105.50	107.36	5.42	4.61	7.14	7.24
IST	"	124.18	104.29	128.44	130.09	99.57	101.95	106.18	105.82	4.35	5.73	10.07	5.86
MHY	"	123.20	117.16	139.86	135.95	109.25	102.04	107.28	103.21	7.93	8.27	4.09	3.15
SBE	"	125.07	125.03	123.43	132.01	101.48	97.11	99.33	102.36	5.74	5.41	6.23	7.78
MKY	"	119.54	132.04	124.63	134.53	109.25	106.88	96.37	104.70	6.05	6.69	7.52	8.31
MBD	"	124.20	140.86	116.29	133.63	102.37	103.59	98.47	109.38	6.34	5.82	4.68	3.18
WTN	Delay in Dismissal	117.89	115.56	119.90	138.46	95.70	87.88	102.60	102.77	5.26	4.93	6.40	3.41
GMY	Normal	110.00	112.00	130.27	143.25	102.02	93.14	102.57	107.59	4.37	5.72	4.96	5.06
SWT	"	115.29	121.38	117.41	125.40	97.64	101.59	100.59	103.29	7.09	8.93	6.44	6.38
MMY	"	111.26	116.85	129.68	129.69	102.60	103.29	110.32	108.00	3.32	9.04	6.20	8.46
SDN	Mild Paralysis	113.54	121.51	123.37	127.78	93.64	103.37	104.86	110.05	6.78	7.93	7.61	5.04
MFY	"	112.79	121.37	118.13	130.08	99.99	105.38	102.62	108.20	10.86	6.19	9.55	6.22
DME	"	132.03	121.10	128.24	134.03	104.35	97.46	101.54	108.75	6.00	7.10	6.92	6.30
MTN	"	124.50	116.48	114.86	136.37	96.06	102.31	104.95	107.97	14.13	6.04	8.74	5.66
EAN	"	118.47	120.33	128.97	120.03	104.97	108.01	105.15	97.68	3.95	4.65	4.66	5.79
MRS	"	112.14	121.17	114.03	134.62	91.79	93.07	98.49	102.47	3.27	6.43	4.37,	4.89
MGN	"	122.58	128.17	124.01	133.00	102.12	109.32	100.70	107.53	7.59	5.18	6.40	5.04
	Average	120.07	120.42	125.25	133.11	100.92	101.56	102.95	105.72	6.28	6.25	6.53	6.10

TABLE 4 (contd.)

GROUP II.

B. Cases not receiving extra Sodium Chloride.

Patient.	Type of Recovery	Serum Sodium in Milli-Equivalents per Litre				Serum Chlorine in Milli-Equivalents per Litre.				Serum Potassium in Milli-Equivalents per Litre.			
		Admission	8th. Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal.
MPN	Normal	118.31	116.60	107.43	132.59	101.87	106.68	102.02	103.39	10.47	11.27	7.14	6.09
MHN	"	115.42	120.53	109.62	122.95	103.70	108.44	105.77	97.83	8.50	7.79	8.10	4.90
MGT	"	118.09	127.20	129.35	134.63	104.42	106.06	98.37	103.71	6.72	4.31	5.15	3.87
JME	"	113.12	121.62	120.66	127.22	101.17	102.27	105.31	105.61	8.18	6.43	6.74	4.31
HOL	"	120.36	126.36	132.24	135.68	106.68	102.14	102.59	98.90	7.41	6.03	5.85	3.48
JMY	"	119.56	122.04	124.63	126.78	104.65	106.19	103.52	106.87	6.60	6.40	3.02	3.46
DWE	"	110.18	116.20	122.04	126.94	106.99	101.14	103.49	101.69	8.50	6.91	3.32	3.51
EMD	"	113.59	119.49	126.04	131.54	108.35	99.75	102.82	103.16	6.76	7.14	7.88	6.92
EMY	"	114.45	120.79	123.12	129.70	104.44	101.74	103.41	106.66	7.04	9.83	7.20	3.15
HME	"	122.23	119.29	126.00	137.41	103.26	99.86	108.24	105.57	6.37	6.76	7.07	3.50
CDY	"	119.08	129.85	126.08	128.53	100.09	101.19	104.90	104.92	3.66	6.06	6.37	6.31
Average		116.74	122.36	122.49	130.36	104.15	103.22	103.68	103.46	7.29	7.18	6.17	4.50

TABLE 5.

GROUP III.

A. Cases receiving Extra Sodium Chloride.

Patient	Type of Recovery.	Serum Sodium in Milli-Equivalents per Litre				Serum Chlorine in Milli-Equivalents per Litre				Serum Potassium in Milli-Equivalents per Litre.			
		Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal.
JSH	Normal	137.75	135.86	123.98	126.03	107.19	108.15	102.09	102.44	5.04	7.65	9.23	3.81
MST	"	120.50	118.46	120.16	138.69	104.44	103.32	98.79	105.01	9.22	7.53	5.04	9.30
CWE	"	113.31	117.60	115.90	133.98	102.56	102.25	103.29	105.98	9.51	7.09	4.60	8.58
JTH	"	122.36	109.89	112.16	123.43	94.56	89.91	106.31	103.37	8.54	7.63	7.01	5.11
GJE	"	120.80	120.02	105.51	133.25	108.82	106.88	104.93	107.32	6.98	6.59	6.37	6.98
MBE	"	120.81	130.31	129.69	135.18	99.17	106.83	106.34	105.63	7.69	7.33	6.80	7.88
CCE	"	110.96	112.64	124.71	130.63	91.09	105.49	105.79	109.49	7.93	6.72	5.95	4.89
DME	"	130.01	122.54	123.87	129.49	106.06	106.31	106.59	100.07	11.15	15.73	8.90	4.99
MMN	"	121.44	131.24	133.19	131.18	101.06	102.67	101.92	103.97	6.87	9.10	7.61	9.58
EMW	"	122.21	129.04	136.00	130.62	96.13	99.57	97.28	99.28	9.03	6.64	3.78	5.23
MDN	"	111.90	117.10	117.99	138.88	97.29	93.31	106.70	104.36	9.97	6.05	5.60	4.23
	Average	121.10	122.25	122.11	131.94	100.76	102.29	103.64	104.27	8.36	8.01	6.44	6.43

TABLE 5 (contd.)

GROUP III.

B. Cases not receiving Extra Sodium Chloride.

Patient	Type of Recovery	Serum Sodium in Milli-Equivalents per Litre				Serum Chlorine in Milli-Equivalents per Litre				Serum Potassium in Milli-Equivalents per Litre			
		Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal
MMR	Mild Paralysis	134.19	113.99	114.30	123.19	102.10	103.68	106.06	105.00	5.21	8.51	4.34	4.68
CSS	Normal	125.03	117.62	133.32	132.64	104.17	98.01	106.13	103.72	5.28	5.61	4.55	7.16
WAN	"	115.26	121.48	122.11	126.45	95.41	102.36	102.96	101.70	6.35	5.04	5.32	5.21
RWE	Delay in Dismissal	111.90	123.11	114.08	132.47	94.63	94.93	102.04	107.21	11.28	8.00	7.16	11.22
EMN	Normal	114.93	121.55	118.62	126.00	93.90	105.11	107.21	104.14	9.59	7.13	5.49	4.04
PGL	"	119.89	126.13	126.74	134.73	107.68	100.54	100.98	101.31	6.92	5.04	5.73	6.60
LSH	Mild Paralysis	127.31	135.59	117.50	133.48	104.99	106.62	103.03	106.95	5.47	3.86	6.85	4.56
MPN	Normal	121.80	127.56	128.04	133.34	105.18	106.34	105.90	105.16	7.83	5.22	5.85	4.27
	Average	121.29	123.28	121.84	130.28	101.01	102.20	104.29	104.40	7.24	6.05	5.66	5.72

TABLE 6.

GROUP IV.

A. Cases receiving extra Sodium Chloride

Patient	Recovery	Serum Sodium in Milli-Equivalents per Litre				Serum Chlorine in Milli-Equivalents per Litre				Serum Potassium in Milli-Equivalents per Litre.			
		Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal
CDF	Mild Paralysis	116.70	118.41	110.96	134.13	99.64	92.32	102.93	103.04	9.17	7.68	4.52	8.02
ELE	Normal	132.32	115.90	125.27	132.64	106.79	103.03	99.84	109.48	7.09	6.51	6.31	4.18
EBN	Mild Paralysis	128.76	123.54	126.64	130.36	99.52	97.96	105.47	105.61	7.04	5.16	4.76	4.37
JTR	Mild Paralysis	109.82	119.90	126.83	134.59	98.66	108.30	103.43	103.79	10.00	9.02	6.61	5.53
DLK	Severe Paralysis	119.82	122.08	126.87	139.73	97.60	89.51	102.02	107.62	4.29	4.41	3.06	5.66
JDT	Severe Paralysis	126.64	112.05	134.28	135.50	93.67	90.61	107.71	108.17	4.56	3.37	3.72	6.79
ESN	Normal	112.11	109.41	111.26	127.55	101.06	108.64	102.02	106.09	5.85	7.25	6.40	8.18
FMN	"	134.13	142.11	120.08	127.55	104.30	107.91	103.53	99.33	4.65	3.27	4.40	3.41
MKN	Severe Paralysis	125.88	123.98	127.72	126.50	106.95	107.52	106.28	100.44	7.23	10.51	8.36	6.82
	Average	122.91	120.82	123.33	132.06	100.91	100.64	103.71	104.84	6.65	6.35	5.35	5.88

TABLE 6 (contd.)

GROUP IV.

B. Cases not receiving extra Sodium Chloride.

Patient	Recovery	Serum Sodium in Milli-Equivalents per Litre.				Serum Chlorine in Milli-Equivalents per Litre				Serum Potassium in Milli-Equivalents per Litre.			
		Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal	Admission	8th Day	15th Day	Dismissal.
CBR	Mild Paralysis	122.55	134.49	125.88	139.34	103.62	107.21	103.89	106.53	6.20	5.53	7.44	5.11
ATS	Normal	112.45	127.38	124.49	128.03	100.45	106.64	101.00	106.59	11.09	8.70	8.51	4.91
AMR	Delay in Dismissal	115.83	122.29	131.61	131.80	95.21	104.69	104.20	104.74	11.80	8.41	6.62	6.93
JAN	Delay in Dismissal	118.87	128.64	130.46	129.57	97.14	98.31	99.08	101.33	5.58	5.41	5.49	5.31
	Average	117.43	128.20	128.11	132.18	99.10	104.21	102.04	104.80	8.67	7.01	7.02	5.57

TABLE 7.

GROUP IV.

DEATHS.

(No cases receiving extra Sodium Chloride.)

Patient	Serum Sodium in Milli-Equivalents per Litre		Serum Chlorine in Milli-Equivalents per Litre		Serum Potassium in Milli-Equivalents per Litre.	
	Admission	Before Death	Admission	Before Death	Admission	Before Death.
RB	112.23	98.64	90.10	75.37	9.34	* -
JW	124.31	97.72	97.55	77.78	7.46	* -
MG	116.20	107.49	88.23	86.15	8.97	11.08
JM	121.14	104.53	96.47	82.44	7.61	12.23
Average	118.47	100.85	93.09	80.44	8.35	11.66

* Insufficient Serum.

TABLES OF RESULTS AND STATISTICAL CALCULATIONS REGARDING TREATMENT BY SODIUM CHLORIDE

TABLE 8.

SATISFACTORY RECOVERIES.

Showing the Results of Treatment in recovered Cases receiving Sodium Chloride and in recovered Cases not receiving Sodium Chloride according to the severity of the acute stage.

	Treatment	Total Recoveries	Satisfactory Recoveries		Difference between Percentages	Standard Error of Difference.	Difference / Standard Error.
			Number of Cases	Percentage of Total Recoveries			
All recovered Cases	Receiving Sod.Chlor.	185	118	63.78	2.52	4.09	0.62
	Not receiving Sod. Chlor.	555	340	61.26			
Recovered Cases in Group I.	Receiving Sod. Chlor	36	25	69.44	9.55	8.54	1.12
	Not receiving Sod. Chlor	119	94	78.99			
Recovered Cases in Group II.	Receiving Sod. Chlor	71	54	76.06	4.47	5.77	0.77
	Not receiving Sod. Chlor	264	189	71.59			
Recovered Cases in Group III.	Receiving Sod. Chlor	48	32	66.67	31.92	7.90	4.17
	Not receiving Sod. Chlor.	141	49	34.75			
Recovered Cases in Group IV.	Receiving Sod. Chlor	30	7	23.33	2.48	11.02	0.23
	Not receiving Sod. Chlor.	31	8	25.81			

TABLE 9.

UNCOMPLICATED RECOVERIES.

Showing the results of treatment in recovered Cases receiving Sodium Chloride and in recovered Cases not receiving Sodium Chloride according to the severity of the acute stage.

	Treatment	Total Recoveries.	Uncomplicated Recoveries		Difference between Percentages	Standard Error of Difference.	Difference / Standard Error.
			Number of Cases.	Percentage of Total Recoveries.			
All recovered Cases	Receiving Sod. Chlor.	185	139	75.14	0.18	0.84	0.21
	Not receiving Sod. Chlor.	555	418	75.32			
Recovered Cases in Group I.	Receiving Sod. Chlor.	36	32	88.89	1.87	5.87	0.32
	Not receiving Sod. Chlor.	119	108	90.76			
Recovered Cases in Group II.	Receiving Sod. Chlor.	71	61	85.92	3.34	4.74	0.70
	Not receiving Sod.Chlor.	264	218	82.58			
Recovered Cases in Group III.	Receiving Sod.Chlor.	48	37	77.08	21.76	4.61	4.73
	Not receiving Sod. Chlor.	141	78	55.32			
Recovered Cases in Group IV.	Receiving Sod. Chlor.	30	9	30.00	15.16	12.25	1.24
	Not receiving Sod. Chlor.	31	14	45.16			

T A B L E 10.

DELAYED RECOVERIES.

Showing the Results of Treatment in recovered Cases receiving Sodium Chloride and in recovered Cases not receiving Sodium Chloride according to the Severity of the acute stage.

	Treatment	Total Recoveries	Delayed Recoveries		Difference between Percentages	Standard Error of Difference	Difference / Standard Error.
			Number of Cases	Percentage of Total Recoveries.			
All recovered Cases	Receiving Sod.Chlor.	185	21	11.35	2.70	2.76	0.98
	Not receiving Sod. Chlor.	555	78	14.05			
Recovered Cases in Group I	Receiving Sod. Chlor.	36	7	19.44	7.68	7.23	1.06
	Not receiving Sod. Chlor.	119	14	11.76			
Recovered Cases in Group II.	Receiving Sod. Chlor.	71	7	9.86	1.12	4.03	0.28
	Not receiving Sod. Chlor.	264	29	10.98			
Recovered Cases in Group III	Receiving Sod. Chlor.	48	5	10.42	10.15	5.57	1.82
	Not receiving Sod. Chlor.	141	29	20.57			
Recovered Cases in Group IV.	Receiving Sod.Chlor.	30	2	6.67	12.68	8.43	1.51
	Not receiving Sod.Chlor.	31	6	19.35			

TABLE 11.

COMPLICATED RECOVERIES - Cases with Mild Paralysis.

Showing the proportion of cases which recovered with Mild Paralysis with all cases of recovery with Paralysis (mild and severe), according to whether the cases received Sodium Chloride or not, and according to the severity of the acute stage.

	Treatment	Total Recoveries with Paralysis	Recoveries with Mild Paralysis		Difference between Percentages	Standard error of Difference.	Difference / Standard error.
			Number of Cases	Percentage of Total Recoveries with Paralysis			
All recovered cases with Paralysis	Receiving Sod. Chlor.	46	39	84.78	7.20	5.90	1.22
	Not receiving Sod. Chlor.	137	126	91.98			
Recovered cases with Paralysis Group I.	Receiving Sod. Chlor.	4	4	100.00	0	-	-
	Not receiving Sod. Chlor.	11	11	100.00			
Recovered cases with Paralysis Group II.	Receiving Sod. Chlor.	10	9	90.00	3.48	3.76	0.93
	Not receiving Sod. Chlor.	46	43	93.48			
Recovered cases with Paralysis Group III.	Receiving Sod. Chlor.	11	11	100.00	9.52	3.66	2.60
	Not receiving Sod. Chlor.	63	57	90.48			
Recovered Cases with Paralysis Group IV.	Receiving Sod. Chlor.	21	15	71.43	16.81	12.58	1.34
	Not receiving Sod.Chlor.	17	15	88.24			

TABLE 12.

FATAL RESULTS - Deaths within twenty-four hours of Admission.

Showing the proportion of cases which died within twenty-four hours of admission as compared with all the cases which died, according to whether the cases received Sodium Chloride or not and according to the severity of the acute stage.

	Treatment	Total Deaths.	Deaths within twenty-four hours of admission.		Difference between Percentages	Standard error of Difference.	Difference / Standard Error
			Number of cases	Percentage of Total Deaths.			
All Deaths	Receiving Sod.Chlor.	15	3	20.00	5.00	13.05	0.38
	Not receiving Sod. Chlor.	20	3	15.00			
Deaths in Subgroups C6 and C7	Receiving Sod. Chlor.	4	0	0.00	16.67	15.22	1.10
	Not receiving Sod. Chlor.	6	1	16.67			
Deaths in Subgroup C8.	Receiving Sod. Chlor.	11	3	27.27	12.99	16.36	0.79
	Not receiving Sod. Chlor.	14	2	14.29			

TABLE 13.

FATAL RESULTS - Deaths within four days of admission.

Showing the proportion of cases which died within four days of admission as compared with all the cases which died, according to whether the cases received Sodium Chloride or not, and according to the severity of the acute stage.

	Treatment	Total Deaths.	Deaths within four days of Admission.		Difference between Percentages	Standard error of Difference	Difference / Standard error.
			Number of cases	Percentage of Total Deaths			
All Deaths	Receiving Sod. Chlor.	15	5	33.33	11.67	11.77	0.99
	Not receiving Sod. Chlor.	20	9	45.00			
Deaths in Sub-Groups C6 & C7	Receiving Sod. Chlor.	4	0	0.00	66.67	19.24	3.47
	Not receiving Sod. Chlor.	6	4	66.67			
Deaths in Sub-Group. C8.	Receiving Sod. Chlor.	11	5	45.45	9.74	19.73	0.49
	Not receiving Sod. Chlor.	14	5	35.71			

TABLE 14.

FATAL RESULTS (excluding those in first twenty-four hours in Hospital) - Deaths between second and fourth days inclusive.

Showing the proportion of cases, which died after the end of the first day and before the end of the fourth day in hospital, as compared with all the cases which died after the end of the first day in hospital, according to whether the cases received Sodium Chloride or not and according to the severity of the acute stage.

	Treatment.	Total Deaths after the 1st. day in hospital	Deaths after two to four days' residence inclusive		Difference between Percentages	Standard error of Difference	Difference / Standard error.
			No. of cases	Percentage of Total Deaths.			
All Deaths	Receiving Sod. Chlor.	12	2	16.67	18.63	15.81	1.18
	Not receiving Sod. Chlor.	17	6	35.29			
Deaths in Sub-Groups C6 & C7	Receiving Sod. Chlor.	4	0	0.00	60.00	21.91	2.74
	Not.receiving Sod.Chlor.	5	3	60.00			
Deaths in Sub-Group. C8.	Receiving Sod. Chlor.	8	2	25.00	0.00	19.77	0.00
	Not receiving Sod. Chlor.	12	3	25.00			

TABLE 15.

FATAL RESULTS - Deaths within one week of admission.

Showing the proportion of cases which died within one week of admission as compared with all the cases which died, according to whether the cases received Sodium Chloride or not, and according to the severity of the acute stage.

	Treatment.	Total Deaths.	Deaths within one week of admission.		Difference between percentages.	Standard error of Difference	Difference / Standard error.
			No. of Cases	Percentage of Total Deaths			
All Deaths	Receiving Sod. Chlor.	15	8	53.33	1.67	17.02	0.10
	Not receiving Sod. Chlor.	20	11	55.00			
Deaths in Sub-Groups C6 & C7	Receiving Sod. Chlor.	4	2	50.00	16.67	31.55	0.53
	Not receiving Sod. Chlor.	6	4	66.67			
Deaths in Sub-Group C8.	Receiving Sod. Chlor.	11	6	54.55	4.55	20.10	0.23
	Not receiving Sod. Chlor.	14	7	50.00			

TABLE 16.

FATAL RESULTS (excluding those in cases less than twenty-four hours in Hospital - Deaths between second and Seventh days inclusive.

Showing the proportion of cases, which died after the end of the first day and before the end of the seventh day in hospital, as compared with all the cases which died after the end of the first day in hospital, according to whether the cases received sodium chloride or not and according to the severity of the acute stage.

	Treatment	Total Deaths after 1st.day in hospital	Deaths after two to seven Days in Hospital inclusive		Difference between Percentages	Standard Error of Difference	Difference / Standard error.
			No.of Cases	Percentage of Total Deaths.			
All Deaths	Receiving Sod.Chlor.	12	5	41.67	5.92	18.68	0.32
	Not receiving Sod. Chlor.	17	8	47.59			
Deaths in Sub-Groups C6 & C7	Receiving Sod.Chlor.	4	2	50.00	10.00	33.24	0.30
	Not receiving Sod.Chlor.	5	3	60.00			
Deaths in Sub-Group C8	Receiving Sod. Chlor.	8	3	37.50	4.17	22.27	0.19
	Not receiving Sod. Chlor.	12	5	41.67			

TABLE 17.

FATAL RESULTS - Deaths within two weeks of admission.

Showing the proportion of cases which died within two weeks of admission as compared with all the cases which died, according to whether the cases received Sodium Chloride or not, and according to the severity of the acute stage.

	Treatment	Total Deaths.	Deaths within two weeks of admission		Difference between Percentages	Standard error of Difference	Difference / Standard error.
			No. of Cases	Percentage of Total Deaths			
All Deaths	Receiving Sod. Chlor.	15	12	80.00	15.00	11.42	1.31
	Not receiving Sod. Chlor.	20	19	95.00			
Deaths in Sub-Groups C6 & C7	Receiving Sod. Chlor.	4	3	75.00	25.00	21.65	1.15
	Not receiving Sod. Chlor.	6	6	100.00			
Deaths in Sub-Group C8	Receiving Sod. Chlor.	11	9	88.89	3.97	13.52	0.29
	Not receiving Sod. Chlor.	14	13	92.86			

TABLE 18.

FATAL RESULTS (Excluding those cases which are less than twenty-four hours in hospital) - Deaths between second and fourtheenth days inclusive.

Showing the proportion of cases, which died after the end of the first day and before the end of the fourteenth day in hospital, as compared with all the cases which died after the end of the first day in hospital, according to whether the cases received Sodium Chloride or not and according to the severity of the acute stage.

	Treatment	Total Deaths after 1st.day in hospital	Deaths after two to Fourteen days in Hospital (inclusive).		Difference between Percentages	Standard error of Difference	Difference / Standard error
			No.of Cases	Percentage of Total Deaths.			
All Deaths	Receiving Sod.Chlor.	12	9	75.00	19.12	13.76	1.39
	Not receiving Sod. Chlor.	17	16	94.12			
Deaths in Sub-Groups C6 & C7	Receiving Sod. Chlor.	4	3	75.00	25.00	21.65	1.15
	Not receiving Sod. Chlor.	5	5	100.00			
Deaths in Sub-Group C8.	Receiving Sod. Chlor.	8	6	75.00	16.67	17.26	0.97
	Not receiving Sod. Chlor.	12	11	91.67			

TABLE 19.

Showing the Distribution of all cases in Group IV in the Series which received Sodium Chloride and in the Series which did not receive Sodium Chloride.

	Cases in Group IV receiving Sodium Chloride.		Cases in Group IV not receiving Sodium Chloride.	
	Number	Percentage	Number	Percentage
Subgroup C6	14	31.11	27	52.94
Subgroup C7	9	20.00	9	17.65
Subgroup C8	22	48.89	15	29.41
Group IV: TOTAL	45	100.00	51	100.00

T A B L E 20.

Showing the distribution of cases in Group IV, with the exception of cases which died within twenty-four hours of admission in the series which received Sodium Chloride and in the series which did not receive Sodium Chloride.

	Cases in Group IV (Except Deaths within 24 hours receiving Sodium Chloride		Cases in Group IV (Except Deaths within 24 hours) not receiving Sodium Chloride	
	Number	Percentage	Number	Percentage
Subgroup C6	14	33.33	26	54.17
Subgroup C7	9	21.43	9	18.75
Subgroup C8	19	45.24	13	27.08
Group IV: TOTAL	42	100.00	48	100.00

T A B L E 21.

GROUP IV: RECOVERIES.

Showing the proportion of recoveries to all cases (recoveries and deaths) according to whether the cases received Sodium Chloride or not, and according to the severity of the acute stage within the group.

	Treatment	Group IV All Cases	Group IV: Recoveries		Difference between Percentages	Standard error of Difference	Difference / Standard error.
			Number	Percentage			
Group IV.	Receiving Sod. Chlor.	45	30	66.67	5.89	9.80	0.60
	Not receiving Sod. Chlor.	51	31	60.78			
Subgroup C6	Receiving Sod. Chlor.	14	14	100.00	11.11	6.05	1.84
	Not receiving Sod. Chlor	27	24	88.89			
Subgroup C7	Receiving Sod. Chlor.	9	5	55.56	11.11	22.85	0.48
	Not receiving Sod. Chlor.	9	6	66.67			
Subgroup C8	Receiving Sod. Chlor.	22	11	50.00	43.33	12.46	3.48
	Not receiving Sod. Chlor.	15	1	6.67			

TABLE 22.

GROUP IV: RECOVERIES.

Showing the proportion of recoveries to all cases (recoveries and deaths), with the exception of deaths within the first twenty-four hours of treatment, according to whether the cases received Sodium Chloride or not, and according to the severity of the acute stage within the Group.

	Treatment	Group IV. All cases except groups within 24 hrs.	Group IV: Recoveries		Difference Between Percentages	Standard error of Difference.	Difference / Standard error.
			Number	Percentage			
Group IV	Receiving Sod. Chlor.	42	30	71.43	6.85	9.91	0.69
	Not receiving Sod. Chlor.	48	31	64.58			
Subgroup C6	Receiving Sod. Chlor.	14	14	100.00	7.69	5.23	1.47
	Not receiving Sod. Chlor.	26	24	92.31			
Subgroup C7	Receiving Sod. Chlor.	9	5	55.56	11.11	22.85	0.48
	Not receiving Sod. Chlor.	9	6	66.67			
Subgroup C8	Receiving Sod.Chlor.	19	11	57.89	50.20	13.52	3.71
	Not receiving Sod. Chlor.	13	1	7.69			

TABLE 23.

GROUP I.

Showing detailed results in each subgroup and according to age-group in cases which received extra Sodium Chloride and in cases which did not receive extra Sodium Chloride.

GROUP I.		AGE-GROUPS										All Age-Groups	
		0-4		5-9		10-14		15-19		20 -			
Sub-Group	Result.	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl
A1	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paralysis	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Mild Paralysis	0	0	1	1	0	0	0	1	0	1	1	3
	Recovery - Delay	0	3	2	4	0	1	0	0	1	0	3	8
	Recovery - Normal	0	0	1	10	0	3	1	4	4	3	6	20
	Total	0	3	4	15	0	4	1	5	5	4	10	31
A2	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paralysis	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Mild Paralysis	0	1	2	7	1	0	0	0	0	0	3	8
	Recovery - Delay	0	2	2	1	1	2	1	0	0	0	4	5
	Recovery - Normal	5	15	3	25	7	14	1	8	3	3	19	65
	Total	5	18	7	33	9	16	2	8	3	3	26	78
B1	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paral.	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Mild Paral.	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Delay	0	0	0	1	0	0	0	0	0	0	0	1
	Recovery - Normal	0	0	0	1	0	0	0	0	0	1	0	2
	Total	0	0	0	2	0	0	0	0	0	1	0	3
TOTAL: All Subgroups		5	21	11	50	9	20	3	13	8	8	36	112

TABLE 24.

GROUP II

Showing detailed results in each subgroup and according to age-group in cases which received extra Sodium Chloride and in cases which did not receive extra Sodium Chloride.

Sub-Group	GROUP II. Result	AGE GROUPS 0-4 NaCl	0-4 No NaCl	5-9 NaCl	5-9 No NaCl	10-14 NaCl	10-14 No NaCl	15-19 NaCl	15-19 No NaCl	20 - NaCl	20 - No NaCl	All Age-Groups NaCl	All Age-Groups No NaCl
B2	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paralysis	0	1	1	1	0	1	0	0	0	0	1	3
	Recovery - Mild Paralysis	1	7	4	14	1	5	0	3	1	1	7	30
	Recovery - Delay	1	6	3	3	1	5	1	2	0	2	6	18
	Recovery - Normal	7	33	16	50	7	34	3	10	6	17	39	144
	Total	9	47	24	68	9	45	4	15	7	20	53	195
B3 B4 B5	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paralysis	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Mild paralysis	0	1	0	7	0	1	0	0	1	0	1	9
	Recovery - Delay	0	1	0	5	0	0	0	0	0	0	0	6
	Recovery - Normal	1	7	1	13	4	9	0	4	0	3	6	36
C2	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paralysis	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Mild Paralysis	0	2	0	1	1	1	0	0	0	0	1	4
	Recovery - Delay	0	2	1	3	0	0	0	0	0	0	1	5
	Recovery - Normal	3	2	4	3	2	2	0	2	0	0	9	9
	Total	3	6	5	7	3	3	0	2	0	0	11	18
TOTAL: All Subgroups		13	62	30	100	16	58	4	21	8	23	71	264

TABLE 25.

GROUP III.

Showing detailed results in each subgroup and according to age-groups in cases which received extra Sodium Chloride and in cases which did not receive extra Sodium Chloride.

GROUP III		AGE GROUPS										All Age-Groups	
		0-4		5-9		10-14		15-19		20-			
Sub-Group	Result.	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl
C3	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paralysis	0	0	0	1	0	0	0	0	0	1	0	2
	Recovery - Mild Paralysis	2	13	2	21	0	1	0	1	1	2	5	38
	Recovery - Delay	0	3	1	10	0	2	0	3	0	0	1	18
	Recovery - Normal	4	8	2	11	5	3	2	1	4	3	17	26
	Total	6	24	5	43	5	6	2	5	5	6	23	84
C4	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paralysis	0	0	0	0	0	2	0	0	0	0	0	2
	Recovery - Mild Paralysis	0	3	3	4	0	5	0	0	0	0	3	12
	Recovery - Delay	0	3	1	4	1	0	0	0	0	0	2	7
	Recovery - Normal	4	3	5	9	2	4	1	2	1	1	13	19
	Total	4	9	9	17	3	11	1	2	1	1	18	40
C5	Death	0	0	0	0	0	0	0	0	0	0	0	0
	Recovery - Severe Paralysis	0	1	0	1	0	0	0	0	0	0	0	2
	Recovery - Mild Paralysis	1	2	2	5	0	0	0	0	0	0	3	7
	Recovery - Delay	1	0	1	3	0	0	0	1	0	0	2	4
	Recovery - Normal	0	1	1	1	1	0	0	1	0	1	2	4
	Total	2	4	4	10	1	0	0	2	0	1	7	17
TOTAL: All Subgroups		12	37	18	70	9	17	3	9	6	8	48	141

TABLE 26

GROUP IV: SUBGROUP C6

Showing detailed results according to age-groups in cases which received extra Sodium Chloride and in cases which did not receive extra sodium Chloride.

GROUP IV - SUBGROUP C6		AGE GROUPS.										All Age-Groups	
		0-4		5-9		10-14		15-19		20-			
	Result.	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl
Death	1st Day	0	1	0	0	0	0	0	0	0	0	0	1
	2nd - 4th Days	0	0	0	1	0	0	0	0	0	0	0	1
	5th - 7th Days	0	0	0	0	0	0	0	0	0	0	0	0
	8th - 14th Days	0	1	0	0	0	0	0	0	0	0	0	1
	Over 14th Day	0	0	0	0	0	0	0	0	0	0	0	0
Recovery	Severe Paralysis	0	0	0	0	0	1	0	1	1	0	1	2
	Mild Paralysis	0	2	4	5	2	2	0	1	0	0	6	10
	Delayed	0	0	0	1	1	3	0	1	0	0	1	5
	Normal	0	2	3	3	0	1	2	1	1	0	6	7
TOTAL		0	6	7	10	3	7	2	4	2	0	14	27

TABLE 27.

GROUP IV: SUBGROUP C7.

Showing detailed results according to age-groups in cases which received extra Sodium Chloride and in cases which did not receive extra Sodium Chloride.

GROUP IV - SUBGROUP C7		AGE GROUPS											
		0-4		5-9		10-14		15-19		20-			
Result		NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl
Death	1st Day	0	0	0	0	0	0	0	0	0	0	0	0
	2nd - 4th Days	0	1	0	1	0	0	0	0	0	0	0	2
	5th - 7th Days	0	0	2	0	0	0	0	0	0	0	2	0
	8th - 14th Days	0	0	1	1	0	0	0	0	0	0	1	1
	Over 14th day	1	0	0	0	0	0	0	0	0	0	1	0
Recovery	Severe Paralysis	0	0	1	0	0	0	0	0	0	0	1	0
	Mild Paralysis	0	0	3	2	1	0	0	2	0	0	4	4
	Delayed	0	0	0	0	0	0	0	0	0	1	0	1
	Normal	0	0	0	0	0	0	0	1	0	0	0	1
TOTAL		1	1	7	4	1	0	0	3	0	1	9	9

TABLE 28.

GROUP IV: SUBGROUP C8.

Showing detailed results according to age-groups in cases which received extra Sodium Chloride and in cases which did not receive extra Sodum Chloride.

GROUP IV - SUBGROUP C8.		AGE GROUPS										All Age-Groups	
		0-4		5-9		10-14		15-19		20-			
	Result.	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl	NaCl	No NaCl
Death	1st Day	2	1	1	0	0	0	0	0	0	1	3	2
	2nd - 4th Days	0	2	1	1	1	0	0	0	0	0	2	3
	5th - 7th Days	0	1	1	1	0	0	0	0	0	0	2	3
	8th - 14th Days	0	1	2	3	1	1	0	0	0	1	3	6
	Over 14th Day	0	0	2	0	0	0	0	0	0	1	2	1
Recovery	Severe Paralysis	0	0	3	0	1	0	0	0	0	0	4	0
	Mild Paralysis	0	0	3	0	2	0	0	1	9	0	5	1
	Delayed	0	0	1	0	0	0	0	0	0	0	1	0
	Normal	0	0	0	0	1	0	0	0	0	0	1	0
TOTAL		2	5	14	5	6	1	0	1	0	3	22	15

TABLE 29.

GROUP I: SUBGROUPS A1 AND B1.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride: The Total in each age-group in the latter series is made equal to the former.

GROUP I.- SUBGROUPS A1 & B1	AGE GROUPS									All Age-Groups	
	0-9*			10-19*			20-				
Result.	NaCl	No NaCl Actual	No NaCl Comparative	NaCl	No NaCl Actual	No NaCl Comparative	NaCl	No NaCl Actual	No NaCl Comparative	NaCl	No NaCl Comparative
Death	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery - Severe Paralysis	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery - Mild Paralysis	1	1	0.20	0	1	0.11	0	1	1.00	1	1.31
Recovery - Delayed	2	8	1.60	0	1	0.11	1	0	0.00	3	1.71
Recovery - Normal	1	11	2,20	1	7	0.78	4	4	4.00	6	6.98
TOTAL	4	20	4.00	1	9	1.00	5	5	5.00	10	10.00

* Two age-groups combined because of paucity of results.

TABLE 30.

GROUP I: SUBGROUP A2.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride: The Total in each age-group in the latter series is made equal to the former.

GROUP I - SUBGROUP A2	AGE GROUPS															All Age-Groups	
	0-4			5-9			10-14			15-19			20-				
Result	Na Cl	No NaCl Act.	No NaCl Comp.	Na Cl	No NaCl Act.	No NaCl Comp.	Na Cl.	No.NaCl Act.	No.NaCl Comp.	Na Cl	No NaCl Act.	No NaCl Comp.	Na Cl	No NaCl Act.	No NaCl Comp.	Na Cl	No NaCl Comp.
Death	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery Severe Paralysis	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery Mild Paralysis	0	1	0.28	2	7	1.48	1	0	0.00	0	0	0.00	0	0	0.00	3	1.76
Recovery Delayed	0	2	0.56	2	1	0.21	1	2	1.13	1	0	0.00	0	0	0.00	4	1.90
Recovery Normal	5	15	4.17	3	25	5.30	7	14	7.88	1	8	2.00	3	3	3.00	19	22.35
TOTAL	5	18	5.01	7	33	6.99	9	16	9.01	2	8	2.00	3	3	3.00	26	26.01

Act./Comp. = Actual/Comparative.

TABLE 31.

GROUP II: SUBGROUP B2.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride: The Total in each age-group in the latter series is made equal to the former.

GROUP I - SUBGROUP B2	AGE GROUPS										All Age-Groups	
	0-4			5-9			10-14			15-19			20-				
Result.	Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl
		Act.	Comp.		Act.	Comp.		Act.	Comp.		Act.	Comp.		Act.	Comp.		Comp.
Death	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery Severe Paralysis	0	1	0.19	1	1	0.35	0	1	0.20	0	0	0.00	0	0	0.00	1	0.74
Recovery Mild Paralysis	1	7	1.34	4	14	4.94	1	5	1.00	0	3	0.80	1	1	0.35	7	8.43
Recovery Delayed	1	6	1.15	3	3	1.06	1	5	1.00	1	2	0.53	0	2	0.70	6	4.44
Recovery Normal	7	33	6.32	16	50	17.65	7	34	6.80	3	10	2.67	6	17	5.95	39	39.39
TOTAL:	9	47	9.00	24	68	24.00	9	45	9.00	4	15	4.00	7	20	7.00	53	53.00

Act. = Actual
Comp. = Comparative.

T A B L E 32.

GROUP II: SUBGROUPS B3, B4 & B5.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride; the Total in each age-group in the latter series is made equal to the former.

GROUP II - SUBGROUPS B3, B4, B5	AGE GROUPS.												All Age-Groups	
	0-4			5-9			10-14			15- *				
Result	Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl
		Act.	Comp.		Act.	Comp.		Act.	Comp.		Act.	Comp.		Comp.
Death	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery-Severe Paralysis	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery-Mild Paralysis	0	1	0.11	0	7	0.28	0	1	0.40	1	0	0.00	1	0.79
Recovery Delayed	0	1	0.11	0	5	0.20	0	0	0.00	0	0	0.00	0	0.31
Recovery Normal	1	7	0.78	1	13	0.52	4	9	3.60	0	7	1.00	6	5.90
TOTAL:	1	9	1.00	1	25	1.00	4	10	4.00	1	7	1.00	7	7.00

* Two age-groups combined because of paucity of results.

Act. = Actual
Comp. = Comparative.

TABLE 33.

GROUP II: SUBGROUP C2.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride: the Total in each age-group in the latter series is made equal to the former.

GROUP II - SUBGROUP C2.	AGE GROUPS									All Age-Groups.	
	0-4			5-9			10- *				
		No NaCl			No NaCl			No NaCl			
	NaCl	Actual	Compar.	NaCl	Actual	Compar.	NaCl	Actual	Compar.	NaCl	No NaCl Compar.
Death	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery - Severe Paralysis	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery - Mild Paralysis	0	2	1.00	0	1	0.71	1	1	0.60	1	2.31
Recovery Delayed	0	2	1.00	1	3	2.14	0	0	0.00	1	3.14
Recovery Normal	3	2	1.00	4	3	2.14	2	4	2.40	9	5.54
TOTAL:	3	6	3.00	5	7	4.99	3	5	3.00	11	10.99

* Two age-groups combined because of paucity of results.

Compar. = Comparative.

TABLE 34.

GROUP III: SUBGROUP C3.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride. The Total in each age-group in the latter series is made equal to the former.

GROUP III SUBGROUP C3 / Result	AGE GROUPS														All Age-Groups	
	0-4			5-9			10-14			15-19			20-			
	Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl		Na. Cl	No NaCl
		Act.	Comp.		Act	Comp.		Act	Comp.		Act	Comp.		Act	Comp.		Comp.
Death	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery – Severe Paralysis	0	0	0.00	0	1	0.12	0	0	0.00	0	0	0.00	0	1	0.83	0	0.95
Recovery – Mild Paralysis	2	13	3.25	2	21	2.44	0	1	0.83	0	1	0.40	1	2	1.67	5	8.59
Recovery Delayed	0	3	0.75	1	10	1.16	0	2	1.67	0	3	1.20	0	0	0.00	1	4.78
Recovery Normal	4	8	2.00	2	11	1.28m	5	3	2.50	2	1	0.40	4	3	2.50	17	8.68
TOTAL:	6	24	6.00	5	43	5.00	5	6	5.00	2	5	2.00	5	6	5.00	23	23.00

Act. = Actual
Comp. = Comparative.

TABLE 35.

GROUP III: SUBGROUPS C4 and C5.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride. The Total in each age-group in the latter series is made equal to the former.

GROUP III SUBGROUPS C4 & C5.	AGE GROUPS															All Age-Groups	
	0-4			5-9			10-14			15-19			20-				
Result	Na Cl	No NaCl		Na Cl	No NaCl		Na Cl	No NaCl		Na Cl	No NaCl		Na Cl	No NaCl		Na. Cl	No NaCl
		Act.	Comp.		Act.	Comp.		Act.	Comp.		Act.	Comp.		Act.	Comp.		Comp.
Death	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery - Severe Paralysis	0	1	0.46	0	1	0.48	0	2	0.73	0	0	0.00	0	0	0.00	0	1.67
Recovery - Mild Paralysis.	1	5	2.31	5	9	4.33	0	5	1.82	0	0	0.00	0	0	0.00	6	8.46
Recovery Delayed	1	3	1.38	2	7	3.37	1	0	0.00	0	1	0.25	0	0	0.00	4	5.00
Recovery Normal	4	4	1.85	6	10	4.81	3	4	1.45	1	3	0.75	1	2	1.00	15	9.86
TOTAL:	6	13	6.00	13	27	12.99	4	11	4.00	1	4	1.00	1	2	1.00	25	24.99

Act. = Actual
Comp. = Comparative.

TABLE 36.

GROUP IV: SUBGROUP C6.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride. The Total in each age-group in the latter series is made equal to the former.

GROUP IV. SUBGROUP C6. Result.		AGE GROUPS 0-9* Na Cl	0-9* No NaCl Act.	0-9* No NaCl Comp.	10-14 Na Cl	10-14 No NaCl Act.	10-14 No NaCl Comp.	15- * Na Cl	15- * No NaCl Act.	15- * No NaCl Comp.	All Age Groups NaCl	All Age Groups No NaCl. Comparative
Death	1st day	0	1	0.44	0	0	0.00	0	0	0.00	0	0.44
	2nd-4th days	0	1	0.44	0	0	0.00	0	0	0.00	0	0.44
	5th-7th days	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
	8th-14th days	0	1	0.44	0	0	0.00	0	0	0.00	0	0.44
	over 14th day	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
Recovery	Severe Paralysis	0	0	0.00	0	1	0.43	1	1	1.00	1	1.43
	Mild Paralysis	4	7	3.06	2	2	0.86	0	1	1.00	6	4.92
	Delayed	0	1	0.44	1	3	1.29	0	1	1.00	1	2.73
	Normal	3	5	2.19	0	1	0.43	3	1	1.00	6	3.62
TOTAL:		7	16	7.01	3	7	3.01	4	4	4.00	14	14.02

* Two age-groups combined because of paucity of results.

Act. = Actual
Comp. = Comparative.

TABLE 37.

GROUP IV: SUBGROUP C7.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride. The Total in each age-group in the latter series is made equal to the former.

GROUP IV: SUBGROUP C7.		AGE GROUPS 0-4			5-9			10- *			All age groups	
Result.		NaCl	No NaCl Act.	No NaCl Comp.	NaCl	No NaCl Act.	No NaCl Comp.	NaCl	No NaCl Act.	No NaCl Comp.	NaCl	No NaCl Comparative
Death	1st day	0	0	0.00	0	0	0.00	0	0	0.00	0	0.00
	2nd-4th days	0	1	1.00	0	1	1.75	0	0	0.00	0	2.75
	5th-7th days	0	0	0.00	2	0	0.00	0	0	0.00	2	0.00
	8th-14th days	0	0	0.00	1	1	1.75	0	0	0.00	1	1.75
	over 14th day	1	0	0.00	0	0	0.00	0	0	0.00	1	0.00
Recovery	Severe Paralysis	0	0	0.00	1	0	0.00	0	0	0.00	1	0.00
	Mild Paralysis	0	0	0.00	3	2	3.50	1	2	0.50	4	4.00
	Delayed	0	0	0.00	0	0	0.00	0	1	0.25	0	0.25
	Normal	0	0	0.00	0	0	0.00	0	1	0.25	0	0.25
TOTAL:		1	1	1.00	7	4	7.00	1	4	1.00	9	9.00

* Two age-groups combined because of paucity of results.

Act. = Actual
Comp. = Comparative.

TABLE 38.

GROUP IV: SUBGROUP C8.

Showing comparative values of results in cases receiving extra Sodium Chloride and not receiving extra Sodium Chloride. The Total in each age-group in the latter series is made equal to the former.

GROUP IV: SUBGROUP C8	Result.	AGE GROUPS									All Age-Groups	
		0-4			5-9			10- *				
			No NaCl			No NaCl			No NaCl		NaCl	No NaCl
		Na. Cl	Act.	Comp.	Na. Cl	Act.	Comp.	Na. Cl	Act.	Comp.		Comparative
Death	1st day	2	1	0.40	1	0	0.00	0	1	0.20	3	1.60
	2nd-4th days	0	2	0.80	1	1	2.80	1	0	0.00	2	3.60
	5th-7th days	0	1	0.40	1	1	2.80	0	0	0.00	1	3.20
	8th-14th days	0	1	0.40	2	3	8.40	1	2	2.40	3	11.20
	over 14th day	0	0	0.00	2	0	0.00	0	1	1.20	2	1.20
Recovery	Severe Paralysis	0	0	0.00	3	0	0.00	1	0	0.00	4	0.00
	Mild Paralysis	0.	0	0.00	3	0	0.00	2	1	1.20	5	1.20
	Delayed	0	0	0.00	1	0	0.00	0	0	0.00	1	0.00
	Normal	0	0	0.00	0	0	0.00	1	0	0.00	1	0.00
TOTAL:		2	5	2.00	14	5	14.00	6	5	6.00	22	22.00

* Two age-groups combined because of paucity of results.

Act. = Actual
Comp. = Comparative.

TABLE 39.

Showing Results summed in each Group.
From Results in Tables 29 to 38 inclusive.

ALL GROUPS.		GROUP I		GROUP II		GROUP III		GROUP IV			
Result		Act.	Comp.	Act.	Comp.	Act.	Comp.	Act.	Comp.	Act.	Comp.
Death	1st day	0	0.00	0	0.00	0	0.00	3	2.04	3	2.04
	2nd-4th days	0	0.00	0	0.00	0	0.00	2	6.79	2	6.79
	5th-7th days	0	0.00	0	0.00	0	0.00	3	3.20	3	3.20
	8th-14th days	0	0.00	0	0.00	0	0.00	4	13.39	4	13.39
Recovery	Severe Paralysis	0	0.00	1	0.74	0	2.62	6	1.43	7	4.79
	Mild Paralysis	4	3.07	9	11.53	11	17.05	15	10.12	39	41.77
	Delayed	7	3.61	7	7.89	5	9.78	2	2.98	21	24.26
	Normal	25	29.33	54	50.83	32	18.54	7	3.87	118	102.57
TOTAL:		36	36.01	71	70.99	48	47.99	45	45.02	200	209.01

Act. = Actual.
Comp. = Comparative.

T A B L E 40.

Derived from Table 39 by integral summation according to increasing severity, showing actual results in series of cases which received extra Sodium Chloride and estimated results in series of cases which did not receive Sodium Chloride when age-groups and severity have been altered to correspond with former series.

Description of Case	Cases which received extra Sodium Chloride Actual Number	Cases which did not receive extra Sodium Chloride. Comparative Number.
Satisfactory Recovery	118	102.57
Uncomplicated Recovery	139	126.83
Recovery without severe Paralysis	178	168.60
Recovery: All Types	185	173.39
Alive after 14 days' Treatment	188	174.59
Alive after 7 days' Treatment	192	188.98
Alive after 4 days' Treatment	195	192.18
Alive after 24 hours' Treatment	197	198.97
TOTAL ADMITTED	200	200.01