A STUDY OF THE BLOOD-SUGAR

IN

CONVULSIONS

AND

OTHER DISTURBANCES OF THE CENTRAL NERVOUS SYSTEM.

By

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P R E F A C E.

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I. HISTORICAL INTRODUCTION.

Since Claude Bernard[1] in 1855 published the results of his classical piqûre experiments, much interest has been aroused in the question of hyperglycaemia and glycosuria following cerebral insult. Glycosuria has been observed in cerebral haemorrhage,[2, 3, 4, 5] tumour,[6, 7] concussion[8, 9] and meningitis, and much controversy has waged as to the method by which this phenomenon is produced. It is now generally accepted that the intracranial disturbance causes widespread stimulation of the splanchnic sympathetic with rise in the output of adrenaline and consequent hepatic glycogenolysis; Bernard himself thought that the action was a reflex through a ganglion of the sympathetic nervous system. Allied to this group of cerebral glycosurias is that of emotional glycosuria which was first noted by Böhm and Hoffman[10] in a cat excited by the barking of a dog, and later described at length by Cannon and his co-workers,[11, 12, 13] who found that the condition was due to an increased quantity of epinephrin in the cat's blood. Cannon's belief that emotional impulses acting on the adrenals excite a discharge of adrenaline which mobilises the defence mechanisms of the body, among them the conversion of glycogen into glucose, has withstood much criticism.

It was not till 1920 however, that Fischer[14] pointed out that changes occurring in the blood as a result of an epileptic fit could be reproduced by adrenaline. Kersten,[15] a year later, noted a sharp rise in the blood-sugar value following both minor and major epileptic seizures. The rise occurred within a few minutes of the onset of the convulsion and was followed by marked fluctuation before the original level was finally attained in about one-and-a-half hours. These variations in the blood-sugar Kersten attributed to a fluctuation in the activity of the adrenals. Macleod[16] noted a rise in the blood-sugar following a convulsion due to insulin overdosage. Drury and Farran-Ridge,[17] in the course of an investigation of the sugar-tolerance curve in epileptics, had the opportunity of observing the behaviour of the blood-sugar after a fit in one patient. This man took a severe seizure just after the fasting determination had been made, and within half-an-hour his blood-sugar rose to 293 mgm. per cent., falling again to 127 mgm. per cent. in one-and-a-half hours. A similar observation was made by Metcalf and Moriarty[18] in a patient who took a severe convulsion in the course of an attack of acute haemorrhagic nephritis. They came to the conclusion that the rise in blood-sugar could not be attributed to the nephritis but rather to the severe generalised disturbance of the nervous system occasioned by the convulsion, a consequent sharp increase in the output of epinephrin, and a subsequent mobilisation of

sugar from the liver. Rathery, Dérot and Sterne,[19] on the other hand, noted a low blood-sugar level (54 mgm. per cent.) in a man of forty-three years, three days after a convulsion due to meningeal haemorrhage. In the convulsions of eclampsia Titus et alia[20] noted what they termed a period of relative hypoglycaemia just before a convulsion, and a sharp rise in the blood sugar immediately following it; this latter they attributed to the increased muscular exertion and the asphyxia produced by the fit. Haury and Hirschfelder[21] noted a constant, sharp rise in the blood sugar of epileptics following a fit, the rise being roughly proportional to the severity of the seizure, and in no instance found hypoglycaemia before or during a fit.

Following the recognition of hyperinsulinism as a cause of convulsions, the possibility has been recognised that hypoglycaemia may be an aetiological factor in the production of convulsions in infancy and childhood, and there is no doubt that convulsions due to this occasionally occur.

Two cases have been recorded by Kramer, Grayzel and Solomon,[22] and Josephs[23, 24] has suggested that the combination of fever and a short fast may produce a state of hypoglycaemia which may explain the convulsions occurring at the onset of an acute infection. Other workers, however, while noting the presence of hypoglycaemia in the post-convulsive state have hesitated to attribute the seizure to the disturbance

in carbohydrate metabolism. Thus, Griffiths[25] recorded the association of hypoglycaemia with convulsions but could not decide whether the relationship was one of cause or effect. Peterman[26] was unable to establish a diagnosis of hypoglycaemia in any of his five hundred cases of convulsions in children, and Brown[27] in four hundred cases found hypoglycaemia only once, in a child who was admitted to hospital comatose, having had a convulsion the previous day. Higgons[28] observed hypoglycaemia following convulsions in two new-born babies. Fleming, Herring and Morris[29] reported three patients who were admitted to hospital comatose and had either glycosuria or a raised blood-sugar, which in two of them was followed by hypoglycaemia, and Darrow[30] recently reported two examples of recurring convulsions in mentally defective children in whom hypoglycaemia was frequently noted but on two occasions shortly after a convulsion, the blood sugar was found to be high. It was found that when these children were drowsy and hypoglycaemic, intravenous glucose produced no marked improvement, an observation which had previously been made by Josephs and Griffiths in at least one case each.

The suggestion that some disturbance of carbohydrate metabolism might be an aetiological factor in "idiopathic" epilepsy led many workers to investigate the sugar tolerance in this condition. Daly, Pryde and Walker[31] thought there might be some relationship between low blood-sugar values and

the onset of epileptic seizures but found the results inconclusive and Shaw and Moriarty,[32] though they found low fasting blood-sugar values in epileptic children, did not think that this was the result of the epileptic tendency. Holmstrom[33] and Mann[34] came to the conclusion that carbohydrate tolerance was normal in epilepsy, and Drury and Farran-Ridge[17] found that the blood-sugar curves approximated closely to the normal in fourteen of fifteen epileptics examined. Noting an increase in the incidence of epilepsy in Russia at a time when sugar was scarce Wladyczko[35] endeavoured to treat some epileptics with sugar, and believed that certain cases benefited thereby. Goodall,[36] on the other hand, was convinced that epileptic fits are not brought on by hypoglycaemia and found no consistent difference in the amount of sugar in the blood either before, during, or after a fit. Lennox and his co-workers[37, 38] investigating a large series of patients came to the conclusion that the great majority of epileptics presented no disturbance of carbohydrate metabolism, and that any changes in the sugar content of the blood following a convulsion were merely "passive adjustments" to physiological conditions associated with the muscular exertion involved. A theory that certain patients with epileptiform seizures suffered from a dysinsulism or hyposuprarenalism was postulated by Nielsen and Eggleston[39] who claimed to have cured the convulsions by the oral administration of dried suprarenal gland. Investigating the sugar tolerance curves of sixty-six epileptics MacKay and Barbash[40]

found that over forty per cent. of these patients belonged to what they called the markedly subnormal group, while Munch-Petersen and Schon[41] found in a large series of epileptic patients that the fasting blood-sugar corresponded closely to the normal. Minchin[42], on the other hand, felt convinced that hypoglycaemia was an important if not essential factor in the production of a large proportion of epileptic convulsions, but Bouche[43] reporting a case of epilepsy in which the blood sugar was constantly low demonstrated that the association was mere coincidence. Attacking the problem from the therapeutic angle Baudouin and his co-workers[44] administered ten units of insulin intravenously to sixteen epileptic patients. The fasting blood-sugar levels were normal except in one case where the value was 62 mgm. per cent.; the blood-sugar fell following the insulin but in no case was a fit precipitated, though slight symptoms of hypoglycaemia such as sweating and hunger were common. Nielsen[45] found that ninety per cent. of patients with idiopathic epilepsy showed a tendency to hypoglycaemia though he did not consider that hypoglycaemia alone was sufficient to cause an attack, and Tyson et alia[46] in a recent paper noted a tendency in epileptics as a whole, towards subnormal fasting blood-sugar values.

In view of the confusion of opinion regarding the role of hypoglycaemia in the convulsions of childhood, the following investigation was undertaken. Interest in the

question was particularly aroused by the observation in this department of three cases of coma with glycosuria not due to diabetes mellitus, which were reported by Fleming, Herring and Morris.(29)

II. PRESENT INVESTIGATION.

A. STUDY OF THE BLOOD-SUGAR IN CONVULSIONS.

Blood-sugar estimations were made in a series of seventy-four children admitted to hospital convulsing or with a history of recent convulsions, and an attempt was made to follow the changes occurring in the blood-sugar level from the beginning of a convulsion till some hours after it had ceased. It will be understood that it was impossible in many cases to obtain as complete a record of the fluctuation in the blood-sugar as one would have wished, since frequently the child was not brought to hospital till some hours after the convulsion. Unfortunately, too, it was not possible, except in the case of very minor spasmophilic fits, to obtain a blood-sugar reading immediately before a convulsion. The patients were of ages ranging from two days to eleven-and-a-half years and the seizures were due to various causes such as birth injury, spasmophilia, meningitis, epilepsy or acute infection, some, however, for which no cause could be found, being classed as 'idiopathic convulsions'. The blood-sugar was estimated by the method of Folin and Wu modified by Herbert and Bourne.(47)

The blood obtained by pricking the ear or the heel with a Hagedorn needle was collected in a small tube containing a minute quantity of potassium oxalate and sodium fluoride. The oxalate prevented clotting and the fluoride inhibited glycolysis. The proteins of the blood were precipitated by sodium tungstate and sulphuric acid and the protein-free filtrate heated with an alkaline copper solution, using a special tube to prevent re-oxidation. The cuprous oxide formed was treated with phospho-molybdic acid solution, a blue colour being obtained which was compared with that of a standard.

1. Illustrative Cases.

Seventy-four patients were examined and of these four were examined on two separate occasions. The blood-sugar was estimated at frequent intervals, every half hour when the child was seen soon after the onset of the convulsion and thereafter hourly.

The following typical case presents many interesting features.

Case 3. E.F., a girl aged $1^{6}/12$ years, was delivered by Caesarean section and appeared healthy at birth. She throve and developed normally and had always been healthy except for an attack of chicken-pox six months before admission. During the night before admission to hospital she vomited once and slept badly. At 6 a.m. on the day of admission she was found unconscious and from then until she was admitted to hospital had frequent attacks of generalised rigidity each lasting for about five minutes. On admission at 8.30 a.m. she was still unconscious and breathing stertorously. She was a small pale child and on examination no abnormality was found in heart, lungs or abdomen. The knee-jerks were active and the plantar responses extensor. The rectal temperature was 101.8°F. She recovered consciousness at noon but remained drowsy all day. Next day she appeared quite well. The blood-sugar content was estimated frequently throughout the day.

The urine obtained at 10 a.m. contained neither sugar nor acetone.

Time	8.45 a.m.	9.15	9.45	10.15	10.45	11.15	11.45	12.15 p.m.	12.45	1.15	2.15	2.45	3.15	4	4.30	5
Blood-Sugar in mgm.%	232.6	158.7	144.9	102.0	92.6	88.5	74.6	108.7	113.6	113.6	84.7	71.9	58.1	49.5	62.5	42.8
No. of hours after convulsion.	$2\frac{3}{4}$	$3\frac{1}{4}$	$3\frac{3}{4}$	$4\frac{1}{4}$	$4\frac{3}{4}$	$5\frac{1}{4}$	$5\frac{3}{4}$	$6\frac{1}{4}$	$6\frac{3}{4}$	$7\frac{1}{4}$	$8\frac{1}{4}$	$8\frac{3}{4}$	$9\frac{1}{4}$	10	$10\frac{1}{2}$	11

It will be seen that following the convulsion there was a period of definite hyperglycaemia though this apparently was not of a height sufficient to cause glycosuria. Kersten,[15] quoted above, made a similar observation, but in his cases the blood-sugar returned to normal levels in about one-and-a-half hours, after which he did not continue his investigations. The steep rise in the blood-sugar was also noted by Drury and Farran-Ridge[17] and Metcalf and Moriarty.[18] The other salient feature of this case is the low level to which the blood-sugar had sunk eleven hours after the convulsion. Further readings of this child's blood-sugar are not reported at present since they involve an investigation to be discussed later. Of the hypoglycaemia which undoubtedly existed there were few symptoms as, except for slight drowsiness and irritability, the child appeared moderately well. At 9 a.m. the next morning the blood-sugar was still rather low, 65.8 mgm. per cent., and remained so throughout the day but by next day it had risen to 89.3 mgm. per cent.

The following cases further illustrate these points.

Case 19. E.G., a girl aged $1\frac{11}{12}$ years, was admitted to hospital on July 10, 1935. She had been a normal, full-time baby and was well till three days before admission when she was noticed to be constipated and irritable. At 3 p.m. on the day of admission she suddenly became unconscious with rigid limbs, staring eyes, frothing at the mouth and some vomiting.

On admission to hospital two hours later she was found to be a moderately well-nourished child, deeply cyanosed and unconscious. The right arm and leg were held rigid and showed occasional twitching. The pupils were dilated and fixed. The

knee-jerks were active but the abdominal and plantar reflexes could not be elicited. Chvostek's sign was absent. The heart, lungs and abdomen showed no abnormality. The urine contained neither albumin nor sugar. Lumbar puncture revealed the cerebro-spinal fluid under increased pressure, clear and containing three cells per c.mm. Pandy's test was negative. T. 103°F., P. 132, R. 28.

The next morning the child appeared quite well though rather irritable.

The blood-sugar content, high on admission, fell gradually till twenty-four hours later it was only 36.7 mgm. per cent.

Urine obtained by catheterisation at 1 a.m. on 11/7/35 contained acetone but no sugar.

	←--------10/7/35----------→					←--11/7/35------→				
Time	5 p.m.	6 p.m.	7 p.m.	8 p.m.	9 p.m.	11 p.m.	12 m.n.	9 a.m.	noon	5 p.m.
Blood-Sugar. mgm.%	250.0	204.1	125.0	71.2	56.2	52.3	52.0	42.6	55.5	36.7
No. of hours after convul-sion.	2	3	4	5	6	8	9	18	21	26

The fasting blood-sugar forty hours after admission was normal (82.6 mgm. per cent.) but the blood sugar curve after 9 gms. glucose showed rather a high rise and a delayed fall.

Fasting.	½ hour.	1 hour.	1½ hours.	2 hours.
82.6	188.6	153.8	126.5	101.0 mgm.per 100 c.c.

A skiagram of the skull showed a suggestion of 'paw-marking' in the parietal and occipital regions. The sutures did not appear to be separated. Convalescence was uneventful and the child was dismissed well.

Four months later the child was readmitted comatose seven hours after a convulsion. She had been well till four days before admission when she took a slight attack of diarrhoea. At 11.30 a.m. on the day of admission she suddenly became stiff all over and later began to have twitching movements of the left side of the face and body. On examination about 6.30 p.m. she was found to be comatose but the pupils were equal and active. The plantar responses were doubtfully extensor and other reflexes were present on the right side but could not be elicited on the left side. An hour later she was recovering consciousness and there was a definite left-sided paresis with an extensor plantar response. The urine obtained by catheterisation contained a trace of sugar and abundant acetone.

	←8.11.35→			←9.11.35→		
Time	6.30 p.m.	7.30 p.m.	9.30 p.m.	10.30 p.m.	11.30 p.m.	10.30 a.m.
Blood-Sugar. mgm.%	49.5	52.3	59.5	59.5	53.7	40.9
No.of hours after convulsion.	7	8	10	11	12	23

A close parallel may be traced between the course of the blood-sugar following the convulsion on each of the two admissions to hospital. The glycosuria observed on the second occasion is strong presumptive evidence of the occurrence of just such a transient hyperglycaemia as was noted on the first occasion, especially since sugar was never again found in the urine during the remainder of the child's stay in hospital. It will be seen that in both instances the blood-sugar was at a low level six to eight hours after the convulsion and that the hypoglycaemia tended to persist throughout many hours in spite of the admin-

istration of carbohydrate in the form of milk and sugar. Of this child's subsequent history more will be said later.

Case 23. A.B., a boy aged $2^{3}/12$ years. The early history was uneventful. Five days before admission the child developed fever, abdominal pain and vomiting. Five hours before admission he had a convulsion lasting two-and-a-half hours but he had recovered consciousness on admission. Symptoms and signs were vague till three days later when at 8 p.m. he took a convulsion which continued in spite of chloral till a lumbar puncture was performed at 10.30 p.m. The cerebro-spinal fluid contained 587 cells per c.mm., of which sixty-five per cent. were lymphocytes. The protein was markedly increased and tubercle bacilli were found in the pellicle which formed during the night, thus establishing beyond question the diagnosis of tuberculous meningitis. Urine obtained at 10.30 p.m. contained sugar and acetone.

←----------20/6/36----------→ ←--21/6/36--→

Time	8.15 p.m.	8.30 a.m.	9 p.m.	10 p.m.	11 p.m.	1 a.m.	2 p.m.
Blood-Sugar. mgm.%	256.4	434.8	264.7	200.0	145.1	71.9	73.2
No.of hours after convul-sion.	¼	½	1	2	3	5	18

Here again a period of marked hyperglycaemia was observed, followed by a sharp fall to a relatively low level within five hours of the convulsion. Unfortunately no determinations of the blood-sugar were made between 1 a.m. and 2 p.m. the next day. There can be little doubt that the sharp disturbance of the blood-sugar was the direct outcome of the convulsion; that the

transition from high to low levels may take place rather suddenly is further illustrated by the following case.

Case 16, D.McL., a boy aged $5^{9}/12$ years. He had been a puny baby and had never thriven well. At $1^{6}/12$ years he developed a tuberculous affection of the left elbow and spent two years in a sanatorium. Thereafter he was fairly well till about two weeks before admission when he took severe rhinitis. About 4.30 p.m. on the day of admission he took a convulsion which lasted for about four hours. He was admitted at 7.30 p.m. and on examination was found to be a neglected and much emaciated child with many sores, unconscious and rigid. Nothing abnormal was discovered in the heart, lungs or abdomen. The pupils were unequal and fixed. The knee-jerks were active and the plantar responses extensor but the abdominals were not obtained. The urine a few hours after admission contained a trace of albumin and acetone but no sugar, and microscopically abundant pus cells.

20.12.35

←--------18/12/35---→ ←----------19/12/35----------→ < >

	7.30 p.m.	8.30 p.m.	9.30 p.m.	10.30 p.m.	10 a.m.	11 a.m.	12 noon	1 p.m.	3 p.m.	4 p.m.	5 p.m.	10 a.m.
Blood-Sugar. mgm.%	188.7	50.0	44.0	57.4	63.8	66.6	54.3	46.9	77.5	52.6	44.6	47.6
No.of hours after convul-sion.	3	4	5	6	$17\frac{1}{2}$	$18\frac{1}{2}$	$19\frac{1}{2}$	$20\frac{1}{2}$	$22\frac{1}{2}$	$23\frac{1}{2}$	$24\frac{1}{2}$	$41\frac{1}{2}$

The persistent low levels throughout the day following the convulsion are well seen here. The association of acetonuria with a low blood sugar is also a feature.

Two months later the child was readmitted many hours after a similar convulsion and when seen was conscious but drowsy. Apart from pus cells nothing abnormal was detected in the urine.

Time	12 noon	1 p.m.	3 p.m.	4 p.m.
Blood-Sugar. mgm.%	36.5	46.3	47.6	55.5
No. of hours after con-vulsion.	18	19	21	22

There can be little doubt that with this second convulsion there had been a rise in the blood-sugar similar to that observed following the first. Further readings on this patient will be reported later.

2. General Results.

Seventy-four children were examined in the course of this investigation and four of these on two separate occasions.

Thirty-nine of the patients were seen within three hours of the onset of the convulsion and of these sixteen showed definite hyperglycaemia, this being observed on two occasions in one case. In addition, three children seen soon after the onset, had glycosuria though the actual hyperglycaemic value was missed; thus 49 per cent. of the children seen within three hours of the convulsion showed evidence of a sharp rise in the sugar-content of the blood. A blood-sugar level of more than 180 mgm. per cent. was taken as indicating hyperglycaemia, and one of less than 50 mgm. per cent. as hypoglycaemia. Twenty-seven cases, i.e. 36 per cent., showed hypoglycaemia at some point during the investigation and in two patients this was observed on two occasions.

TABLE I.

Number of Cases		Number showing Hyper- * glycaemia	No. showing Hypoglycaemia		Total No. of Cases showing Disturbance of the Blood-Sugar.
			No. showing Hyperglycaemia & Hypoglycaemia	No. showing only Hypo-glycaemia.	
No. seen within 3 hours of onset.	39	19	6	3	22
No. seen after 3 hours.	35	3	1	17	20
Total	74	22	7	20 (27)	42
Percentage		30%	36%		57%

* This group includes those in whom the actual hyperglycaemic stage was missed but who had glycosuria.

Table II shows the early blood-sugar readings from the patients seen within three hours of the onset of the convulsion.

TABLE II.

No.	Name	Age in years	BLOOD-SUGAR IN MILLIGRAMS PER 100 c.c.							Disease.
			15 mins. after	30 mins. after	1 hour after	1½ hours after	2 hours after	2½ hours after	3 hours after	
1	M.G.	$10\frac{1}{2}/12$			250.0		144.9			Tub.meningitis.
2	D.McL.	$5^{9}/12$							188.7	Tub.kidney.
3	H.K.	$2^{1}/12$							317.4	Idiopathic convulsion.
4	M.McL.	$4^{8}/12$			303.0	270.0	476.1	312.5		Tub.meningitis.
5	W.M.	$2^{3}/12$			85.4	133.3	100.0	109.8	91.6	Idiopathic convulsion.
6	M.M.	$8^{6}/12$	76.3	78.1		88.5	94.3			Uraemia.
7	J.K.	$4^{9}/12$					144.9			Idiopathic convulsion.
8	A.M.	$5^{8}/12$		277.7	238.1					Anaemia.
9	A.M.	$3^{2}/12$		144.9	144.9	250.0	303.0	119.0	135.7	Tub.meningitis.
10	H.McV.	$1^{6}/12$	72.5	69.4	78.1		59.5			M.D.
11	C.N.	$1^{10}/12$		109.9		85.4				Gastro-enteritis
12	E.W.	$^{4}/12$	96.1	98.0	90.9		97.1			Tub.meningitis.
13	C.McA.	$^{14}/52$	204.1		178.6	156.2	162.6	196.1	204.1	Anaemia.
14	J.R.	$^{7}/12$	103.1						33.0	Ileo-colitis.
15	J.W.	2		250.0	250.0		169.5		188.7	Tub.meningitis.
16	E.M.	(a) $2\frac{5}{12}$		80.6	88.5	119.0	156.2	188.6	196.1	(Miliary (tubercu-
		(b)	135.1		188.7	96.1	144.9	126.5	126.5	(losis.
17	D.R.	$8^{8}/12$					129.8	147.6	156.2	Epilepsy.
18	M.H.	11	86.9	87.9	71.9	102.0				Thoracic tuberculosis.

TABLE II (contd.).

No.	Name	Age in years	BLOOD-SUGAR IN MILLIGRAMS PER 100 c.c.							Disease.
			15 mins. after	30 mins. after	1 hour after	1½ hours after	2 hours after	2½ hours after	3 hours after	
19	J.C.	1 3/12	131.6		151.5		158.7			Tub.meningitis.
20	D.McR.	3			90.9		78.1			M.D.
21	A.H.	11 6/12	140.8		125.0		95.2			Epilepsy.
22	E.F.	1 6/12						232.6		Idiopathic convulsion.
23	T.B.	10/12			149.7					Pneumonia.
24	T.H.	1 2/12			154.3					Tub.meningitis.
25	D.F.	2 8/12					125.0		102.0	Tub.meningitis.
26	D.McL.	1 1/12						81.3		Bronchitis.
27	J.D.	2							232.6	Idiopathic convulsion.
28	M.G.	5			218.5		169.5		156.3	Strept.meningitis
29	C.T.	4	65.3	68.5	65.3		54.3		65.3	M.D.
30	A.B.	1 5/12					70.9			Pneumonia.
31	A.B.	2	256.4	434.8	264.7		200.0		145.1	Tub.meningitis.
32	T.W.	8/12							149.2	P.C. meningitis.
33	C.O.	11	101.0	95.2				102.2		Epilepsy.
34	E.G.	1 11/12					250.0		204.1	Idiopathic convulsion.
35	C.S.	1 10/12						86.2		Ileo-colitis.
36	M.B.	9							256.4	Uraemia.
37	C.S.	1 3/12						78.1		Bronchitis.
38	R.D.	5						96.1		Tonsillitis.
39	M.B.	2 5/12							312.5	Idiopathic convulsion.

Table III shows the maximum and minimum blood-sugar values of all the cases during four periods - (1) from the onset till three hours; (2) from the fourth till the twelfth hour; (3) from the thirteenth till the twenty-fourth hour; and (4) from the twenty-fifth till the forty-eighth hour.

(See TABLE III overleaf).

From a study of these figures it would appear that in a considerable number of cases of convulsions there is a profound disturbance of the mechanism controlling the blood-sugar so that during or immediately after the convulsion the percentage of the sugar in the blood is markedly raised. This rise is frequently followed by a fairly rapid fall, sometimes to very low levels, which may persist for hours or days. It is convenient to speak of a hyperglycaemic phase and a subsequent hypoglycaemic phase.

TABLE III.

Maximum and Minimum Blood-Sugar Readings after Convulsions.

No.	Name	Age years	0-3 hours		4-12 hours		13-24 hours		25-48 hours		Diagnosis.
			Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	
1	M.G.	$10\frac{1}{2}$/12	250.0	144.9	98.8	81.9	112.3				Tuberculous meningitis.
2	H.K.	2^{1}/12	322.6	317.4	253.2	89.3	111.1	87.7	69.5		Idiopathic convulsion.
3	E.F.	1^{6}/12	232.6		158.7	42.8					Idiopathic convulsion.
4	W.McK.	18/52			158.7	105.2	121.9	80.6			Nasal diphtheria.
5	B.T.	2/365			39.8		62.2	45.8	90.1	67.1	Intracranial birth injury.
6	T.B.	10/12	149.7		88.5	81.3	136.9				Broncho-pneumonia.
7	A.M.	5^{8}/12	277.7	238.1	152.6						Anaemia.
8	M.McL.	4^{8}/12	476.1	270.0	104.1	91.8	112.3	75.2	144.9	91.8	Tuberculous meningitis.
9	G.W.	1^{1}/12			172.3		78.1	71.9	75.2	49.7	Tetany.
10	A.P.	18/52			21.0	3.7					Gastro-enteritis.
11	D.R.	8^{8}/12	156.2	129.8	116.2	90.9	82.2	71.9			Epilepsy.
12	M.C.	12/365			88.5	49.2	49.2	24.8	57.8	27.1	Intracranial birth injury.
13	C.McA.	14/52	204.1	156.2	204.1	72.9					Anaemia.
14	D.F.	2^{8}/12	125.0	102.0	89.3	85.4	93.4		135.1		Tuberculous meningitis.
15	A.G.	3/365							50.2	42.0	Intracranial birth injury.

TABLE III (contd.).

No.	Name	Age years	0-3 hours		4-12 hours		13-24 hours		25-48 hours		Diagnosis.
			Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	
16	D.McL.	$5\frac{9}{12}$ (a)	188.7		57.4	44.4	77.5	46.9	47.6	44.6	(Tub. kidney.
		(b)					55.5	36.5			(? Cerebral tuberculoma.
17	J.McL.	$11/52$					135.1	90.9	89.3	62.1	Tetany.
18	J.H.	$5/52$					94.3	56.2	73.0	23.6	Gastro-enteritis.
19	E.G.	$1\frac{11}{12}$ (a)	250.0	204.1	125.0	52.0	55.5	42.6	36.7		(Idiopathic convulsions.
		$2\frac{2}{12}$ (b)			59.5	49.5	40.9				
20	C.N.	$1^{10}/12$	109.9	85.4	54.6	44.4	27.0	22.3			Gastro-enteritis.
21	J.W.	$2/12$	250.0	169.5							Tuberculous meningitis.
22	T.M.	$3/12$			90.1	55.8	90.1	78.1	102.0	56.5	Tetany.
23	A.B.	$2^{3}/12$	434.8	145.1	71.9		73.2		88.5		Tuberculous meningitis.
24	C.T.	4	68.5	54.3	73.0	65.3					Mental deficiency.
25	E.O'N.	$5^{9}/12$			117.6	80.6	66.6	43.5	45.2	42.2	Encephalitis.
26	J.A.	1			101.0		78.1	54.9	88.5		Bronchitis.
27	E.M.	(a)	196.1	80.6	232.5	126.5	138.8	59.1	119.0	52.6	(Miliary Tuberculosis.
		(b)	188.7	96.1	178.6	60.6	97.1	95.2			(Tub. meningitis.
28	W.M.	$3\frac{3}{12}$ (a)	133.3	85.4	77.5	36.3			66.6		(Idiopathic convulsion.
		$3\frac{7}{12}$ (b)	133.3	81.3	149.2	75.2	56.2		82.6	42.9	
29	A.M.	$3^{2}/12$	303.0	119.0							Tuberculous meningitis.
30	C.S.	$1^{10}/12$	86.2	77.5	86.9	76.3	82.8	66.6			Ileo-colitis.

TABLE III (contd.).

No.	Name	Age years	0-3 hours		4-12 hours		13-24 hours		25-48 hours		Diagnosis.
			Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	
31	D.M.	1 11/12			100.0	93.4	140.8	66.2	113.6	91.7	Tonsillitis.
32	J.K.	4 9/12	144.9		52.1		49.5	36.5			Idiopathic convulsion.
33	C.O.	11	102.2	95.2	97.1	91.7					Epilepsy.
34	E.S.	8/52			63.3		133.3	87.7			Tetany.
35	H.McV.	1 6/12	78.1	59.5	86.9	69.9					Mental deficiency.
36	J.G.	2			31.2	11.2					Broncho-pneumonia.
37	A.H.	11 6/12	140.8	95.2							Epilepsy.
38	A.McM.	12/52			64.9	60.9	144.9	56.2	109.9	99.0	Tetany.
39	J.R.	7/12	103.1	33.0	12.9						Ileo-colitis.
40	J.C.	1 3/12	158.7	131.6	120.5	102.0					Tuberculous meningitis.
41	R.D.	6½	96.1		95.2	95.2	105.3	78.1	109.2	80.0	Tonsillitis.
42	T.W.	8/12	149.2		59.5	50.5					Pneumococcal meningitis.
43	J.J.	8/52					86.6	44.2	83.3	59.0	Tetany.
44	J.W.	6 10/12			120.5	68.9	89.3		112.3	85.5	Epilepsy.
45	M.G.	5	218.5	156.3			76.4		72.5		Streptococcal meningitis.
46	J.C.	2/365					46.5				Intracranial birth injury.
47	C.S.	1 3/12	80.0	78.1	102.0	87.7			79.4		Bronchitis.
48	O.M.	1/12			50.0	23.7	66.6				Intracranial birth injury.
49	P.C.	7/52			95.2	61.7	119.0	90.1	108.7	79.4	Tetany.
50	M.G.	4			11.7	8.5					Ileo-colitis.
51	D.W.	2 3/12			88.5	75.7	63.6	45.0	61.7	54.1	? Cerebral tuberculoma.

TABLE III (contd.).

No.	Name	Age years	0-3 hours		4-12 hours		13-24 hours		25-48 hours		Diagnosis.
			Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	Max. mgm.%	Min. mgm.%	
52	D.McL.	1 1/12	81.3		59.5		112.3	81.9			Bronchitis.
53	M.H.	11	102.0	71.9	94.7	78.1	74.0				? Cerebral tuberculoma.
54	T.H.	1 2/12	154.3		91.7	78.1	82.9		73.7		Tuberculous meningitis.
55	M.McA.	9/52			40.1	23.9					Gastro-enteritis.
56	M.M.	8½	94.3	76.3			133.2	63.7			Uraemia.
57	E.W.	4/12	98.0	90.0							Tuberculous meningitis.
58	J.S.	6 4/12			120.5	82.6					Pneumonia.
59	M.B.	2 5/12	312.5		259.7	63.2					Idiopathic convulsion.
60	D.N.	3/365							25.2		Intracranial birth injury.
61	A.B.	1 5/12	70.9		30.8	15.1					Pneumonia.
62	C.McG.	1 5/12			67.1	60.2	54.0				Tonsillitis.
63	M.G.	4/12							111.1	55.8	Tuberculous meningitis.
64	J.D.	2	232.6		52.9	39.7					Idiopathic convulsion.
65	M.McL.	3/12			19.7						Gastro-enteritis.
66	W.M.	27/52			104.1	92.6			112.3		Tetany.
67	O.McC.	3 11/12					92.6	81.9	142.8	70.4	German measles.
68	V.C.	6/52			21.0	18.8					Gastro-enteritis.
69	D.McR.	3	90.9	73.0							Mental deficiency.
70	M.O'H.	10					78.7	69.4	97.1	58.5	Nephritis.
71	M.B.	9	256.4								Uraemia.
72	I.G.	5 3/12					57.8	47.1			Tonsillitis.
73	W.McD.	6/12					91.7	63.3			Tetany.
74	E.D.	6/52					61.7	51.5			Tetany.

3. The Hyperglycaemic Phase.

It seems probable that the sharp rise in the blood-sugar level is due to the profound disturbance of the central nervous system occasioned by the convulsion, and that this causes mobilisation of glucose from glycogen - a condition similar to that produced by Bernard's diabetic puncture. The question of the actual nerve mechanism involved in the production of the hyperglycaemia following piqûre has excited much experimental work over a period of years and many conflicting results have been obtained. Thirty-six years ago Blum[48] suggested that piqure influenced the adrenals causing them to discharge adrenaline, and a little later Mayer[49] noted that glycosuria failed to appear following piqûre in twenty-five rabbits who had previously undergone an operation for the removal of both adrenals. Wertheimer and Battez[50] were rather inclined to support the contention that the adrenals were involved, but Freund and Marchand,[51] and later Stewart and Rogoff,[52] emphatically denied such a possibility. The experiments of Freund and Marchand were done on adrenalectomised rabbits but since they were performed under ether anaesthesia their results are questionable. Stewart and Rogoff performing piqûre on five adrenalectomised rabbits obtained a significant rise in the blood sugar in only two cases, but from these results decisively abandoned the hypothesis that piqûre hyperglycaemia was due to increased liberation of adrenaline into the blood stream. That there is, in fact, a large increase in

medulli-adrenal secretion in consequence of piqûre was definitely demonstrated by Carrasco-Formiguera[53] by experimental work on denervated hearts. This supported the earlier testimony of Kahn[54, 55] that the actual epinephrin content of the adrenals was diminished by piqûre and that after piqûre the adrenal medulla showed histological changes similar to those observed in other glands after secretory activity. Trendelenburg[56] thought that the effect on the adrenals was sufficient cause for the glycosuria produced while Bulatao and Cannon[57] and other workers[58] came to the conclusion that while an increased concentration of adrenaline in the blood was not perhaps the only factor, it was probably the major one. Houssay and Molinelli[59] verified the existence of such a hyperadrenalinaemia following piqûre by the method of suprarenojugular anastomosis. In more recent years Vidal[60] convinced himself that, at least in rabbits, the presence of the adrenal capsules was necessary for the appearance of hyperglycaemia following piqûre, but Hatano[61] in the same animals noted a slight and transient rise in the blood-sugar level following piqûre even in the absence of the adrenals.

Though the controversy still wages it is now fairly generally accepted that the adrenals play the major part in the production of the phenomenon. It seems reasonable therefore to postulate, in these patients in whom a hyperglycaemia was found following a convulsion, a sudden increase in the amount of circulating adrenaline occasioned by the profound

disturbance of the whole nervous system; such an increase in the amount of adrenaline in the organism would immediately initiate glycogenolysis and rapidly raise the level of the sugar in the blood. This sequence of events is analogous to the "emotional hyperglycaemia" of Cannon[12] and Britton[62] and has already been suggested by Fischer and Kersten.[15]

The question immediately arises - why did not all these patients with convulsions show a disturbance of the blood-sugar?

Firstly it is clear that in several cases the hyperglycaemic phase was missed since the child was not brought soon enough to hospital. Of the thirty-nine children examined within three hours of the onset of the convulsion, sixteen had definite hyperglycaemia and three others showed glycosuria; in addition three of the thirty-five children not seen till many hours after the convulsion, had glycosuria of varying degree. Since none of these children were observed to have sugar in the urine at any time during their subsequent stay in hospital, the inference is strong that there had been a sharp rise in the blood-sugar level, which had been missed, and which had resulted in an overflow of sugar into the urine. Thus twenty-two children had definite evidence of a sudden and transient increase in the amount of sugar in the blood.

Considering again the patients seen soon after the onset of the convulsion (Table II), it will be noticed that in eight cases the blood sugar was over 130 mgm. per cent. Of

the remaining fifteen the initial reading was under 70 mgm. per cent. in only one. Since it was impossible in many of these cases to say whether food had recently been taken, the moderately high values of between 130 mgm. per cent. and 180 mgm. per cent. could not be attributed definitely to the effect of the convulsion, but it is permissible to conjecture here also, whether one had not missed a sudden and very transient rise in the blood-sugar level.

In the light of the claims of Josephs[23, 24] and others[63, 64, 65] that hypoglycaemia plays a large part in the production of convulsions in infancy and childhood, it is significant to note that of the first readings made after the convulsion the lowest was 65.3 mgm. per cent.

4. The Influence of Various Factors on the Occurrence and Degree of Blood-Sugar Disturbance.

(a) The severity and length of the convulsion.

A factor which appeared to have a direct bearing upon whether or not there was a disturbance of the blood-sugar was the character of the convulsion. Table IV shows the length and degree of severity of the convulsion in those patients showing hyperglycaemia or glycosuria, and Table V similar observations in the remainder of the patients seen within three hours and in whom hyperglycaemia was not detected. It will be seen that the average length of the convulsion in the first group is just over three times the length of that in the second group, and on the whole the convulsions in the first group were more severe than those in the second. There was no significant difference in the average age. In experimental work on pressure on the central nervous system and its relation to hyperglycaemia Tychowski and Crowell[66] postulated a 'stimulation threshold' and such a hypothesis would adapt itself well to the results of this investigation. Certainly where the convulsion was very short and mild, one found no significant disturbance of the blood-sugar. Similar results were obtained by Britton[62] in his work on the medulli-adrenal influence in emotional hyperglycaemia in animals; he found that larger increments in the blood-sugar in normal animals followed the longer period of excitation. Cannon and Rapport[67] from experimental evidence located the reflex centre for

TABLE IV.

Length and character of the convulsion in patients seen within three hours of the onset and showing hyperglycaemia or glycosuria.

No.	Name	Age in years	Length of convulsion. (hours).	Character of Convulsion.				
				Severe	Tonic	Clonic	Generalised	Followed by coma.
1	M.G. ♂	10½/12	1½	+	+	+	+	+
2	D.McL.	5⁹/12	3	+	+	+	+	+
3	H.K.	2¹/12	2	+	+	±	+	+
4	M.McL.	4⁸/12	1	+	+	+	+	+
5	A.M. ♂	5⁸/12	½	+	+	+	L.	+
6	E.M.(a)	2⁵/12	5	+	+	+	+	+
	(b)		2	+	+	+	+	+
7	A.M. ♀	3²/12	1	+	+	+	+	+
8	C.McA.	14/52	2	+	+	+	R.	+
9	E.F.	1⁶/12	3	+	+	+	+	+
10	E.G.	1¹¹/12	2	+	+	±	R.	+
11	J.W.	2	1	+	+	+	L.	+
12	A.B. ♂	2	2½	+	+	+	R. L.	+
13	M.B.	2⁵/12	3	+	+	+	+	+
14	J.D.	2	3	+	+	+	R.+ L.	+
15	M.G. ♀	5	½	-	+	+	L.	±
16	M.B.	9	3½	+	+	+	+	+
17	J.K.	4⁹/12	1	+	+	+	+	+
18	W.M.	2³/12	2	+	+	+	L.	+
19	T.H.	2	1	+	+	+	+	+

Average length of convulsion = 2 hours 8 minutes.
Average age = 3⁴/12 years.

TABLE V.

Length and character of the convulsion in patients seen within three hours of the onset and not showing hyperglycaemia.

No.	Name	Age in years	Length of convulsion.	Character of Convulsion.				
				Severe	Tonic	Clonic	Generalised	Followed by coma.
1	M.M.	8	15 mins.	-	+	+	+	+
2	J.C.	$1^{3}/12$	15 mins.	-	+	+	+	-
3	D.R.	$8^{8}/12$	15 mins.	-	+	+	R.	-
4	A.H.	$11^{6}/12$	2 mins.	-	+	+	R.	-
5	C.N.	$1^{10}/12$	10 mins.	+	+	+	+	+
6	D.F.	$2^{8}/12$	2½ hrs.	+	+	+	R.	+
7	D.McL.	$1^{1}/12$	1 hr.	-	+	+	+	-
8	T.W.	$^{8}/12$	1 hr.	+	+	+	+	+
9	H.McV.	$1^{6}/12$	1 min.	-	+	+	+	-
10	M.H.	11	15 mins.	-	+	-	+	-
11	C.O.	11	5 mins.	-	+	+	+	-
12	T.B.	$^{10}/12$	½ hr.	+	+	+	+	+
13	C.T.	4	1 min.	-	+	+	+	-
14	R.D.	6½	2 hrs.	+	+	+	+	+
15	J.R.	$^{7}/12$	2 mins.	-	+	+	+	+
16	E.W.	$^{4}/12$	3 mins.	-	+	+	+	-
17	A.B.♀	$1^{5}/12$	5 mins.	-	+	+	+	-
18	C.S.	$1^{3}/12$	10 mins.	-	+	+	L.	-
19	D.McR.	3	4 hrs.	-	+	+	L.	+
20	C.S.	$1^{10}/12$	½ hr.	-	+	+	+	-

Average length of convulsion = 39 minutes.
Average age = $3^{11}/12$ years.

adrenal secretion near the upper or front edge of the floor of the fourth ventricle and one is led to conclude that in some cases the stimulus was insufficient to affect the centre.

(b) The underlying disease.

The question arises as to whether there was any association between the underlying pathological condition and the development of hyperglycaemia following a convulsion. Table VI shows the incidence of hyperglycaemia and hypoglycaemia in the various conditions.

i. Idiopathic convulsions. The most striking feature of this table is the high incidence of blood sugar disturbances in those cases where no obvious cause was found for the convulsion. Though hyperglycaemia was observed in only five of these patients the remaining two had glycosuria (Table VII) and there can be no doubt that the hyperglycaemic phase was missed in the latter. It will be noted that in two of these children, though a high blood-sugar level was registered soon after the convulsion, sugar did not appear in the urine, suggesting that the threshold for glucose for these individuals had not been reached. Gilchrist[68] in an investigation in children found the average renal threshold for glucose to be 238 mgm. per cent. and in one case a blood-sugar of 310 mgm. per cent. was attained without the appearance of sugar in the urine.

TABLE VI.

Disease.	No. of Cases	No. showing Hyper-glycaemia.	No. showing Glycosuria alone.	No. showing Hypo-glycaemia.
Idiopathic convulsions	7	5	2	5
Meningitis	13	7	1	-
Epilepsy	4	-	1	-
Tetany	10	-	-	2
Anaemia	2	2	-	-
Uraemia	3	1	-	-
Tuberculosis	3	1	-	2
Encephalitis	1	-	1	1
Mental deficiency	3	-	-	-
Birth injury	6	-	-	6
ACUTE INFECTIONS:				
(a) Gastro-enteritis	6	-	-	6
(b) Ileo-colitis	3	-	-	2
(c) Tonsillitis	5	-	1	1
(d) Bronchitis	2	-	-	-
(e) Pneumonia	4	-	-	2
(f) Infectious fevers	2	-	-	-
		16	6	
	74	22		27

TABLE VII.

Idiopathic Convulsions.

No.	Name.	Age in years.	Maximum Blood-Sugar. mgm.%	Glycosuria.	Minimum Blood-Sugar mgm.%
1	E.F.	$1^{6}/12$	232.6	-	42.8
2	M.B.	$2^{5}/12$	312.5	++	63.2
3	W.M.	$2^{3}/12$	133.3	+	36.3
4	H.K.	$2^{1}/12$	317.4	+	87.7
5	E.G.	$1^{11}/12$	250.0	-	36.7
6	J.K.	$4^{9}/12$	144.9	+	36.5
7	J.D.	2	232.6	++	39.7
Average age = $2^{5}/12$ years.					

Throughout their subsequent stay in hospital the urine of all these children was repeatedly tested for sugar with negative result, thus strongly suggesting that the disturbance of the blood-sugar was transient and directly due to the convulsion.

Four of this group had no history of a previous convulsion, two had one short fit some months before admission and one had two fits of varying length. There was therefore no justification for placing them in the epilepsy group which comprised children of an average age of nine-and-a-half years and with a long history of well-established epileptic fits behind them.

Four of the seven children had fever on admission,

which subsided within a day or two, and no other clinical evidence of an acute infection than a reddening of the fauces. All had been normal, full-time, healthy babies with no history of trauma at birth. When the initial disturbance due to the convulsion had subsided none of them presented any abnormality of the nervous system. It thus appears that for the present at least these cases must remain unclassified though it is very probable that one or two will eventually become true cases of epilepsy.

ii. Meningitis. It will be seen that in this group also, there is a fairly high percentage of patients showing disturbance of the blood-sugar level. Eleven of the children were suffering from tuberculous meningitis and it is significant to recall the post-mortem findings in this disease. The fact that the purulent exudate is thickest at, and in some cases confined to, the base of the brain, considered in the light of Cannon and Rapport's[67] and Brooks'[69] conviction that there is a centre in the medulla oblongata controlling adrenal secretion and the blood-sugar level, suggests a possible explanation of the relatively high incidence of hyperglycaemia in convulsions due to tuberculous meningitis. It is reasonable to suppose that any sudden increase in intracranial pressure, such as that occasioned by a convulsion, would stimulate the more readily, a nerve centre already irritated by the presence of a constricting inflammatory exudate.

iii. Epilepsy. Regarding the four cases of undoubted epilepsy in the series it will be seen that no hyperglycaemic values were observed but in one patient, (J.W., Case 44), in whom the convulsion was severe and prolonged, sugar was discovered in the urine obtained eight-and-a-half hours after the fit. Thus it would appear that the time factor is of great importance since in the other cases the seizures lasted no more than a few minutes.

iv. Tetany. No case of tetany in the series showed a hyperglycaemic value, a finding which strengthens one in the conviction that the length and severity of the convulsion had a direct bearing on the occurrence of blood sugar disturbance.

TABLE VIII.

Convulsions due to Tetany.

No.	Name	Age in years	First Blood-Sugar Observation mgm.%	No.of hours after first fit.	Serum Calcium mgm.%	Serum Phosphorus mgm.%	Plasma Phospha-tase. units.	Clinical Rickets.
1	E.S.	8/52	63.3	12	7.8	-	17.8	-
2	W.M.	27/52	92.6	6½	6.5	4.8	13.7	Enlarged radial epiphyses.
3	P.C.	7/52	91.3	6	9.6	3.6	13.0	-
4	T.M.	3/12	90.1	8½	8.5	2.9	19.2	-
5	E.D.	6/52	54.3	12	7.2	3.9	18.2	-
6	J.J.	8/52	59.1	18	6.5	4.0	23.6	-
7	G.W.	1 1/12	172.3	8 (a)	10.7	4.5	14.0	Enlarged radial epiphyses.
				(b)	8.9	-	-	Rachitic rosary
8	J.McL.	11/52	126.5	13	8.7	4.0	13.2	Craniotabes. Beading of ribs
9	W.McD.	6/12	75.7	13	6.5	3.6	20.6	Enlarged radial epiphyses.
10	A.McM.	12/52	64.9	5	8.1	5.6	14.4	-

The clinical history of all these patients was exactly the same; each had had a long series of small fits lasting no more than a few minutes, and occurring at intervals of an hour or two. The very nature of the fit suggests a possible explanation as to why no case of tetany was seen till many hours had elapsed from the time of the first fit. The criticism may be levelled that since no case of tetany was seen sooner than five hours after the first fit one is not entitled to assume that there had been no disturbance of the blood-sugar regulating mechanism. While not denying the possibility of such an occurrence, it is pertinent to point out that in no case was glycosuria ever observed though assiduously sought. Furthermore in a few patients the blood-sugar was estimated immediately after several of the short fits and in no case was a significant rise noted. Reference will be made later to the fact that in only two of these patients was a hypoglycaemic level observed and in neither was it of severe degree. Thus it would appear that there is no gross disturbance of carbohydrate metabolism in tetany. Svengaard[70] found low fasting blood-sugar values in infantile tetany but no abnormality of metabolism. It is interesting to note in this connection that Greisheimer[71] in experiments on decerebrate dogs found that when the blood-sugar fell the muscle irritability increased and vice versa.

v. Anaemia. One of these patients (A.M., Case 7), a boy of $5^{8}/12$ years, was a straightforward case of nutritional anaemia under treatment with ferrous sulphate. Suddenly during one night he took a severe left-sided convulsion which lasted about half-an-hour and was followed by a period of deep coma. By the next morning, however, the boy's condition appeared to be normal and he had no further fits during his stay in hospital. The sugar content of the blood fifteen minutes after the onset of the convulsion was 277.7 mgm. per cent. The origin of the convulsion remained entirely unexplained.

In the other patient with anaemia the fit was a terminal feature and at autopsy a slight pachymeningitis interna haemorrhagica was found, along with an early longitudinal sinus thrombosis.

vi. Uraemia. Strictly speaking only two of the patients in this section really had uraemia, since the convulsion in the other child occurred at the onset of an attack of acute nephritis and was thus probably due to cerebral oedema. This latter patient (M.O'H., Case 70) developed a chill and vomited a little four days before admission. Two days later she complained of severe abdominal pain and had a short dry cough. At 6 a.m. on 19/1/36 she had a generalised convulsion lasting fifteen minutes and four more in the next three hours. At 9 p.m. and again at 10.30 p.m. she had further fits. On admission the blood-sugar was 78.7 mgm. per cent. and the urine contained albumin, blood, acetone, red corpuscles and casts, but no sugar. The blood-sugar

readings throughout the day were not significant.

←----------20/1/36----------→

Time.	12.30 a.m.	1 a.m.	1.30 a.m.	2 a.m.	9.30 a.m.	10 a.m.	10.30 a.m.	11 a.m.	12 noon	1 p.m.
Blood-Sugar. mgm.%	78.7	69.4	72.4	71.9	69.4	69.1	69.9	64.1	62.5	58.5
No.of hours after convul-sion.	18½	19	19½	20	27½	28	28½	29	30	31

etc.

It is clear that any change which occurred in the blood-sugar in this case was missed. Metcalf and Moriarty[18] record a very similar case of acute haemorrhagic nephritis where after a convulsion the blood sugar rose to 312 mgm. per cent. and fell rapidly to normal levels.

The first case of true uraemia was in M.M. (Case 56), a girl of eight years, who had been suffering from a nephrotic type of nephritis for some months and was steadily going down-hill. The urine was loaded with albumin and casts but contained no blood. At 3.45 p.m. on 1/6/36 she took a generalised con-vulsion lasting five minutes. As will be seen the blood-sugar suffered no significant change.

Time	4 p.m.	4.15 p.m.	4.30 p.m.	5 p.m.
Blood-Sugar. mgm.%	76.2	78.1	88.5	94.3
No.of hours after con-vulsion.	15 mins	30 mins.	45 mins.	1¼ hrs.

The second case was interesting in that the convulsion was a prolonged and severe one and the blood-sugar level three hours after the onset was distinctly high. The girl (M.B., Case 71) had an attack of tonsillitis five weeks before admission. Two weeks later she developed oedema of the feet and albuminuria. The oedema quickly became generalised and the child became very ill. About 7.30 a.m. on the day of admission she took a convulsion and this continued till admission when she was found to be moribund. The blood-sugar at 10.30 a.m. was 256.4 mgm. per cent. The urine obtained by catheterisation was loaded with albumin and contained also sugar and acetone. The child died at 11.30 a.m.

vii. Tuberculosis. The three patients suffering from tuberculosis, other than meningeal, are interesting in that the presence of a cerebral tuberculoma was postulated to explain the fits.

Case 16 (D.McL.) has been described at some length earlier in this study. It will be remembered that he was observed on two separate admissions to hospital, on the first occasion three hours after a convulsion, and on the second eighteen hours after a similar convulsion. This boy had a tuberculous elbow joint at the age of one-and-a-half years. The Mantoux test was strongly positive and he was found to have a heavy pyuria which was later proved to be tuberculous. There were, however, no localising signs in the examination of the nervous system. A skiagram of the skull showed no evidence of calcification.

Case 53 (M.H.), a girl of eleven years, was suffering from hilum tuberculosis. At 5.15 p.m. on 6/11/35 she screamed and became rigid. The pupils were dilated and fixed and the child quite unconscious. There was no clonic stage and she had quite recovered in fifteen minutes.

Time.	5.30 p.m.	6 p.m.	6.30 p.m.	7 p.m.	9 p.m.	12 midnight
Blood-sugar mgm.%	86.9	87.9	71.9	102.0	78.1	94.7
No.of hours after convulsion.	15'	45'	1 hr.15'	1 hr.45'	3 hrs.45'	6 hrs.45'

The urine contained neither sugar nor acetone. The Mantoux skin test was strongly positive and a skiagram of the chest revealed enlarged hilum and mediastinal glands and a probable Ghon's focus. Here again, when the child had recovered from the effects of the convulsion, no abnormality could be detected in the nervous system.

Case 51 (D.W.), a girl of $2^{3}/12$ years, was healthy till two months before admission when she became cross and irritable and did not sleep well. She developed a right-sided cervical adenitis which was diagnosed as tuberculous and treated by evacuation and scraping. She seemed to be doing fairly well after this but on 23/9/36 she was out of sorts and put to bed early. After vomiting several times early in the evening, she took a generalised convulsion at 8.30 p.m. The head and eyes were turned to the right and the convulsive movements persisted for about five hours. The urine contained nothing abnormal.

←----------24/9/36----------→ (25/9/36)

Time	1.30 a.m.	3 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	3.30 p.m.	5 p.m.	6 p.m.	8 p.m.	12 mid-night	9.30 a.m.
Blood-Sugar. mgm.%	88.5	75.7	58.1	55.8	56.8	63.6	60.6	46.5	45.0	52.9	54.1	55.2	61.7
No.of hours after convulsion.	5	$6\frac{1}{2}$	13	14	15	16	18	19	$20\frac{1}{2}$	$21\frac{1}{2}$	$23\frac{1}{2}$	$27\frac{1}{2}$	37

On examination next morning she was found to be a well-nourished girl, conscious but drowsy, with a right-sided malar flush. There was marked spasticity of the right arm and leg and the right hand was held in the claw position. The knee-jerks and arm-jerks were equal and active and the plantar responses flexor. The abdominal reflexes were active on the left side and

sluggish on the right, the lower one being absent. Lumbar puncture revealed a normal cerebro-spinal fluid and a skiagram of the skull was negative. The Mantoux skin test was markedly positive, however, and on ophthalmoscopic examination a tubercle was observed on the right choroid plexus.

In all three cases the possibility of cerebral tuberculoma must be raised though the evidence is too slender to be definite. None of the children had a history of previous convulsions and none of them exhibited a fresh acute infection which might have accounted for the fit. It is interesting to note that only in one case (Case 16) was a marked fluctuation of the blood-sugar level observed. While it was not surprising in view of the shortness of the convulsion to find no significant change in the second case, one rather expected to find higher values in the third case. Considering, however, that the child was not seen till five hours after the onset it seems not unreasonable to assume that the higher readings were missed. That a disturbance of the blood-sugar did occur is evidenced by the low level recorded nineteen hours after the convulsion.

viii. Encephalitis. This case is interesting in that on repeated occasions glycosuria was observed following a fit before ever this investigation into the blood-sugar in convulsions was begun.

Case 25 (E.O'N.) was a healthy baby and was first admitted at $1^{4}/12$ years with a history of having fallen on the back of her head and four or five hours later gone into a severe generalised convulsion which lasted about ten hours. The condition was diagnosed as encephalitis and the left face, arm and leg were left in a state of spastic paralysis. This gradually improved with massage but the child continued to take bouts of

tonic fits every few months. These came on every ten minutes and she often had as many as twenty in one day. At 2 a.m. on 13/7/35 she had a minor fit and at 2.30 p.m. she took a severe clonic spasm which lasted about three hours. After the fit the right arm and leg were very spastic and the left arm and leg flaccid. The knee-jerks were not elicited and the plantar responses equivocal. The child had bronchitis and the throat was red. Lumbar puncture revealed no abnormality but the urine obtained some hours after the fit contained abundant sugar but no acetone. The phenyl-hydrazine test showed definite glucosazone crystals.

Two months later the child was readmitted after a severe convulsion affecting chiefly the right arm and leg. The urine again contained sugar and on this occasion acetone also.

At 11.45 p.m. on 26/3/37 the patient (now aged $5\frac{9}{12}$ years) took a severe fit which lasted about one hour. When seen at 9.30 a.m. the next morning she was very dull, but not acutely ill. There was nothing abnormal in the heart, lungs or abdomen but the tonsils were enlarged and red. The left knee-jerk was very active and the right sluggish, while the left plantar response was extensor. The urine obtained at 9 a.m. contained sugar and acetone.

←----------27/3/37----------→ ←-28/3/37---→

Time	9.45 a.m.	10.45 a.m.	11.45 a.m.	12.45 p.m.	1.45 p.m.	2.45 p.m.	3.45 p.m.	4.15 p.m.	4.45 p.m.	12 mid-night	4 a.m.	8 a.m.
Blood-Sugar. mgm.%	113.6	117.6	80.6	55.2	59.1	52.9	43.5	52.1	50.2	66.6	42.2	45.2
No.of hours after convul-sion.	10	11	12	13	14	15	16	$16\frac{1}{2}$	17	$24\frac{1}{4}$	$28\frac{1}{4}$	$32\frac{1}{4}$

It will be seen therefore that following every major fit, this child had a disturbance of her blood-sugar regulating mechanism, evidenced in all cases by glycosuria. There can be little doubt that had a specimen of blood been obtained from this patient soon after her convulsion it would have shown a high

percentage of sugar. Once more the progressive fall in blood-sugar during the day following the convulsion is well illustrated. That this fall is little influenced by the ingestion of carbohydrate is shown by the fact that this child was receiving seven ounces of whole milk and one teaspoonful of sugar every four hours. The examination of the urine on repeated occasions revealed no sugar, and a sugar tolerance curve done a few days later was normal except for rather a high rise.

The child was given 14 gm. glucose in 140 c.c. water.

Fasting.	½ hour.	1 hour.	1½ hours.	2 hours.
71.9	169.5	120.5	86.2	61.7 mgm. per 100 c.c.

ix. Mental deficiency. None of the three cases in this section showed any marked change in the blood-sugar level as the result of the convulsion.

Case 35 (H.McV.), a girl of 1 6/12 years, had been a normal full-time baby but at the age of ten months began to take fits almost daily. This continued for two months and she was admitted to hospital, but the blood-sugar was not examined on this occasion. The fits stopped and for a few months she was very well. On 14/9/36, however, she was readmitted with a history of having been well till twelve hours before, when she began to have generalised convulsions, each lasting about three minutes and recurring at hourly intervals. On admission these ceased. The child was healthy-looking and well-nourished but had the appearance of being mentally defective. The serum calcium was normal. On 17/9/36 she had a convulsion which lasted less than one minute. The urine was in no way abnormal.

Time	10.50 a.m.	11.20 a.m.	11.50 a.m.	12.50 p.m.	2.50 p.m.	3.50 p.m.	4.50 p.m.
Blood-Sugar mgm.%	72.5	69.4	78.1	59.5	86.9	78.1	69.9
No.of hours after	5'	35'	1 hr.5'	2 hrs.5'	4hrs.5'	5 hrs.5'	6 hrs. 5'

Case 24 (C.T.), a girl of four years, had a long history of recurring convulsions from infancy. She was admitted to hospital on two occasions and it became obvious that she was mentally defective. For two months before her last admission the convulsions had become more frequent and severe. At 9.25 a.m. on 21/7/36 she had a generalised convulsion which lasted about one minute. The urine contained no abnormal constituent.

Time	9.30 a.m.	10 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	3.30 p.m.	4.30 p.m.
Blood-Sugar. mgm.%	65.3	68.5	65.3	54.3	65.3	66.6	73.0	65.3
No.of hours after convul-sion.	5'	35'	1 hr.5'	2 hrs.5'	3 hrs.5'	5 hrs.5'	6hrs.5'	7 hrs.5'

Case 69 (D.McR.) was a boy of three years who had failed to develop normally. He was unable to feed himself or walk and had incontinence of urine and faeces. Three days before admission he began to take convulsions. He was a pale child obviously mentally defective, with a left-sided hemiplegia. A few days after admission the boy took a generalised convulsion which lasted about four hours.

Time	9.30 a.m.	10 a.m.	10.50 a.m.	10.55 a.m.
Blood-Sugar. mgm.%	90.9	90.9	78.1	73.0
No.of hours after convulsion.	40'	1 hr.10'	2 hrs.	2 hrs.5'

At 11 a.m. the child was given glucose intravenously and this will be discussed later.

It is impossible to draw any conclusion from three cases, but it would appear that the frequent short convulsions so common in conditions of mental defect are unaccompanied by any disturbance

of the blood-sugar level. This is not in accordance with the findings of Darrow[30] who noted both high and low blood-sugar values in two mentally defective children after convulsions.

x. Birth injury. The striking feature of this group is that while hyperglycaemia was never observed, hypoglycaemia was common to all.

TABLE IX.

No.	Name.	Age in years.	First Blood-Sugar Reading. mgm.%	Length of Time from Onset. hours.
1	B.T.	2/365	39.8	12
2	A.G.	3/365	42.0	48
3	J.C.	2/365	46.5	18
4	D.N.	3/365	25.2	27
5	M.C.	12/365	88.5	5
6	O.M.	1/12	24.7	5

Though the patient M.C. (Case 12) had a blood-sugar content of 88.5 mgm. per cent. when first seen, six hours later it had fallen to 49.2 mgm. per cent. and in another six hours had reached the very low level of 24.8 mgm. per cent.

The normal blood-sugar content of the healthy infant has been variously placed by different workers. Sedgwick and Ziegler[72] found that it was essentially the same as in a normal adult and gave 70 mgm. per cent. as an average figure, while

Lucas et alia[73] thought it was somewhat lower than this. According to Hellmuth[74] it is always lower in the infant than in the mother but changes parallel with that of the mother. In a series of ninety-four infants Greenwald and Pennell[75] found that during the first two weeks of life the post-absorptive blood-sugar is at a distinctly lower level than at any later period and came to the conclusion that the average range of values was between 71 and 80 mgm. per cent. It will thus be seen that the values recorded for these children are distinctly abnormal.

It is impossible to be sure that hyperglycaemia ever occurred in these children since in every case the convulsion had been going on for a considerable time before the patients were seen. That it was possible in one child to follow the blood-sugar from a moderate level down to a very low level rather favours the theory. Further reference will be made to these cases in a later section.

xi. ACUTE INFECTIONS. Lastly comes the group of frank acute infections where it will be seen not a single hyperglycaemic value was observed.

(a) Gastro-enteritis.

TABLE X.

No.	Name	Age in years	First Blood-Sugar Reading mgm.%	Time after Onset of Fit.	Urine.	Result.
1	M.McA.	9/52	40.1	4½ hrs.	Not obtained	Died
2	A.P.	18/52	21.0	9 "	Not obtained	Died
3	M.McL.	12/52	19.7	3½ "	No sugar. Acetone +	Died.
4	V.C.	6/52	18.8	9½ "	Not obtained.	Died.
5	J.H.	5/52	81.3	21 "	Tr.albumin. Nil else.	Died.
6	C.N.	1 10/12	109.9	½ hr.	No sugar. Acetone +	Irregularly dismissed.

The only child who was seen within a short time of the convulsion was the last and it will be noted that he has the highest blood-sugar value of the series.

C.N. (Case 20), a boy of 1 10/12 years, was a healthy child till about seven hours before admission when he became drowsy, disinclined to play and developed diarrhoea. Fifteen minutes before admission he took a generalised convulsion which lasted about ten minutes.

	←---------26/8/36--------→				←--------27/8/36----------→			
Time	3.50 p.m.	4.50 p.m.	7 p.m.	10 p.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.
Blood-Sugar. mgm.%	109.9	85.4	54.6	44.4	27.0	23.3	22.3	24.6
No.of hours after convul-sion.	½	1½	3 hrs. 40'	6 hrs. 40'	18 hrs. 10'	19 hrs. 10'	20 hrs. 10'	21 hrs. 10'

From the subsequent behaviour of the blood-sugar it seems reasonable to postulate a sudden, sharp but transient rise which was observed on the downgrade. The fact that no sugar appeared in the urine does not entirely negative this suggestion since, as we have seen from cases in this investigation and from the quoted works of others, glycosuria may not occur with quite high blood-sugar levels.

The first four children in this section had fulminating infections and died soon after admission.

M.McA. (Case 55), a baby of $^{9}/_{52}$ year, was artificially fed from birth and throve well till 8 a.m. on the day of admission when he began to have generalised twitchings affecting especially the legs. These continued till admission three-and-a-half hours later.

On examination the child was convulsing and unconscious and the pupils were dilated and fixed. He had two very loose bright yellow stools during examination.

Time	12.30 p.m.	2.45 p.m.
Blood-Sugar. mgm.%	40.1	23.9
No. of hours after convulsion.	$4\frac{1}{2}$	$6\frac{3}{4}$

The child died just after the second specimen of blood had been taken.

At post-mortem there were marked inflammatory changes in the small bowel and the liver and kidneys showed cloudy swelling.

A.P. (Case 10), a boy of 18/52 year, was healthy till two days before admission when he seemed cross and out of sorts. At 6.30 a.m. on 9/3/36 he had a convulsion during which he became rigid and the eyes rolled. The respirations were gasping and rapid. He recovered from this but remained very ill throughout the day and had three stools, two of which were pale and slimy. He had another convulsion at 3.15 p.m.

Time	3.25 p.m.	4.25 p.m.
Blood-Sugar. mgm.%	21.0	3.7
No. of hours after convulsion.	9	10

On admission the child was moribund and during examination had two loose, green, slimy stools.

An intravenous infusion of 75 c.c. of 10 per cent. glucose in normal saline was given into the superior longitudinal sinus at 5.45 p.m., but the child died at 7.30 p.m.

Time	6 p.m.	6.30 p.m.	7 p.m.	7.30 p.m.
Blood-Sugar. mgm.%	256.4	208.4	163.9	84.0

At post-mortem there was found congestion and lymphoid hyperplasia of the whole bowel and cloudy swelling of the liver.

M.McL. (Case 65), a girl of 3/12 year, was a twin and had never thriven well. Three weeks before admission she had an attack of gastro-enteritis from which she appeared to have recovered. During the night before admission she became ill with rapid respirations, vomiting and diarrhoea and by 3 a.m. she was very ill. At 7 a.m. she had a generalised convulsion which lasted about one hour.

On admission she was found to be a marasmic child, cyanosed and collapsed. The liver was enlarged and both

kidneys palpable. The blood-sugar at 10.30 a.m. was 19.7 mgm. per cent. The urine contained acetone and a trace of albumin but no other abnormal constituent.

The child died at 7.35 p.m. and at autopsy there was found irregular, patchy congestion of the small intestine and marked, fatty degeneration of both kidneys and liver. The suprarenals appeared to be normal.

V.C. (Case 68), a boy of 6/52 year, was breast fed and throve well till two weeks before admission shen he developed a cough and became constipated. Two days before admission he was given one teaspoonful of castor oil and next day developed loose, green, slimy stools which persisted. On examination he was a rather under-nourished baby, acutely ill. He had two convulsions between midnight and 1 a.m. on 31/1/36 and rapidly became more ill. The liver was much enlarged and the spleen was just palpable.

Time	9.30 a.m.	11.30 a.m.
Blood-Sugar. mgm.%	18.8	21.0
No.of hours after convulsion.	9½	11½

The child died at 4.30 p.m. and at autopsy the liver was found to be much enlarged and giving almost the appearance of acute yellow atrophy. There was marked congestion of both small and large bowel.

These cases show such a striking and constant diminution in the blood-sugar level that one hesitates to attribute it solely to the effects of the convulsion. It is impossible to prove that there had been a sharp rise soon after the fits though the child C.N. who showed a rapid fall in the blood-sugar seemed to suggest it. Let us recall the sequence of

events which has been postulated as consequent on a convulsion of any magnitude; the adrenals are stimulated through the splanchnic nerves by the disturbance of the central nervous system and there is a consequent outpouring of adrenaline which mobilises glucose from the glycogen of the liver, causing a sharp rise in the blood-sugar. This is often followed by a fairly rapid fall, sometimes to very low levels, which tend to persist in spite of the ingestion of food; this second phase is probably due to the adrenal exhaustion. In view of the fact that the illness in each of these four children was fulminating and that there was thus severe toxaemia, it seems reasonable to suppose that the point of exhaustion of the adrenals would be reached much earlier and after less effort than in a comparatively healthy child. It may well be that even the maximum effort of their adrenal glands acting as it did on a liver also damaged by circulating toxins, failed to produce a striking change in the blood-sugar but nevertheless used up the store of adrenaline and permitted the over-action of insulin. It is generally accepted[76] that the actual level of sugar in the blood is the result of the balance between the antagonistic actions of endogenous adrenaline and endogenous insulin, certain other factors participating on either side.

The factor of hepatic insufficiency apart altogether from hypo-adrenalinism is probably of great importance in these cases. It has frequently been demonstrated that toxic and poisonous agents such as phosphorus may produce a marked fall

in the blood-sugar and it is certain that the severe toxaemia incident upon an illness which caused death in each case within twenty-four or forty-eight hours would produce marked changes in the liver. In point of fact one of the patients (Case 68) showed evidence of acute yellow atrophy of the liver and in all the others there was cloudy swelling and fatty degeneration.

The remaining patient, J.H. (Case 18), recovered from the effects of the convulsion but died later from a protracted gastro-enteritis. Very low blood-sugar readings were recorded between twenty-six and forty-three hours after the series of fits and these would appear to be the expression of an exhausted sympathico-adrenal system and a damaged liver.

(b) Ileo-colitis.

TABLE XI.

No.	Name	Age in years	First Blood-Sugar Reading. mgm.%	Time after Onset.	Urine.	Result.
1	J.R.	7/12	103.1	2 mins.	Not obtained	Died.
2	M.G.	4	8.5	4 hrs.	Not obtained	Died.
3	C.S.	1 10/12	86.2	½ hr.	nil.	Well.

The first two of these cases were acute dysenteric infections, the first child dying within twelve hours of the onset of any symptoms and the second within twenty-four hours.

J.R. (Case 39), a girl of 9/12 year, had been a normal full-time baby. She was well until the morning of 26/4/36 when she was noticed to be breathing heavily and had rhinitis and some cough. She vomited once and soon became extremely ill. She was brought to hospital about noon profoundly ill and on admission had a convulsion. The blood-sugar at this time was 103.1 mgm. per cent. She rapidly became moribund and the blood-sugar three hours later had fallen to 33 mgm. per cent. This was the lowest level obtained among the patients examined within three hours of the primary convulsion. The child died four hours after admission to hospital and the blood-sugar at death was found to be 12 mgm. per cent. At autopsy fairly marked congestion of the terminal portion of the ileum and the whole of the large intestine, was noted. The liver was the seat of cloudy swelling.

M.G. (Case 50), a boy of 4 years, had been well till 11 a.m. on 22/7/36 when he vomited and began to have diarrhoea with slimy motions. The vomiting persisted and he became dull and listless. While being brought to hospital he began to have twitchings of the face and limbs which continued at irregular intervals. The blood-sugar at 6.30 a.m. on 23/7/36, four hours after the onset of the fits, was 8.5 mgm. per cent. The child died at 7.55 a.m. and the sugar content of blood taken from the heart immediately after death was 11.7 mgm. per cent. At post-mortem a Flexner bacillus was isolated from the wall of the colon and this was found to be identical with the organism isolated from the faeces of a sister of the patient, who was later admitted with a similar history. The condition was, however, much less acute and she made a good recovery.

It is conceivable that the convulsions in this case were truly hypoglycaemic in nature though, as we have seen from the previous child, there may be an exceedingly rapid fall in the blood-sugar, and since a specimen of blood was not taken till some hours after the onset of the fits, one is not justified in assuming that there had not been such a fall. It seems not unlikely that in fulminating enteral infections of this type a vicious circle may be established; with the onset

of the illness there may be a convulsion which stimulates the adrenals to promote glycogenolysis. There is a sharp rise in the blood-sugar and a rapid fall further intensified by the severe toxaemia acting alike on adrenals and liver. The blood-sugar falls lower and lower, and finally the severe hypo-glycaemia begins to manifest itself by twitching, rigidity, etc. There is a direct therapeutic indication here, though, as we have seen in Case 10, the actual raising of the blood-sugar from almost zero to over 200 mgm. per cent. did not prevent the death of the child, since it was undertaken too late. The question of whether glucose is better administered by the oral or intravenous route will be considered later.

It is interesting to note while considering these low blood-sugar values observed in convulsions accompanying diarrhoeal diseases, that Joslin[77] stressed the fall in blood-sugar which occurred in diabetes during an attack of diarrhoea. He pointed out that the tolerance of a diabetic for carbo-hydrate might increase during such an attack. Takeda[78] thought that the ability of the liver to control the amount of sugar in the blood was destroyed by the toxin of dysentery, while Brown,[79] on the other hand, found that diarrhoea per se did not make much difference to the blood-sugar level.

The last case of ileo-colitis, C.S. (Case 30), was not acutely ill. On the day before admission she was noticed to be listless and next day vomited everything. At 8 p.m. on the day of admission she took a generalised convulsion which lasted half-an-hour. Nothing abnormal was observed in the urine but the stools contained blood and mucus.

←--20/11/35-→ ←----------21/11/35----------→

Time	10.30 p.m.	11.30 p.m.	12.30 a.m.	1.30 a.m.	2.30 a.m.	3.30 a.m.	9.30 a.m.	12 noon.
Blood-Sugar. mgm.%	86.2	77.5	76.3	81.3	86.9	84.2	82.6	66.6
No.of hours after convulsion.	$2\frac{1}{2}$	$3\frac{1}{2}$	$4\frac{1}{2}$	$5\frac{1}{2}$	$6\frac{1}{2}$	$7\frac{1}{2}$	$13\frac{1}{2}$	16

Thus no significant change in the blood-sugar occurred as the result of this convulsion at the onset of an acute but not severe infection.

(c) Tonsillitis.

TABLE XII.

No.	Name	Age in years	First Blood-Sugar Reading. mgm.%	Time after Onset. hours.	Urine.	Result.
1	R.D.	$6\frac{1}{2}$	96.1	2	Acetone +	Well.
2	I.G.	$5\frac{3}{12}$	47.1	18	Acetone +	Well.
3	C.McG.	$1\frac{5}{12}$	67.1	11	Acetone +	Well.
4	D.M.	$1\frac{11}{12}$	93.4	$4\frac{1}{2}$	Trace of sugar.	Well.
			(Cerebro-spinal fluid sugar 142.8	$2\frac{1}{2}$	No acetone.	
5	J.A.	1	101.0	?	Tr. acetone	Well.

It will be seen that no case in this group showed a hyperglycaemic value; but on the other hand slight glycosuria was observed in one patient. In the child D.M. (Case 31) it

is significant that the sugar content of the cerebro-spinal fluid which was obtained earlier than the first blood specimen, was considerably higher than that of the blood. As it is usually accepted that the sugar content of the blood and cerebro-spinal fluid run on closely parallel lines, with that of the latter on a lower plane, it seems probable that the blood-sugar was considerably raised soon after the fit.

Four of these children had no history of previous convulsions but the first seems to have had a tendency to take a fit at the onset of any infection.

(d) Bronchitis. Neither of the two cases in this series showed any disturbance of the blood-sugar. Both children were large, fat babies of just over one year, with upper respiratory infections. It was thought at first in each case that the convulsion was hypocalcaemic, but the serum calcium was normal in both. Both were seen early enough for any blood-sugar upset to have been noted.

(e) Pneumonia.

TABLE XIII.

No.	Name	Age in years.	First Blood-Sugar. mgm.%	Time after Onset. hours.	Urine.	Result.
1	J.G.	2	25.0	8	Albumin +	Died.
2	J.S.	6	106.4	5	Acetone +	Died.
3	A.B.	1 5/12	70.9	2	Not obtained.	Died.
4	T.B.	10/12	149.7	1	Not obtained.	Died.

J.G. (Case 36), a boy of two years, had been a healthy baby and throve well and developed normally. He had two convulsions at the age of one year, but made a good recovery. Both ears discharged at intervals. On 10/1/36 he suddenly vomited and became fevered. After lying still all night he took a convulsion at 7.15 a.m. and this continued till admission at 3 p.m. On examination he was comatose, twitching and cyanosed.

Time.	3.15 p.m.	4.15 p.m.	5.15 p.m.	6.15 p.m.	7.30 p.m.
Blood-Sugar. mgm.%	25.0	31.2	28.8	14.5	11.2*
No.of hours after convulsion.	8	9	10	11	12¼

* Specimen of blood taken from the heart immediately post-mortem.

The child did not come out of the convulsion and died at 7.30 p.m. At post-mortem he was found to have had an acute broncho-pneumonia, a left acute otitis media and a longitudinal sinus thrombosis.

In the second patient of this group the convulsion was of short duration and the blood-sugar showed no significant alteration.

A.B. (Case 61), a girl of 1 5/12 years, had been a healthy infant and throve well till three weeks before admission when she took whooping cough. One week later she became listless and developed stomatitis. On the night of 18/6/36 she had four generalised convulsions and was admitted on 19/6/36. On examination she was found to be a fair-sized thin child, acutely ill, cyanosed and comatose. There was a yellowish nasal discharge and the tongue was covered with thick white sordes. There was dulness at the left base and abundant râles all over the left lung. The heart was enlarged to the left, the liver was greatly enlarged and the spleen was just palpable. She remained acutely ill and between 1 a.m. and 3 a.m. on 20/6/36 had two convulsions. The urine was not obtained.

Time.	3 a.m.	9.30 a.m.	11.15 a.m.
Blood-Sugar. mgm.%	70.9	30.8	15.1
No.of hours after convulsion.	2	$8\frac{1}{2}$	$10\frac{1}{4}$

The child died at 11.15 a.m. and at post-mortem there was found a diffuse broncho-pneumonia with foci of suppurative softening. The liver, spleen, kidneys, suprarenals and pancreas were free from any gross pathological change. Both middle ears contained pus.

T.B. (Case 6), a boy of $^{10}/_{12}$ year, was a healthy baby till three weeks before admission when he developed a swelling of the left side of the neck. Three days before admission he began to vomit his feeds, developed a slight cough and became drowsy and constipated. He was admitted on 24/4/36 and was found to be a well-nourished child acutely ill and breathing heavily. There was a swelling in the left sub-maxillary region and the tonsils were enlarged and red. Apart from a few scattered rhonchi nothing could be detected in the lungs. The liver was enlarged and the left kidney palpable. At midnight on 26/4/36 he had a generalised convulsion and the temperature rose to 105°F.

Time.	1 a.m.	10 a.m.	12 noon.	3 p.m.
Blood-Sugar. mgm.%	149.7	88.5	81.3	136.9
No.of hours after convulsion.	1	10	12	15

Two days later, signs of consolidation appeared in the left lung, the child's condition rapidly became worse and he died at 6.20 a.m. on 29/4/36. At post-mortem there was found an extensive area of broncho-pneumonia showing early suppuration, in the left lower lobe. The liver and kidneys showed some fatty change.

It seems probable that the first reading obtained signifies a sharp but transient alteration in the blood-sugar level, though this was not apparently of such a degree as to entail a subsequent stage of hypoglycaemia.

It is interesting to observe the same phenomenon taking place in fulminating cases of broncho-pneumonia as was previously noted in gastro-enteritis and ileo-colitis.

(f) Infectious diseases.

W.Mc.K. (Case 4), a boy of 19/52 year, was well till 8 p.m. on 3/11/36 when he had a convulsion which lasted five minutes and two similar convulsions before admission at 11.30 p.m. On examination he was found to be a small, poorly-nourished child, acutely ill. The fontanelle was bulging and there was some nuchal rigidity, but nothing abnormal was detected in examination of the various systems. Cerebro-spinal fluid obtained by lumbar puncture was quite normal. The urine contained a trace of albumin.

Time.	12.30 a.m.	1.30 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	3 p.m.
Blood-Sugar. mgm.%	158.7	105.2	105.2	80.6	96.1	114.9	121.9
No.of hours after convulsion.	4½	5½	13½	14½	15½	16½	19

The child developed a haemorrhagic nasal discharge from which B. diphtheriae was cultured. Two days later he died suddenly and at autopsy only a slight broncho-pneumonia was found.

It will be seen that following the fit the blood-sugar was moderately elevated but no subsequent hypoglycaemic phase was recorded.

O.McC. (Case 67), a boy of 3 11/12 years, was a healthy child till three weeks before admission when he went off his food and had occasional vomiting. At 1 p.m. on 17/1/36 he had a convulsion lasting five minutes and seven similar convulsions between then and twelve midnight. He was a well-nourished boy, not acutely ill. His throat was rather red but the heart, lungs, abdomen and central nervous system showed no abnormality. There were small red macular spots on the skin.

Time.	11 a.m.	1 p.m.	1.30 p.m.	2 p.m.	2.30 p.m.	3 p.m.	4 p.m.	5 p.m.	
Blood-Sugar, mgm.%	81.9	87.7	92.6	90.9	89.2	142.8	70.4	75.2	etc.
No.of hours after convulsion.	22	24	24½	25	25½	26	27	28	

The urine contained abundant ketones but nothing else of note. Lumbar puncture was done and 20 c.c. clear fluid withdrawn. The Pandy test was faintly positive and there was a moderate pleiocytosis, mainly lymphocytic in character. The serum calcium was 11 mgm. per cent. and the blood Wassermann reaction was negative.

On 25/1/36 he developed a bright red scarlatiniform rash on the thighs, buttocks, lower back and elbows. This was diagnosed as German measles and the child transferred to an infectious diseases' hospital where he had no further convulsions and lumbar puncture was not repeated. Thus the abnormal cerebro-spinal fluid remained unexplained.

The absence of any significant change in the blood-sugar is probably explained by the length of time elapsing between the onset of the convulsions and the first blood-sugar estimation.

From the consideration, therefore, of this series of convulsions in relation to the underlying disease the following facts emerge:-

(1) Disturbance of the blood-sugar is specially liable to occur in so-called 'idiopathic' convulsions in children between the ages of two and three years.

(2) It is also common in convulsions due to meningitis and probably also in prolonged epileptic and uraemic seizures.

(3) In tetany and in the minor fits associated with mental deficiency it does not generally occur.

(4) There is a special liability to the development of low blood-sugar values following the convulsions due to intracranial birth injury.

(5) In convulsions due to fulminating infections it is doubtful whether the blood-sugar is markedly raised but there is a distinct tendency to a progressive and severe fall.

(6) Where the convulsions are associated with less severe infections there may occur a sharp rise in the blood-sugar but there is little tendency for low levels to supervene.

(c) The age of the patient.

TABLE XIV.

Age Incidence.

Age in years.	No. of cases	No. showing hyperglycaemia or glycosuria.	%age showing hyperglycaemia or glycosuria	No. showing hypo-glycaemia.	%age showing hypo-glycaemia
0-6/12	22	1	4.5	12	54.5
6/12-1	6	1	16.7	1	16.7
1-2	14	4	29	5	35.7
2-3	10	7	70	4	40
3-4	4	1	25	1	25
4-5	2	1	50	1	50
5-7	8	5	62.5	3	37.5
7-9	3	1	33.3	-	-
9-12	5	1	20	-	-
Totals:	74	22	30	27	36

The age of the patient appeared to have some bearing on the development of hyperglycaemia following convulsions. In Table XIV it will be noted that 7 of the total twenty-two cases in whom either hyperglycaemia or glycosuria were observed were between two and three years old and that this group shows the highest percentage of positively reacting cases. The explanation of this would appear to lie in the fact that of the ten cases in this age group, four were suffering from tuberculous meningitis

and four belonged to the section of idiopathic convulsions and as has been previously demonstrated, hyperglycaemia was most common in these disease groups (Table XV).

TABLE XV.

Diseases in Age Group 2-3 years.

No.	Name	Age in years	Disease.	Maximum Blood-Sugar Value. mgm.%
1	D.F.	$2^{8}/12$	Tuberculous meningitis	125.0
2	E.M.	$2^{5}/12$	Tuberculous meningitis	232.6
3	H.K.	$2^{1}/12$	?	322.6
4	J.D.	2	?	232.6
5	A.B.	2	Tuberculous meningitis	434.8
6	J.W.	2	Tuberculous meningitis	250.0
7	M.B.	$2^{5}/12$	?	312.5
8	J.G.	2	Broncho-pneumonia	31.2
9	D.W.	$2^{3}/12$	? Cerebral tuberculoma	88.5
10	W.M.	$2^{3}/12$	?	133.3

The peak of the incidence of tuberculous meningitis is in the third year of life. There is thus no reason to postulate an increased lability of the blood-sugar or hypersensitivity of the regulating mechanism in the third year of life, though it is interesting to note in this connection that Rector and Jennings[65] found the highest incidence of hypoglycaemia in association with convulsions between two and three years. The

earliest age at which a high blood-sugar value was observed following a convulsion was at fifteen weeks in a child with a gross anaemia. That it was not seen in younger infants is probably due to the time factor. Five of the six cases of intracranial birth trauma had low blood-sugar values when first seen and had been convulsing for many hours but one child M.C. (Case 12) was seen five hours after the onset when the blood-sugar was at a normal level. It was then possible to trace the blood-sugar throughout the day and to observe that it fell steadily to just over 20 mgm. per cent. Thus it would seem that the biphasic nature of the blood-sugar disturbance is independent of the age of the patient.

It will be noted that the incidence of hypoglycaemia is relatively high in the third year of life though not so high as in the youngest group which includes the cases of convulsion due to intracranial birth injury. It is probable that the factor of adrenal exhaustion which we are about to postulate as the cause of the hypoglycaemic phase is, in this latter group, abetted by the natural tendency of the blood-sugar of infants to fall rapidly when fasting. These babies were naturally unable to suck well and though they were fed by pipette or by gavage on admission to hospital they had been virtually fasting for many hours at home. Schonfield[80] stressed the hunger hypoglycaemic of infants, and Winter[81] and Bott[82] were of the opinion that the carbohydrate-regulating mechanism was unstable in young and premature infants.

Conclusions.

1. Blood-sugar disturbance following the convulsions of infancy and childhood is roughly proportional to the length and severity of the convulsion.

2. It is not related to any one pathological condition but is specially common in tuberculous meningitis, 'idiopathic' convulsions, birth injuries and enteral infections.

3. The age of the patient has only an indirect bearing on the possibility of blood-sugar upset, except in the case of very young babies suffering from intracranial birth trauma.

5. The Hypoglycaemic Phase.

No more than passing reference has yet been made to the behaviour of the blood-sugar subsequent to the period of marked elevation immediately following the convulsion. As has been already observed, in a large percentage of cases no significant alteration in the blood-sugar occurred as a result of the convulsion, but early in the investigation it became evident that there was a distinct tendency in some cases to a progressive fall to hypoglycaemic levels. Of the seventy-four patients in the series hypoglycaemic readings were observed in twenty-seven, i.e. 36 per cent. It is significant to recall that, of the thirty-nine children seen within three hours of the onset of the convulsion, the period during which a high blood-sugar value might be expected, it was possible to demonstrate either hyperglycaemic or glycosuria in nineteen, i.e. 49 per cent. Thus there would appear to be a close association between the occurrence of hyperglycaemia following a convulsion and the development of a subsequent hypoglycaemia although it was only possible in a few cases to demonstrate the complete picture.

TABLE XVI.

Total No. of cases.	No. showing Hyper-glycaemia.*	No. showing Hypo-glycaemia.	No. showing Hyperglycaemia & Hypoglycaemia	%age showing Hyperglycaemia & Hypoglycaemia
74	22	27	7	32

* This group includes those in whom the actual hyperglycaemic stage was missed but who had glycosuria.

Figure I shows a typical case.

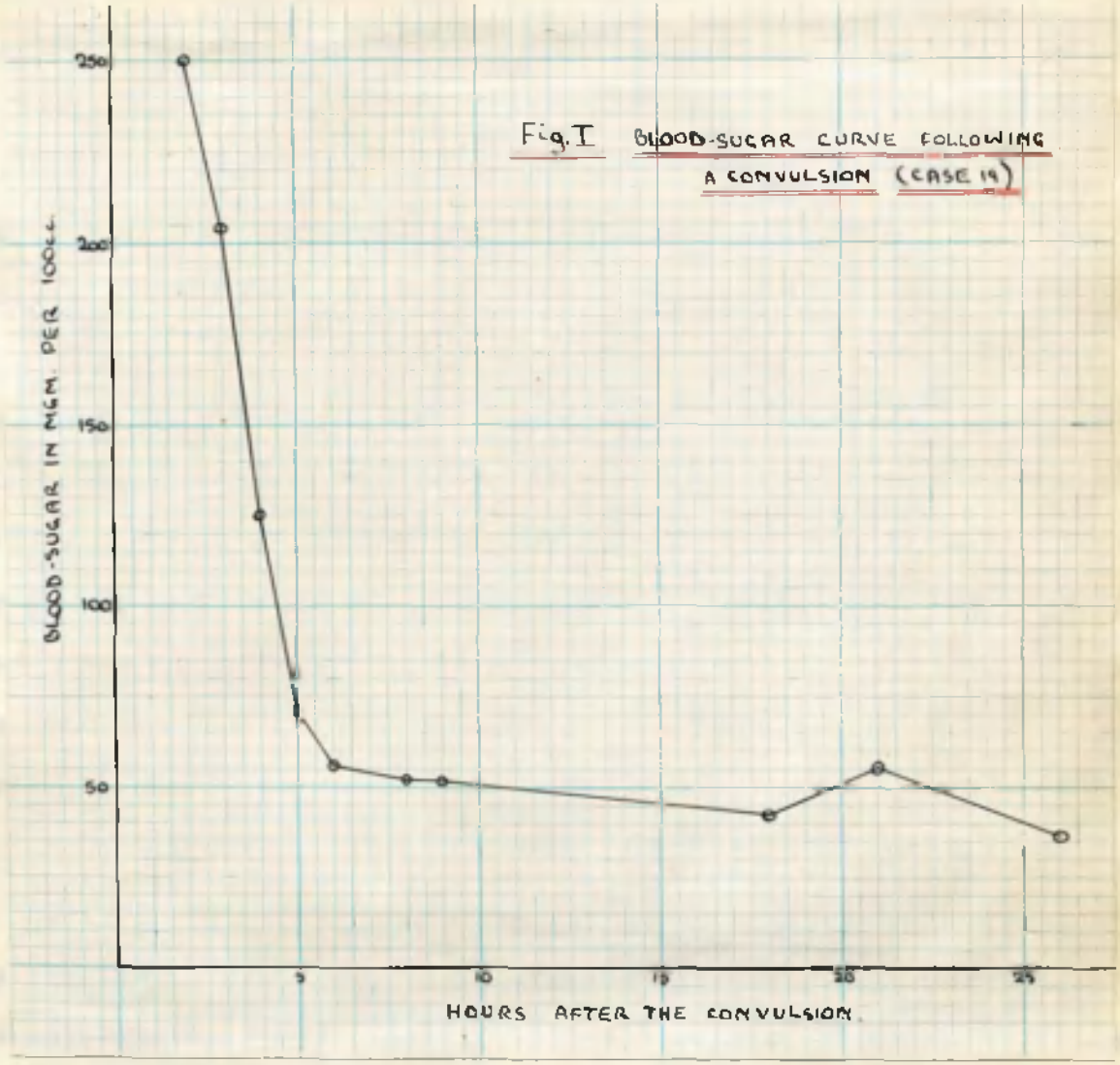


Fig. I BLOOD-SUGAR CURVE FOLLOWING A CONVULSION (CASE 19)

Reverting for a moment to Table III, it will be seen that the hyperglycaemic readings occur most frequently in the first three hours after the convulsion and often persist into the second, whereas the hypoglycaemic readings with one exception are limited to the last three periods. The exception was in the case of J.R. (Case 39), a child of nine months, admitted with profound toxaemia due to ileo-colitis. The child had a

convulsion on admission to hospital and the blood-sugar then was 103.1 mgm. per cent.

She rapidly became moribund and three hours later the blood-sugar had fallen to 33.0 mgm. per cent. The child died four hours after admission to hospital and the blood-sugar immediately after death was found to be 12.0 mgm. per cent.

The time of occurrence of hypoglycaemia was very variable but was usually about twelve hours after the onset of the fit. Frequently it persisted for many hours but occasionally it was very transient and it is possible that it was missed on several occasions in the interval between two observations. Certainly in a few cases the blood-sugar was traced from high to relatively low levels but just failed to sink below the arbitrary point of 50 mgm. per cent.

Figures II and III illustrate this point.

An interesting feature of this hypoglycaemic phase is the comparative lack of symptoms and signs of hypoglycaemia. Several of the older children were slightly drowsy and irritable when disturbed but the majority seemed normal. It is possible that in the infants moribund from gastro-enteritis the twitching of the limbs which persisted till death was due, in fact, to hypoglycaemia, but that the initial convulsion was not hypoglycaemic is shown by the fact that the fall in blood-sugar was traced in two cases (Case 20 and Case 26).

This absence of any clamant symptom of hypoglycaemia when the blood-sugar was at undoubtedly low levels is in accord-

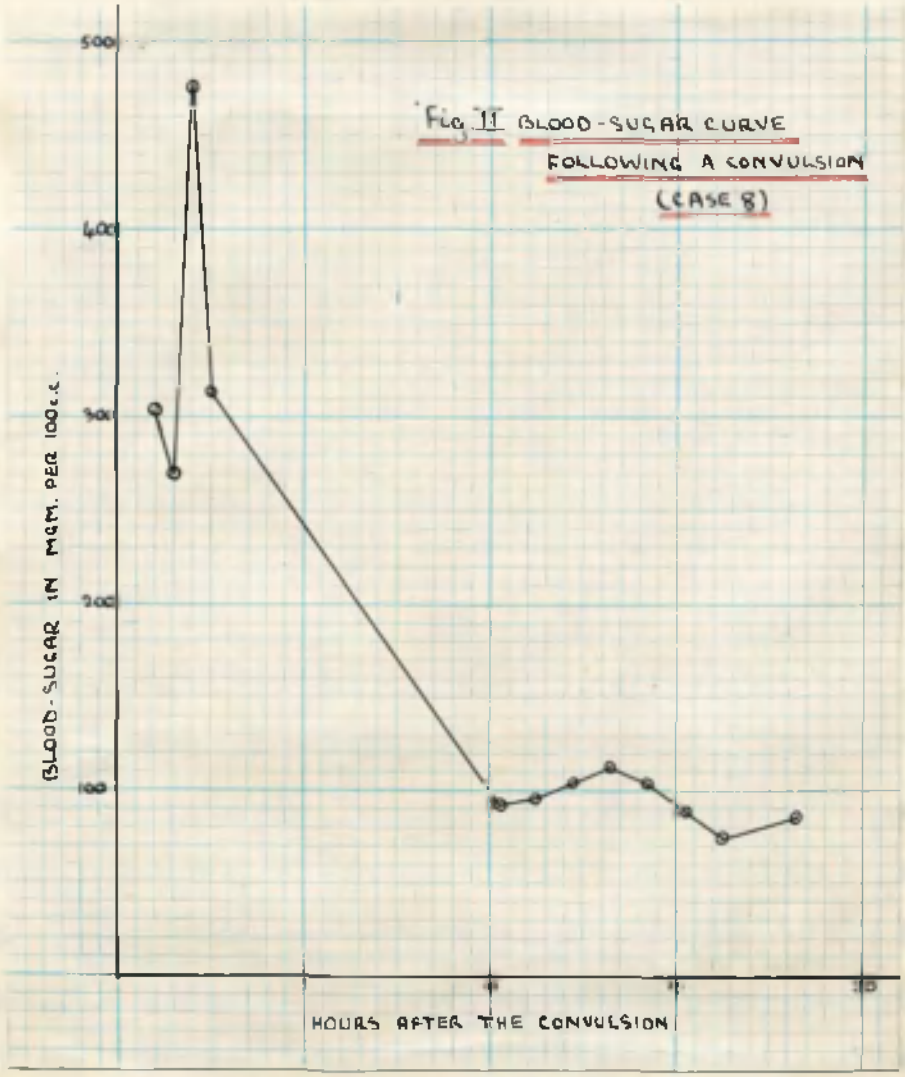


Fig. 11 BLOOD-SUGAR CURVE FOLLOWING A CONVULSION (CASE 8)

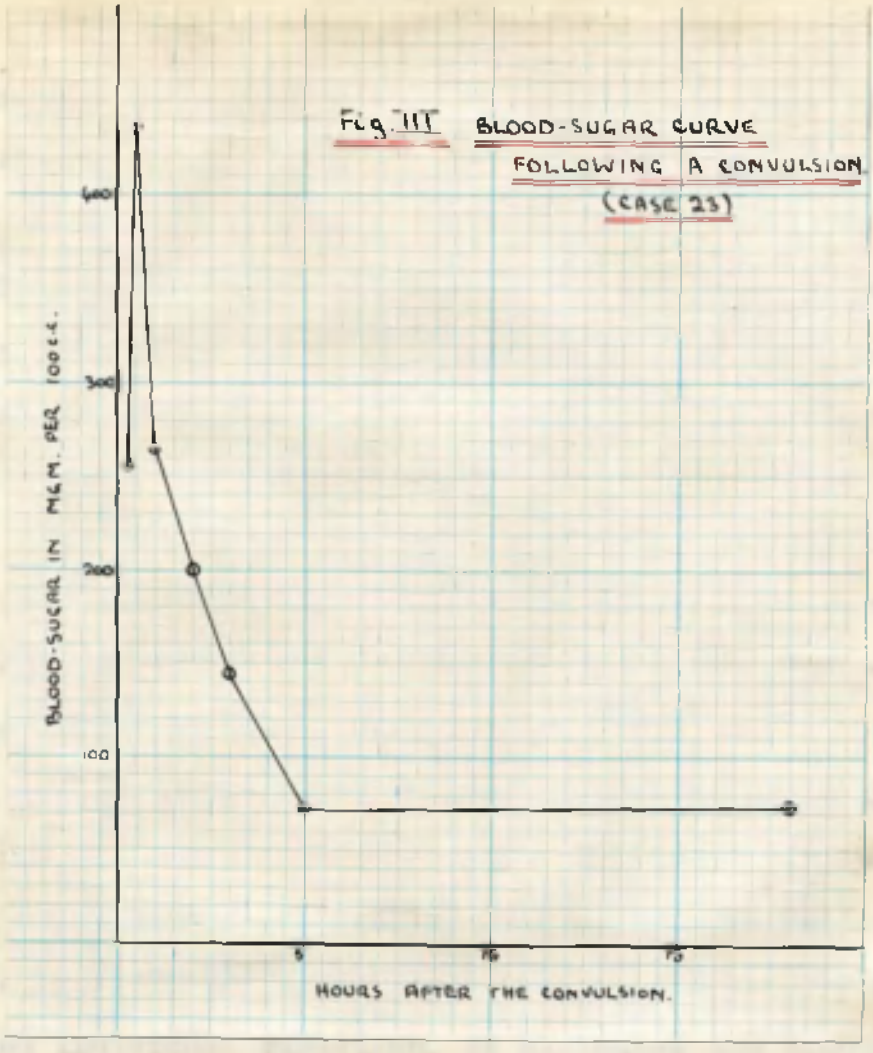


Fig. III BLOOD-SUGAR CURVE FOLLOWING A CONVULSION (CASE 23)

ance with the findings of Harrison[83] and Ashe et alia[84] in children. The latter workers noted that children tolerated hypoglycaemia much more readily than adults, the level of the blood-sugar having to be very low before symptoms became manifest. Shaw and Moriarty[32] observed very few symptoms of hypoglycaemia while fasting epileptic children, though the blood-sugar fell steadily for some days.

Carbohydrate was not withheld from these children unless where specifically stated so that none of the blood-sugar readings can be regarded as fasting values. Since most of these

patients were moderately ill their diet was limited to fluids in the form of whole milk and sugar in appropriate quantities for age every four hours. It was noted that while the blood-sugar might show a transient rise after a feed, the general tendency to fall was unaffected, and indeed in several instances the administration of carbohydrate made no appreciable difference.

The question arises whether the hypoglycaemia observed in a certain percentage of these cases was a purely temporary disturbance or whether it was due to some associated error of carbohydrate metabolism. Sugar tolerance curves were done on certain of the children and in many more, repeated estimations of the fasting blood-sugar were made during convalescence. It will be seen from Table XVII that the average fasting level in these patients was normal, and that though the curves presented individual variation, in all cases the blood-sugar returned to normal levels within two hours. In no case was sugar observed in the urine during the tolerance test. It was not possible to examine the sugar tolerance in all cases since a large percentage of these children died soon after admission, but there can be little doubt that the hypoglycaemia was a purely temporary phase and part of the disturbance of the blood-sugar resulting from the convulsion.

Reference has been made earlier to the suggestion of Josephs[23, 24] and others[25, 65] that hypoglycaemia played a large part in the convulsions of early life. Josephs felt that

TABLE XVII.

Mean fasting levels and sugar tolerance curves in patients showing hypoglycaemia.

No.	Name	Age in years	Mean Fasting Blood-Sugar. mgm.%	Sugar Tolerance Curve.					Amount of glucose. gm.
				F.	½ hr.	1 hr.	1½ hrs.	2 hrs	
1	J.D.	2	71.4						
2	W.M.	3 7/12	82.6	90.1	116.3	106.4	100.0	71.4	15
3	E.G.	1 11/12 (a)	82.6	82.6	188.6	153.8	126.5	101.0	9
		2 2/12 (b)	70.9	87.7	125.0	147.0	135.1	70.4	10
4	E.F.	1 6/12	77.5						
5	J.K.	4 9/12	63.9	56.5	133.3	116.3	116.3	54.7	15
6	D.N.	3/365	90.9						
7	D.W.	2 3/12	76.9						
8	E.O'N.	5 9/12	77.5	71.9	169.5	120.5	86.2	61.7	14
9	J.C.	3/365	90.9						
10	M.C.	12/365	69.4						
11	D.McL.	5 9/12	61.3	59.9	69.9	80.0	69.1	77.5	11
12	B.T.	3/365	90.0						

the combination of a short fast and fever produced a state of hypoglycaemia which might explain the convulsions at the onset of an acute illness. In his first article on the subject he reported ten cases, in seven of whom blood-sugar estimations were not made. In the remaining three the blood-sugar readings are as follows:-

Case 8. 65 mgm. per cent. - 10 hours after the fit.

Case 9. 60 do. - time not stated.

Case 10. 58 do. - 'some hours' after.

One is impelled to make two criticisms, firstly that since only one observation was made of the blood-sugar and that many hours after the convulsion it seems illogical to assume that the convulsion was the result of hypoglycaemia. Secondly and of less importance is the fact that the actual blood-sugar values reported are not particularly low, but in this connection allowance must be made for variations in the technique of the estimation. In a later paper (24) four further cases of spontaneous hypoglycaemia are reported. Two of these children died and at autopsy there was evidence of gross liver damage taking the form of cirrhosis in the first case, and fat accumulation in the second, in whom the question of phosphorus poisoning was raised. There can be little doubt that the hypoglycaemia was secondary to hepatic insufficiency and not truly 'spontaneous'. It would appear therefore that neither of these two cases is relevant to the question of whether spontaneous hypoglycaemia plays a part in the convulsions of early life. In the third case a blood-sugar value of 52 mgm. per cent. was obtained one hour after the convulsion but the child recovered consciousness rapidly without glucose; four hours later the blood-sugar was 56 mgm. per cent. In the last case quoted, the patient, a girl of seven years, was admitted to hospital drowsy and dehydrated.

There was no record of a convulsion nor of how long the patient had been in that condition. The blood-sugar on admission was 40 mgm. per cent. and the urine contained abundant acetone. Dextrose was administered intravenously and orange juice by mouth, but the drowsiness persisted for eighteen hours - a significant observation to which further reference will be made later. No further blood-sugar estimations were made till two weeks had elapsed when the fasting level was found to be 73 mgm. per cent.

Griffiths[25] made similar observations of an association between hypoglycaemia and the convulsions of early life, but hesitated as to whether the relationship was aetiologic. He reported in some detail the clinical history of a child with repeated convulsions. On several occasions the blood-sugar was found to be low, e.g. 20, 28 mgm. per cent. some hours after a fit and he responded well to intravenous glucose. On the last occasion reported, the blood-sugar was 36 mgm. per cent. within twenty minutes of the child becoming unconscious. It was noted however that the boy's carbohydrate metabolism was abnormal in that, even when massive doses of dextrose were given, the rise in blood-sugar was slow and imperfect, and it was considered that there was a persistent tendency to a more or less low blood-sugar content. It seems likely that this case is one of true spontaneous hyperinsulinism such as has been reported by Finney[85] and others[63, 64] in adults and children. On the other hand, the author proceeds to report other cases where the

blood-sugar at a varying number of hours after the convulsion was at a low level and most of these bear a striking resemblance to cases in the present series. It was noted that a low blood-sugar content might exist without convulsions.

Considering therefore these cases reported by American workers in the light of the results obtained in this investigation, it would appear that a misconception has arisen of the part played by hypoglycaemia in the convulsions of childhood. One is able to confirm their observation that a state of hypoglycaemia commonly exists following a convulsion, but one is bound to consider whether they did not, in fact, miss the early and often transient hyperglycaemia, since most of their observations were solitary and made several hours after the attack. Darrow[30] repeatedly observed the blood-sugar after a convulsion in two mentally retarded children. Usually the reading was low but on two occasions definite hyperglycaemic values were recorded, (204 mgm. per cent. and 180 mgm. per cent.) and in no instance was the period elapsing between the onset of the fit and the procuring of the specimen of blood noted. The pertinent observation was made in this paper that the administration of insulin to both these children, though producing a striking fall in the blood-sugar, did not precipitate a convulsion. The effect of intravenous glucose on these patients was never striking and often negligible.

A recent communication by Rector and Jennings[65] discusses functional hypoglycaemia with reference to recurrent

convulsions. Assuming blood-sugar values below 70 mgm. per cent. to represent hypoglycaemia these workers show isolated readings at an unstated period after the convulsions, in eight cases. In two others the sugar content of the cerebro-spinal fluid alone was estimated and found to be very low. It is almost certain that these observations also represent only one phase of the disturbance of the blood-sugar following a convulsion.

Elias and Turner[86] report four cases of what they term spontaneous hypoglycaemia in children. In the first two cases the blood-sugar was estimated after a period of coma lasting five and four hours respectively. No actual convulsive attack was noted in any of these children but since two of them were found unconscious in bed it may be that this was missed. The third case was probably one of true spontaneous hyperglycaemia and in the fourth the blood-sugar was not estimated until after the exhibition of dextrose. The first two cases, however, almost certainly belong to the group under discussion, where an initial stage of hyperglycaemia excited by cerebral stimulation is followed by a more or less prolonged phase of hypoglycaemia.

Various other workers have been sceptical about the role of hypoglycaemia in convulsions in children. Peterman[26] in five hundred cases was unable to establish a diagnosis of hypoglycaemia in any one of them, while Brown[27] reviewing four hundred cases, found only one with hypoglycaemia, though blood-

sugar estimations were made on admission, on all patients in whom the aetiology was obscure. The child in question had a blood-sugar content of 29 mgm. per cent. but the convulsion had occurred on the day before admission. Darrow[30] did not find that hypoglycaemia occurred commonly in children admitted to his hospital with convulsions, his observations being limited to one reading.

It is interesting to note here, that of the total seventy-four cases the lowest blood-sugar content which was observed on admission, when the child was seen within three hours of the onset of the convulsions, was 65.3 mgm. per cent.

It would therefore appear that true spontaneous hypoglycaemia persisting to the point of producing convulsions, is a very rare condition in children. A false conception of its frequency has been established from incomplete observation of the behaviour of the blood-sugar after a convulsion.

(a) Adrenaline curves in the hypoglycaemic state.

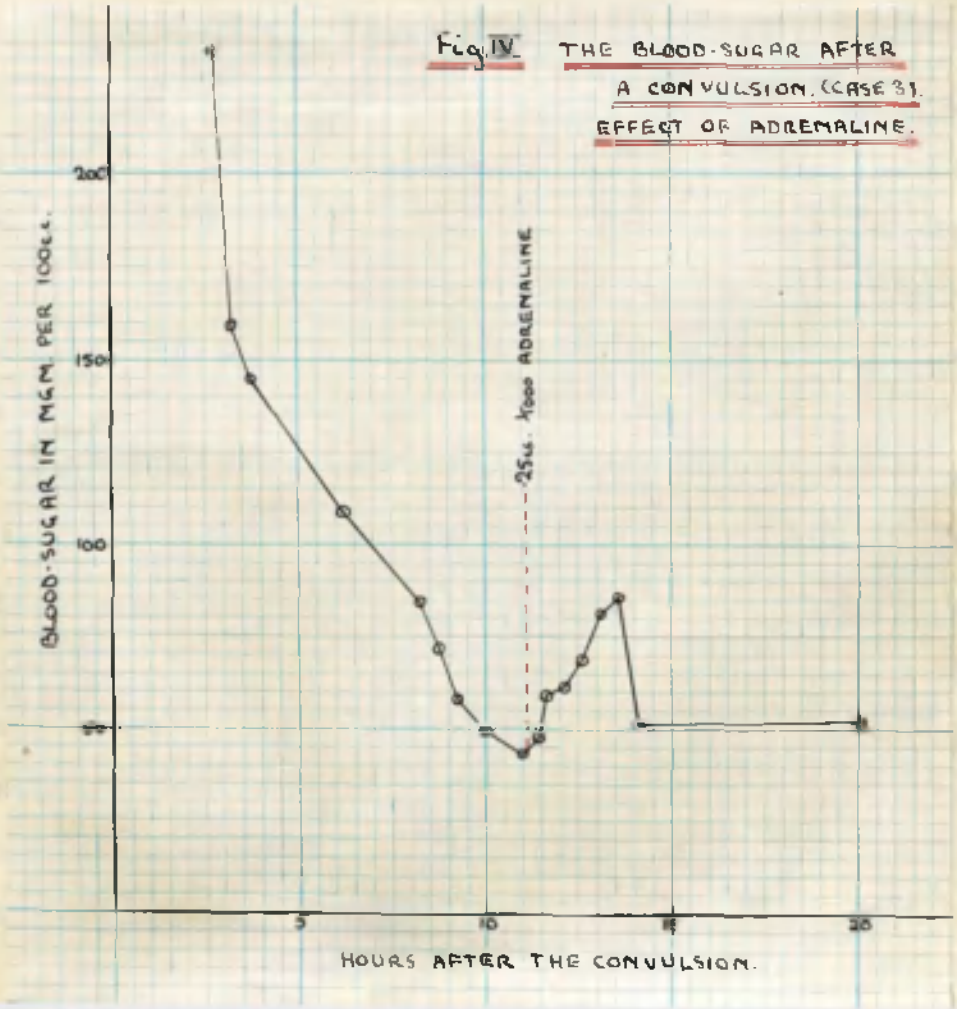
Evidence having therefore been brought forward to show that in a fair percentage of cases of convulsions the blood-sugar mechanism is profoundly upset to the point of producing a state of hypoglycaemia, the question arises as to the actual cause of this hypoglycaemia. Two possibilities emerge. It may be that the violent muscular exertion of the convulsion uses all the available carbohydrate, leaving the body tissues in a state of carbohydrate depletion. In this connection it may be

recalled that strychnine convulsions have been experimentally used to rid the liver of its glycogen and that Levine[87] noted hypoglycaemia in marathon runners. But it has been shown that carbohydrate was not withheld from these patients during the period when the observations on the blood-sugar were being made. It was repeatedly noted that the general tendency of the blood-sugar to fall was more or less independent of the ingestion of carbohydrate, though a transient rise might occur after a meal. Moreover it is generally recognised that even during complete starvation the blood-sugar does not fall very appreciably below the fasting level, owing to the formation of glucose from endogenous sources, presumably protein and fat. In the patients here described, even though the glycogen stores had been reduced to a low level, which was probably not the case as they all received food, there should not therefore have been hypoglycaemia had there been an adequate stimulus for the production of glucose.

As Mann and Magath[88] have emphasised, the burden of maintaining the blood-sugar at a constant level falls upon the liver, while Lawrence[89] recently pointed out that, when there is any deficiency of adrenal, thyroid, or pituitary secretion the resultant hypoglycaemia is probably due to an undue stability of the liver glycogen. In view of the frequent occurrence of hyperglycaemia prior to the hypoglycaemic phase it seems probable that in many of the present series of patients there was an initial over-secretion with consequent exhaustion of the store of adrenaline. If this reasoning is correct administration of adrenaline should readjust, at any rate temporarily, the level of

the blood-sugar. This hypothesis was put to the test on three occasions. Blood-sugar examinations were carried out in the usual way on three children who had had convulsions. When the hypoglycaemic stage was reached adrenaline was injected and the blood-sugar estimations continued at frequent intervals.

The first child in whom the procedure was tried was E.F. (Case 3) whose history and blood-sugar findings have already been described at length (p. 9). It will be recalled that the blood-sugar fell in about eight hours from 232.6 mgm. per cent. to 42.8 mgm. per cent. At 5 p.m. when the blood-sugar was at the latter level 0.25 c.c. adrenaline (1/1,000) was injected subcutaneously. Figure IV shows the changes that occurred.



It will be seen that there was an immediate response. The blood-sugar rose to over 80 mgm. per cent. within two hours of the injection and within three hours of the beginning of the experiment had fallen to its original level. The child was kept fasting from noon for the purposes of this experiment so that the rise in blood-sugar cannot be attributed to the taking of food.

In the second case D.Mc.L., (Case 16) the initial rise in the blood-sugar level had been missed and the observations only began in the hypoglycaemic phase (p.15). Some months previously, however, the child had had a similar convulsion and on that occasion the blood-sugar tests had commenced within three hours of the seizure and the hyperglycaemic phase (188.7 mgm. per cent.) had been registered. On the second admission the boy was brought to hospital eighteen hours after a similar convulsion. At that time he was conscious but drowsy and the blood-sugar level was found to be 36.5 mgm. per cent. There can be little doubt that with the second convulsion there had been a rise similar to that which occurred with the previous convulsion. Figure V shows the changes which occurred after giving 0.5 c.c. adrenaline (1/1,000) subcutaneously during the hypoglycaemic phase. It will be seen that there was an immediate response, the blood-sugar rising to a level of 169.0 mgm. per cent. one hour forty-five minutes after the injection. No food had been given for at least four hours previous to the adrenaline injection.

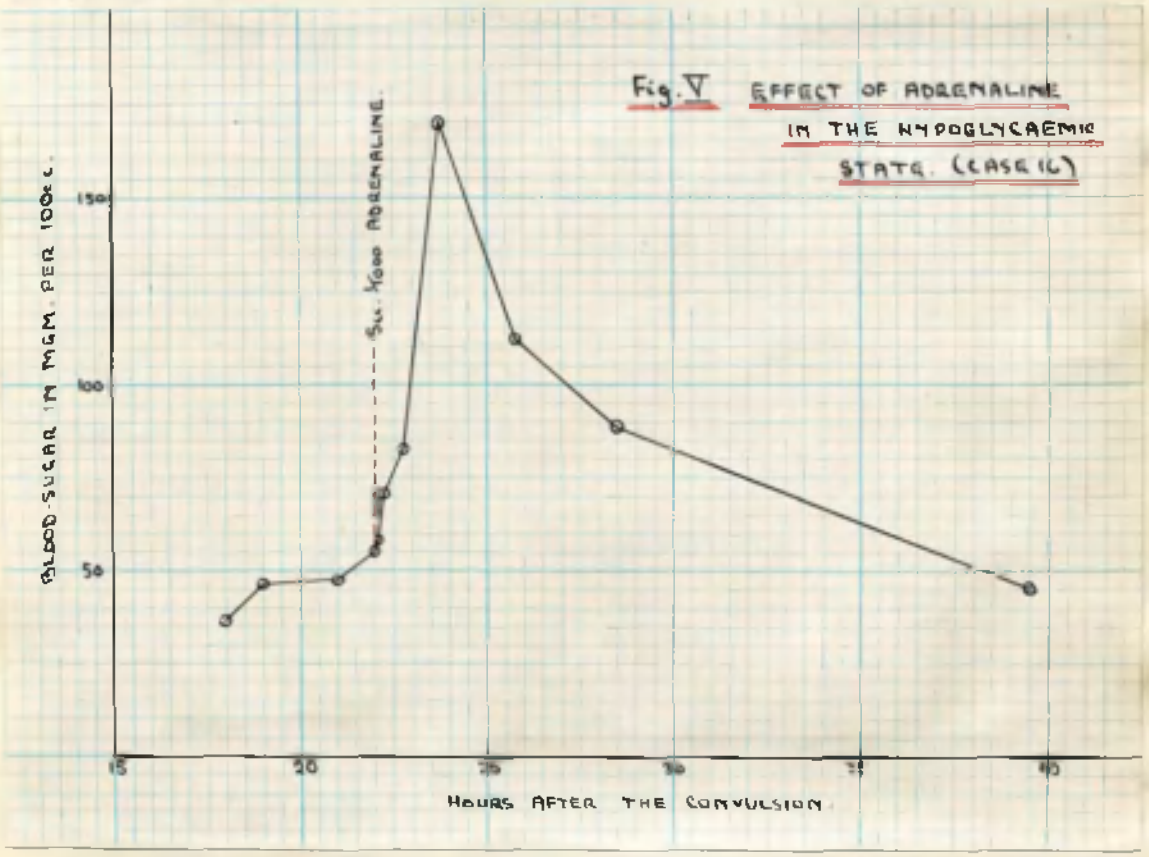
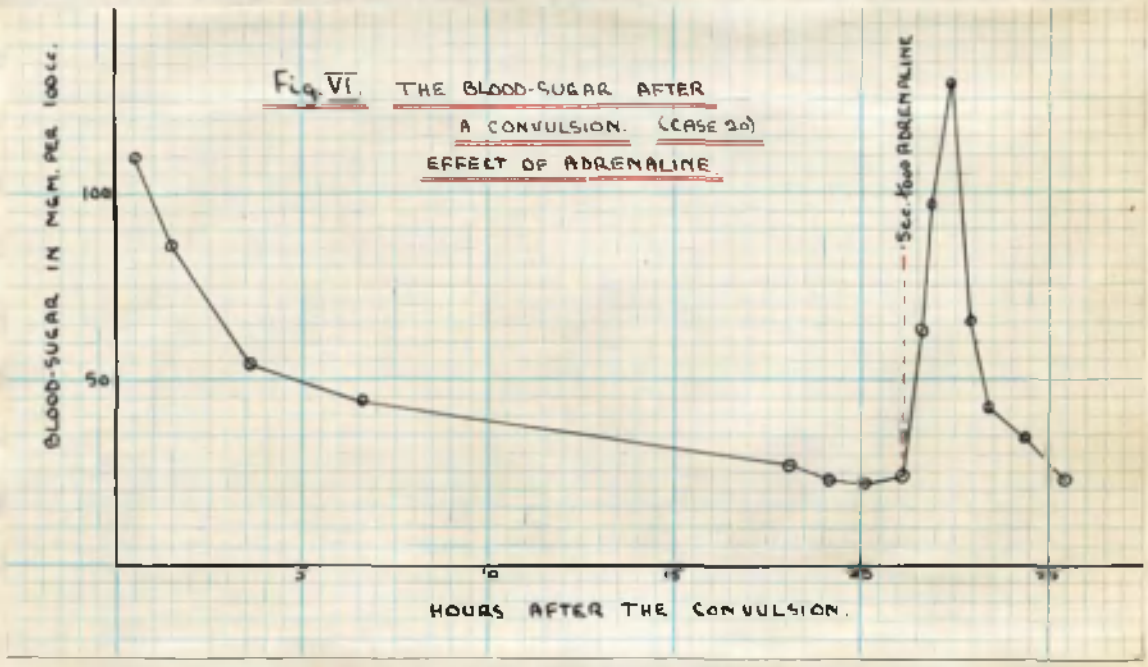


Fig. V
EFFECT OF ADRENALINE
IN THE HYPOGLYCAEMIC
STATE. (CASE 16)
BLOOD-SUGAR IN MGM. PER 100 c.c.
150
100
50
0.1 c.c. 1/1000 ADRENALINE.
15
20
25
30
35
40
HOURS AFTER THE CONVULSION.

The third patient, C.N. (Case 20) was admitted to hospital half-an-hour after a generalised convulsion lasting about ten minutes and was found to be suffering from gastro-enteritis (p. 47). The blood-sugar content on admission was 109.9 mgm. per cent. and it fell rapidly to 44.4 mgm. per cent. in six hours. Twelve hours later it was just over 20.0 mgm. per cent. and at 12.45 p.m. when the level was 24.6 mgm. per cent. 0.5 c.c. adrenaline (1/1,000) was injected subcutaneously.



The immediate response will be seen in Figure VI, and it is interesting to note the rapid return to a low level. At 9.30 a.m. the next day the blood-sugar level was 32.8 mgm. per cent. but unfortunately the child was removed from hospital against advice and further investigation was impossible.

Food was purposely withheld from these three children

during the three hours following the injection of adrenaline so that the blood-sugar curve might not be obscured but thereafter the usual milk and sugar was instituted. Despite this, however, in two of the three cases the blood-sugar was still at a very low level the next morning. Thus it would appear that in some cases the hypoglycaemic phase is prolonged, and persists in spite of the ingestion of food.

The question arises as to why it was not possible to demonstrate this hypoglycaemic phase in all the cases of convulsions which showed an early hyperglycaemia. Firstly there is no doubt that in some cases the low period was missed, especially when it occurred during the night, since it was clearly not feasible to prolong the investigation while the child was asleep. This is almost certainly what happened in Cases 1, 23 and 29. In three cases - 2, 7, and 45 - the estimations were unfortunately stopped before the period at which the hypoglycaemic phase usually manifested itself. One child died (Case 21) while the blood-sugar was still at a high level.

Secondly it is possible that in some cases the adrenal store was adequate and recovery of function was rapid. In Cases 8, 13, 27 and 59 a marked fall in the blood-sugar was demonstrated though no reading below 50 mgm. per cent. was obtained. It may be that in the interval between two estimations the blood-sugar fell to a lower level but the adrenals having been not quite exhausted, quickly recovered function and releasing sugar from the liver restored the blood-sugar to a normal level.

Name	Maximum Blood-Sugar. mgm.%	Minimum Blood-Sugar mgm.%
M.McL.	476.1	75.2
C.McA.	204.1	72.9
E.M.	232.5	59.1
M.B.	312.5	63.2

These adrenaline curves demonstrated three points:-

(1) There was glycogen in the liver had there been an adequate stimulus for its conversion into glucose. Thus the theory that the hypoglycaemia following convulsions is consequent upon exhaustion of liver glycogen is untenable.

(2) Since adrenaline raised the blood-sugar to normal levels it is reasonable to assume a temporary adrenal inadequacy.

(3) In these cases there is an over-action of insulin. It is well established that the level of the blood-sugar at any given moment is the result of the balance between the opposing actions of insulin and adrenaline along with certain other subsidiary factors. Thus in the absence of a sufficient supply of adrenaline, insulin action, which Lawrence[89] believes to be simply the conversion of glucose into glycogen, proceeds unopposed. This explains why only a transient and sometimes insignificant rise in

the blood-sugar was observed in these children after the administration of carbohydrate. Levy Simpson[76] has stressed the fact that insulin retards hepatic glycogenolysis.

(b) Adrenaline curves in normal children.

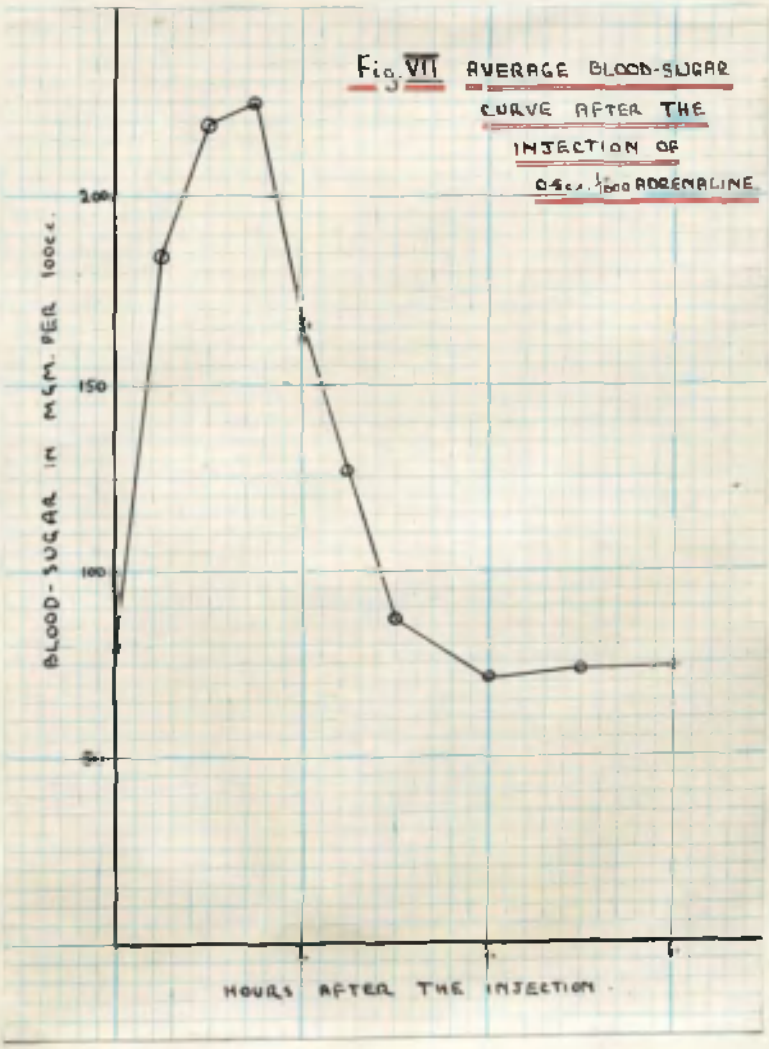
Adrenaline was administered in doses of 1 c.c. by subcutaneous injection to six children convalescent from various diseases and after a fast of about nine hours.

TABLE XVIII.

Blood-Sugar Curves following the injection of 1 c.c. Adrenaline 1/1,000 subcutaneously.

No.	Name	Age in years	BLOOD SUGAR - mgm. per 100 c.c.										Disease.
			Fasting	½ hr.	1 hr.	1½ hrs.	2 hrs.	2½ hrs.	3 hrs.	4 hrs.	5 hrs.	6 hrs.	
1	R.R.	5 9/12	61.1	163.9	169.5	-	85.4	61.1	48.7	64.1	57.1	-	Unresolved pneumonia.
2	J.M.	10	71.4	181.8	208.3	256.4	250.4	204.1	116.2	68.0	75.2	-	Hilum tuberculosis.
3	T.B.	12	93.4	172.4	263.2	196.1	117.6	79.3	61.3	74.1	76.3	84.7	Pyuria.
4	A.W.	6	77.5	222.2	204.0	181.8	120.4	75.0	71.9	78.7	81.3	80.0	Bronchitis.
5	T.L.	10	86.9	166.6	192.3	185.1	158.7	136.9	97.1	64.5	59.5	56.2	N.A.D.
6	J.T.	8	89.2	196.1	270.2	303.2	263.1	204.1	126.5	74.6	91.7	-	Hilum tuberculosis.
Average:			79.9	183.8	217.9	224.5	165.9	126.8	87.0	70.7	73.5	73.6	

It will be seen that some variation normally exists as to the time of occurrence of the highest point of the curve, and also as to the height of the peak. Similarly there is considerable variation in the time taken for the blood-sugar to return to normal levels. In all cases the blood-sugar fell below the original fasting level at some point showing that the induced hyperglycaemia had stimulated a flow of insulin. There was however no tendency for the lower blood-sugar levels to persist, which points to an adequate supply of endogenous adrenaline. Figure VII shows a graph of the average adrenaline curve.



It will be seen, therefore, that the blood-sugar curve following a convulsion of some severity bears a striking resemblance to that following the administration of adrenaline. This bears out the theory that the marked rise in the blood-sugar which may occur after a convulsion is the direct result of a massive increase in the secretion of adrenaline. The hyperglycaemia thus established now excites a flow of insulin though the actual mechanism by which this occurs is obscure. Macleod (90) is of the opinion that the high blood-sugar excites some centre in the brain which then discharges through the vagus, impulses increasing the secretion of insulin. However this may be, insulin action once established, steadily lowers the blood-sugar. As we have seen, this action is a little over-done in the normal child but though the blood-sugar may fall to a lower level than at the outset, there is a ready and rapid swing back to the average fasting level. In the post-convulsive state there is no such process of recovery. There appears to be a temporary inadequacy of adrenaline secretion so that insulin action proceeds unchecked by the glycogenolytic action of endogenous adrenaline.

6. The Urine in Convulsions and Coma.

The urine was examined in sixty of the total seventy-four cases; ten of the remainder died before a specimen could be procured while the other four passed urine involuntarily before an appliance to receive the urine had been fixed. Thus the first and most important specimen was unfortunately lost. Glycosuria of varying degree was observed in seventeen cases, eleven of whom were seen soon enough for the hyperglycaemic phase to be recorded. Since the majority of these children had a clear-cut history of previous convulsions the question of differential diagnosis from diabetic coma did not arise, but it is conceivable that such a difficulty might occur. Fleming, Herring and Morris[29] have described three cases of coma associated with glycosuria, where such a diagnosis was tentatively made and later found to be untenable. Woolley[5] pointed out that the occurrence of glycosuria associated with slight acetonuria in a comatose patient might lead to errors in diagnosis, and reported four cases of subarachnoid haemorrhage exhibiting these signs and symptoms. Thirteen cases of cerebral haemorrhage associated with glycosuria were reported by Römcke and Skouge[3] who were of the opinion that the latter was due to a large increase in the secretion of adrenaline. Even earlier than this Tychowski and Crowell[66] compiled from the literature thirty-four cases of cerebral haemorrhage, etc., where either a high blood-sugar or glycosuria was noted and suggested that an increased output of epinephrin following

central stimulation might be responsible for the sugar mobilisation.

TABLE XIX.

The urine in convulsions and coma.

No.	Name.	URINE			
		0-8 hours		8-16 hours	
		Sugar	Acetone	Sugar	Acetone
1	J.D.	++	-	-	++
2	E.G. (a)	-	-	-	+
	(b)			tr.	++
3	A.B.	+	++		
4	H.K.	+	+		
5	M.McL.	+	+	+	+
6	C.McA.	+	-		
7	A.M.	+	-		
8	J.W.	+	+		
9	M.B.	++	+	+	+
10	E.M.	+	-		
11	W.M. (a)			tr.	++
	(b)	-	-	-	+
12	J.K.	tr.	+	-	++
13	J.W.	++	-	-	++
14	E.O'N.			+	++
15	D.M.	tr.	-	-	-
16	M.B.	+	+		
17	T.H.	tr.	tr.	-	++

Acetone did appear in the urine in small amounts in some of the cases but it was more abundant in the second specimen obtained eight to sixteen hours after the fit. This would seem to be a diagnostic point. Of the cases reported by Fleming, Herring and Morris,[29] the urine of the first contained on admission a moderate amount of acetone, that of the second a faint trace, while that of the third contained none. In the third case acetone was detected in the urine ten hours after the onset of the seizure. Similarly in Woolley's[5] cases acetone could be detected by Rothera's test but not by Gerhardt's, i.e. the amount present was not gross. These findings are in complete contrast to what is found in true diabetic coma where the urine is loaded with acetone from the beginning.

Linked with this question of acetonuria is another diagnostic point - the presence or absence of the Kussmaul type of respiration or 'air-hunger' which is absolutely typical of a severe non-gaseous acidosis and is quite unmistakable. Though in many of our cases the breathing on admission was stertorous this was obviously due to asphyxia from muscular spasms and true 'air-hunger' was never observed. In addition, patients suffering from diabetic coma are never cyanosed except at a late stage where heart failure is incipient; indeed the colour of their lips has been described as 'cherry-red'. Patients comatose from convulsions, on the other hand, are usually cyanosed and rigid or may be very pale.

Acetone may be readily detected in the breath of

children comatose from other causes but it is never so overpowering as in the case of hyperglycaemic diabetic coma.

The importance of diagnosis rests in the fact that the administration of insulin to such cases as have been described involves the definite risk of death from hypoglycaemia, since, as we have seen, the endogenous insulin is enjoying a period of unusual and practically unrestrained activity. Generally speaking, the history of the patient will preclude any such mistakes being made, but circumstances might arise in which a history was unobtainable, inadequate or atypical and there seems to be justification for the plea that the question of cerebral disturbance associated with glycosuria should be considered, before treatment is instituted in a child found comatose and showing sugar in the urine.

B. STUDY OF THE BLOOD-SUGAR IN HEAD INJURIES, FRACTURES AND BURNS.

In view of the findings in the blood-sugar in a large proportion of children following convulsions, it was thought that the study might profitably be extended to include other causes of disturbance of the central nervous system such as head injury with or without fracture, fractures of limbs and burns.

That cranial trauma may be associated with glycosuria was recognised many years ago by Claude Bernard[1], who found abundant sugar in the urine of a rabbit dead from a blow on the skull. In thirty-three cases of head injury of varying degree Berberich[8] found high blood-sugar values in seventeen, while in the remaining sixteen the blood-sugar was at the upper limits of normality. Lurjé[91] was of the opinion that cranial trauma was only associated with hyperglycaemia when it was accompanied by haemorrhage or a condition of shock. Some years ago, two Italian[92] workers, noting high blood-sugar values in surgical shock, concluded that these were due to hyperadrenalinaemia. They postulated an early hyper-function of the adrenals causing a rise in the blood-sugar, followed by a state of hypo-function involving a rapid fall to a low level. Amorosi[93] corroborating the rise in blood-sugar in shock, pointed out that the degree of the increase was roughly proportional to the intensity of the shock. The recent work of Schweers[9] demonstrated that

following cranial trauma there is a disturbance of carbohydrate metabolism which may not rapidly disappear.

The fact that fractures, other than cranial, could provoke glycosuria was recognised many years ago by Konjetsny and Weiland.[94] In experimental work on rabbits Horikawa[95] noted a marked rise in the blood-sugar following fracture of a long bone. This rise was absent or negligible in cases where the splanchnic nerves had been previously sectioned and the author concluded that it was due to an increased secretion of adrenaline excited by splanchnic stimulation. Berberich[8] on the other hand in thirteen cases of limb fracture found a high blood-sugar value in only one. Recently Funsten[96] reported a case of multiple fractures in an elderly woman, in whom the blood-sugar rose to over 300 mgm. per cent.

Turning to the question of the blood-sugar following burns and scalds the experimental work of Ollbrycht[97] merits some attention. He was able to confirm the earlier observations of Kolisko[98] and Weiskotten[99] that the adrenal glands of individuals dead from burning showed marked degenerative changes, and emphasised that in the primary phase of shock following a burn the function of the glands is much increased and it is only later that exhaustion of function ensues. Many of his animals died in hyperglycaemia a few hours after the burning, but where the initial shock was overcome Ollbrycht advocated the stimulation of the weakened chromaffin system by the administration of adrenaline. Luksch[100] made the

interesting observation that there was a diminished amount of adrenaline in the adrenals after burning. Of twenty-five cases of burning and scalding Davidson[101] observed either hyperglycaemia or glycosuria in six, and noted that the degree of elevation of the blood-sugar was usually directly proportional to the severity of the burns. Greenwald and Eliasberg[102] reported two fatal cases of burning in children, associated with extreme hypoglycaemia. This led them to observe the behaviour of the blood-sugar following experimental burns in rabbits, and it was noted that a marked rise occurred uniformly in all rabbits shortly after the burn. The animals might die at this stage but if they lived there ensued a period of hypoglycaemia which the authors thought was due to suprarenal exhaustion and consequent inhibition of glycogenolysis. In the initial stage of shock adrenaline was contra-indicated, while in the second stage its administration in large and frequent doses appeared beneficial. Riehl,[103] on the other hand, from experimental work came to the conclusion that adrenaline secretion was unaltered by burning and Slocum and Lightbody[104] though they noticed a rise in the blood-sugar following burning did not attribute this to increased suprarenal activity.

An effort was made in these surgical cases to follow the behaviour of the blood-sugar after the injury in exactly the same way as had been done in the cases of convulsions.

TABLE XX.

Maximum and minimum blood-sugar readings following fracture of the skull.

No.	Name	Age in years	Shock	Maximum Blood-Sugar mgm.%	No.of hours after injury	Minimum Blood-Sugar. mgm.%	No.of hours after injury	Glycosuria
1	H.S.	4	++	250.0	3	60.9	23	+
2	G.G.	6	±	86.9	3½	45.8	25½	-
3	W.O.	6	+	106.4	3	45.5	41½	-
4	J.G.	3	++	54.3	24	39.7	27	-
5	R.S.	5 5/12	++	61.7	48	-	-	Urine not obtained.
6	M.A.	7	++	208.3	5	67.5	48	+
7	W.McI.	12	+	167.1	½	50.5	39	+
8	G.K.	5	+	89.5	3	51.3	21½	-
9	T.G.	5/12	±	142.8	4	-	-	Urine not obtained.

TABLE XXI.

Maximum and minimum blood-sugar readings following concussion.

No.	Name	Age in years	Shock	Maximum Blood-Sugar mgm.%	No.of hours after injury	Minimum Blood-Sugar. mgm.%	No.of hours after injury	Glycosuria
1	M.A.	11	-	84.0	½	63.6	23	-
2	J.C.	12	+	82.6	2¾	51.2	21¾	-
3	H.C.	6	+	156.2	¾	65.7	20¾	-
4	J.G.	11	+	87.7	11	61.7	19	-
5	A.McD.	5	++	149.2	2	42.7	22	-
6	J.G.	7	±	100.0	12	69.2	36	-
7	A.M.	8	+	74.7	20½	67.1	23½	-

Though every facility was afforded by the surgical staff it was naturally impossible to obtain specimens of blood with such frequency as in the medical cases. However, in a fair number of cases a moderately complete picture of the blood-sugar was obtained. Table XX shows the maximum and minimum blood-sugar values observed following the injury, in nine children with fracture of the skull, and Table XXI shows similar observations in seven children suffering from concussion. It will be seen that in three children of the former section, either a hyperglycaemic value or glycosuria was demonstrated while in the latter group no such finding was recorded. That the degree of shock had a direct bearing on the question of blood-sugar disturbance is shown by the fact that in no case where the shock was severe and the child seen sufficiently soon after the accident, was the blood-sugar below 140 mgm. per cent. It seems probable therefore that the failure to demonstrate a really high blood-sugar content in any child in the second group is largely due to the fact that, on the whole, the degree of shock was less in this group than in the first. This follows naturally from the fact that, within limits, the violence necessary to produce a fracture of the skull is greater than that necessary to cause concussion; added to this there is the risk of intracranial haemorrhage from tearing of meningeal vessels by the bony fragments and, as we have seen, this in itself is sufficient to cause a profound disturbance of the blood-sugar regulating mechanism.

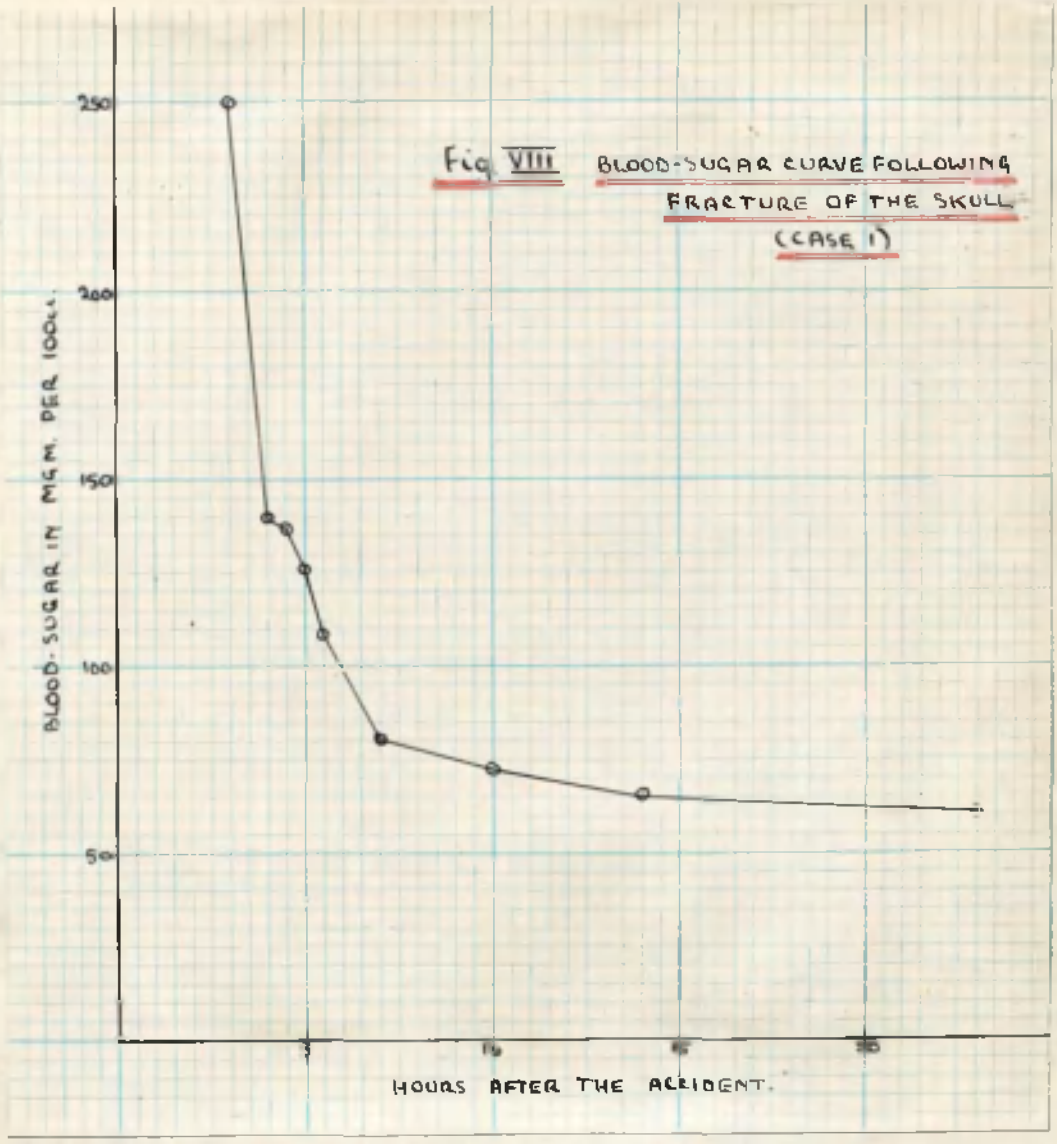
In four of the sixteen children, it was possible to demonstrate a very low blood-sugar value many hours after the injury while in three others the value was just over the arbitrary figure of 50 mgm. per cent. It may seem surprising that no very low value was noted in either of the two children in whom hyperglycaemia was demonstrated in the early stage, but there can be little doubt that the low readings were missed in the interval between observations.

In Case I a striking fall in the blood-sugar was demonstrated though no actual hypoglycaemic value was recorded.

H.S., a girl of four years, was struck on the head by a falling ladder and was admitted to hospital three hours later in a very shocked condition. A skiagram showed multiple linear fractures of the vault of the skull. The urine contained sugar but no acetone.

←--------23/10/35----------→ ←--24/10/35--------→

Time.	1 p.m.	2 p.m.	2.30 p.m.	3 p.m.	3.30 p.m.	5 p.m.	8 p.m.	12 mid-night	9 a.m.	3 p.m.	4 p.m.	5 p.m.
Blood-Sugar. mgm.%	260.0	139.8	136.9	125.7	108.1	80.3	72.4	65.3	60.9	78.1	75.7	62.4
No.of hours after convul-sion.	3	4	$4\frac{1}{2}$	5	$5\frac{1}{2}$	7	10	14	23	29	30	31



It seems likely that between 9 a.m. and 3 p.m. on the day following the accident the blood-sugar fell to a low level.

Case 6. M.A., a girl of seven years, was knocked down by a motor car, sustaining a lacerated wound of the forehead and a linear fracture of the skull. The blood-sugar five hours later was 208.3 mgm. per cent. Unfortunately no further estimation of the blood-sugar was made till forty-three hours later when the value was found to be 67.5 mgm. per cent.

As in the case of the children with convulsions, carbohydrate was administered to these patients in the form of whole milk and sugar at regular intervals, so that the fall in the blood-sugar cannot be regarded as the result of fasting. During the period when the blood-sugar was at a low level the children were drowsy and irritable but no other symptom of hypoglycaemia were noted.

TABLE XXII.

Maximum and minimum blood-sugar readings following fracture of a limb.

No.	Name	Age in years	Shock	Maximum Blood-Sugar. mgm.%	No. of hours after injury	Minimum Blood-Sugar. mgm.%	No. of hours after injury	Glycosuria.
1	C.P.	4	++	270.2	2	75.2	21½	Urine not obtained.
2	F.T.	10	-	100.0	1½	62.8	12	-
3	T.M.	6	-	107.5	4	84.0	21½	-

The blood-sugar was also examined in three children with fracture of a long bone and in only one was a hyperglycaemic value noted. This was in the child C.P. who was admitted to hospital with a fracture of the right femur and suffering from severe shock. The urine was unfortunately not obtained.

←----------28/10/35----------→(29/10/35-->

Time	3.30 p.m.	4 p.m.	4.30 p.m.	5 p.m.	5.30 p.m.	8 p.m.	12 mid-night	9.30 a.m.	11 a.m.
Blood-Sugar. mgm.%	270.2	177.0	156.2	153.0	130.7	142.8	116.3	106.3	75.2
No.of hours after convul-sion.	2	2½	3	3½	4	6½	10½	20	21½

etc.

The next case, F.T., a boy of ten years, was admitted to hospital with a fracture of the right tibia. There was no shock. The urine contained nothing abnormal.

←----------30/10/35----------→ ←----31/10/35--->

Time	2.30 p.m.	3 p.m.	3.30 p.m.	4 p.m.	4.30 p.m.	5.30 p.m.	10.30 p.m.	1 a.m.	9.30 a.m.	10.30 a.m.
Blood-Sugar. mgm.%	100.0	76.9	74.6	86.2	84.0	92.5	73.5	62.8	82.6	69.9
No.of hours after convul-sion.	1½	2	2½	3	3½	4½	9½	12	20½	21½

It will be seen that there was little or no disturbance of the blood-sugar in this case. Similarly in the third case the blood-sugar four hours after the accident was normal in a child with a fracture of the right femur, but showing no sign of shock. In none of these three children was a hypoglycaemic level at any time observed. Though it is impossible to draw conclusions from three cases it seems probable that whether or not the blood-sugar level is affected depends on the presence or absence of shock.

TABLE XXIII.

Maximum and minimum blood-sugar readings following scalds.

No.	Name	Age in years	Shock	Maximum Blood-Sugar. mgm.%	No. of hours after injury	Minimum Blood-Sugar. mgm.%	No. of hours after injury	Glycosuria.
1	T.K.	3 11/12	++	142.8	19½	35.8	16	Urine not obtained.
2	G.D.	1 2/12	+	88.5	6	107.5	22½	Urine not obtained.
3	R.P.	8	+	169.5	1½	-	-	-

In one of the three cases of scalding it was possible to demonstrate the hypoglycaemia which Greenwald and Eliasberg (102) found in two children with extensive burns.

T.K., a boy of 3 11/12 years, was scalded over the thighs and buttocks by boiling water. He was admitted to hospital suffering from severe shock but the blood-sugar was not examined till sixteen hours after the accident when the child was gravely ill, pale and toxic. The urine obtained at the same time contained abundant acetone.

Time	10.30 a.m.	11 a.m.	11.30 a.m.	12 noon	1 p.m.	2 p.m.
Blood-Sugar mgm.%	35.8	62.9	64.1	117.0	130.6	142.8
No. of hours after convulsion.	16	16½	17	17½	18½	19½

It will be seen that the blood-sugar was at a low level when first examined but that it rapidly rose during the course of the day. In view of the child's shocked condition on admission it is highly probable that the blood-sugar was considerably elevated at this

time and that there was a fairly rapid fall.

In the second case the blood-sugar did not appear to be much affected while in the third there was a moderate elevation one-and-a-half hours after the accident. At this point the child was given a general anaesthetic for purposes of treatment and no further estimation of the blood-sugar was made.

Reviewing the results of this short study of the blood-sugar in head injuries, fractures and scalds, it will be seen that a certain amount of evidence has been obtained to show that these conditions may be followed by a state of hyperglycaemia with or without glycosuria. In accordance with Lurjé's[91] findings, it would seem that the main factor in determining the production of such a disturbance in the blood-sugar is the intensity of the shock resulting from the accident. Following the rise in the blood-sugar there is a distinct tendency to a rapid fall sometimes to very low levels, which may be transient, but which frequently persist for many hours. The close parallel existing between this sequence of events and that following a convulsion is immediately evident and there seems no reason to doubt that the physiological mechanism at work is the same in each case. There is intense excitation of the central nervous system from mechanical disturbance, pain and fear, a sudden outpouring of adrenaline into the blood-stream causing glycogenolysis and a rise in the blood-sugar. The fall in the blood-sugar which tends to ensue would appear to be due to adrenal exhaustion and diminished hepatic glycogenolysis. Dogliotti and Giardanengo[92]

postulated in surgical shock an early hyper-function of the adrenals followed by a state of exhaustion, while Ollbrycht[97] and Greenwald and Eliasberg[102] described the same sequence of events in burning. Our results appear to support this hypothesis.

III. THE PATHOGENESIS OF THE DISTURBANCE OF THE BLOOD-SUGAR IN CONVULSIONS, ETC., AND SOME THERAPEUTIC INDICATIONS.

The studies set forth in the foregoing pages have shown that in more than half of the children suffering from convulsions there is a profound disturbance of the blood-sugar. There is also evidence that a similar effect on the blood-sugar may be produced by conditions exciting traumatic shock. The changes in the blood-sugar take the form of a sudden, sharp rise immediately after the convulsion or other stimulus, followed by a fairly rapid fall sometimes to a very low level. It has been recalled that several workers (14, 18, 23, 25) have observed either hyperglycaemia or hypoglycaemia in convulsions but have failed to correlate the two. It would appear that from this failure to recognise the biphasic nature of the change, the importance of hypoglycaemia as a factor in the production of convulsions in childhood has come to be over-emphasised. That cases of true hyper-insulinism do very occasionally occur in children is not disputed but the results of this study lead one to conclude that hypoglycaemia plays no part in the causation of the convulsions of infancy and childhood. It would appear therefore that the disturbance in carbohydrate metabolism is the result and not the cause of the convulsions.

Evidence has been brought forward to show that the low blood-sugar value which may exist many hours after a convulsion

is not due to a lack of liver glycogen and that the injection of adrenaline may speedily raise the figure to normal or higher levels. The theory is therefore advanced that the behaviour of the blood-sugar after a convulsion or other such stimulus is due to a sudden discharge of adrenaline into the blood-stream, excited by widespread stimulation of the central nervous system and followed by a temporary state of exhaustion of the adrenal glands. Rabinowitch and Barden[105] have stressed the fact that one of the functions of adrenaline is to render sugar available for body use, and that where the secretion is diminished or entirely lacking the blood-sugar is correspondingly reduced. The duration of the period of hypoglycaemia is very variable but frequently the low blood-sugar value persists for many hours in spite of the ingestion of carbohydrate, an indication of the over-action of endogenous insulin. That this sequence of events occurred in surgical shock and burning was some years ago suggested by two groups of workers[92, 102] quoted above.

Cori[106] has emphasised the fact that apart from its immediate effect on the liver, adrenaline has a constant action on muscle tissue, converting the glycogen into lactic acid, which is later built up by the liver into a form of glycogen readily available for the body's needs. It is well established[76] that muscle glycogen cannot be converted into glucose without the intermediate formation of liver glycogen from lactic acid. Thus, a rise in the lactic acid content of the blood is a constant effect of adrenaline, and since we have postulated a sudden in-

crease in the amount of circulating adrenaline in certain of these cases of convulsions, it should have been possible to demonstrate this rise. The blood lactic acid was accordingly estimated in five cases using the technique of Laszlo and Dische.(107)

The proteins of the blood are precipitated by sulphuric acid and sodium metaphosphate. The filtrate is cleared of carbohydrates by precipitation with copper and lime. The lactic acid is converted by heating with concentrated sulphuric acid into acetaldehyde and carbon monoxide. Hydroquinone is added and the colour formed by its reaction with acetaldehyde measures the amount of the latter present.

The normal concentration of lactic acid in the blood is about 20 mgm. per cent.

TABLE XXV.

No.	Name	Age in years	Blood-Sugar. mgm.%	Blood Lactic Acid. mgm.%	No. of hours after convulsion.	Disease.
1	W.M.	3 8/12	a. 88.5	16.6	2	Idiopathic convulsions.
			b. 149.2	23.8	7	
			c. 42.9	17.3	26	
2	J.D.	2	a. 39.7	24.3	12	Idiopathic convulsions.
			b. 66.2	15.2	17½	
3	D.McR.	3	a. 73.0	53.0	2	Mental deficiency.
			b. 129.8	33.5	8	
4	J.A.	1	a. 54.9	19.5	16	Tonsillitis.
			b. 78.1	17.3	21½	
5	E.D.	6/52	54.3	17.0	12	Tetany.

It will be seen that in three of the cases there is distinct evidence of a rise in the blood lactic acid. In the

first case the rise occurred slowly, while in the third case the rise was very marked and noticed within two hours of the onset of the convulsion. The results are too few to be really conclusive but it would seem that they supply additional evidence of a sudden stimulation of the adrenal medulla excited by the cerebral disturbance due to convulsions.

Therapeutic Indications.

It has been noted already in this study that during the hypoglycaemic phase which frequently follows a convulsion there are none of the clamant signs of hypoglycaemia. Nevertheless on the day after the convulsion it is common to find the child pale, drowsy and irritable and there can be little doubt that this is directly due to the low blood-sugar. Efforts to raise the blood-sugar to normal are therefore indicated, but recalling the sequence of events which produce the hypoglycaemia, it will be seen that this is attended with peculiar difficulty.

Clearly, glucose by mouth in large and repeated doses is essential. The adrenal exhaustion may be fairly transient and it seems important that the liver should be well stocked with glycogen in anticipation of their recovery of function. Where the low blood-sugar persists for many hours or even days, adrenaline in small and, if necessary, repeated doses is indicated. That one injection of adrenaline may be insufficient is shown by the fact that in two of the children to whom adrenaline was administered, the blood-sugar after a sharp rise fell once more to its former low level.

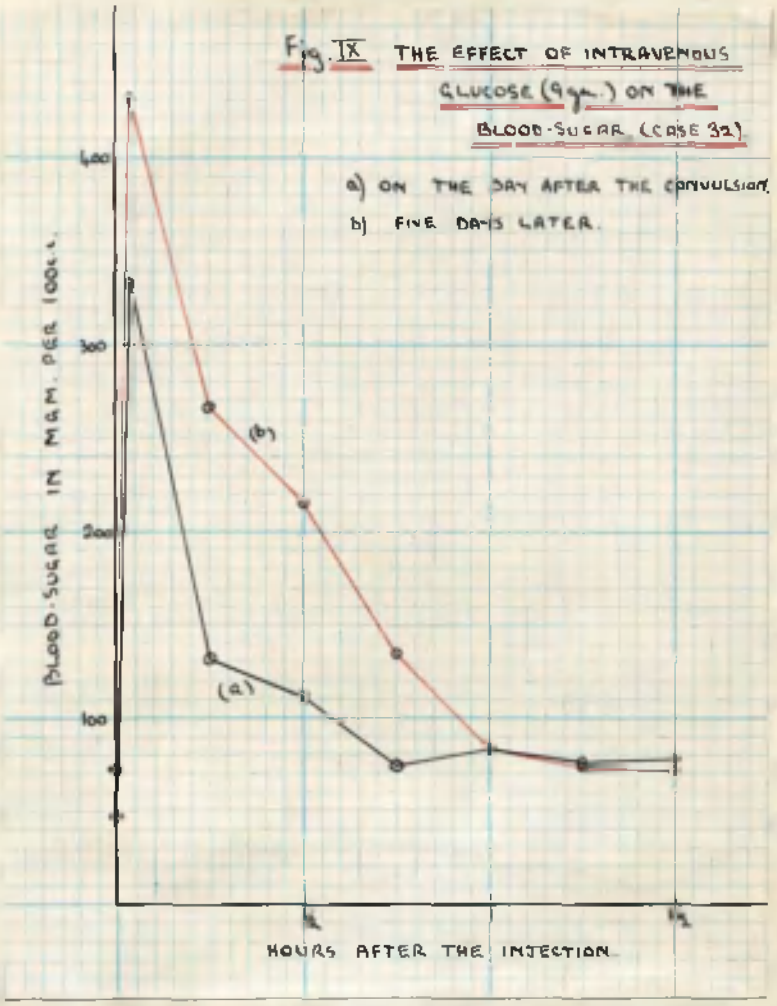
In cases of true spontaneous hyper-insulinism spectacular success attends the administration of glucose intravenously and it was decided to try the effect of this on children suffering from hypoglycaemia following convulsions.

The first child in whom this procedure was tried was A.P. (Case 10), and the behaviour of the blood-sugar has already been detailed at length (p. 49). The child was admitted moribund from gastro-enteritis, having had a convulsion nine hours previously. The blood-sugar on admission was 21.0 mgm. per cent. and one hour later had fallen to 3.7 mgm. per cent. At this point 75 c.c. of a ten per cent. solution of glucose in normal saline were administered intravenously. The blood-sugar rose to 256.4 mgm. per cent. and began to fall rapidly, but the child died within two hours. It is obvious that treatment was undertaken too late in this case to be of any avail.

It will be recalled that Darrow[30] was not impressed by the results of glucose given intravenously to his two cases when they were drowsy and hypoglycaemic following a convulsion. Similarly Josephs[24] and Griffiths[25] in one each of their cases of convulsions associated with hypoglycaemia noted that intravenous glucose had no effect on the drowsiness which followed the convulsion. In two of the cases in this series glucose was given intravenously at a time when the blood-sugar was very low.

Case 32. J.K., a boy of 4 9/12 years, had a convulsion lasting one hour. The actual hyperglycaemic stage was missed but sugar was observed in the urine passed twelve hours later. Sixteen hours after the convulsion when the blood-sugar was 47.1 mgm. per cent. 9 gms. of glucose were given intravenously as a twenty per cent. solution in normal saline.

Before	2'	15'	30'	45'	60'	75'	90'	
47.1	333.3	131.7	111.1	74.1	83.3	76.8	78.1	mgm.% per 100 c.c.



The striking feature of this curve is the extraordinary rapidity with which the blood-sugar fell, thus supporting the contention that the endogenous insulin was undergoing a period of unrestrained activity, the glucose introduced into the blood-stream being converted into liver glycogen within a very short

time. It would appear that towards the end of the curve enough adrenaline was being secreted to prevent the blood-sugar falling again to a low level. This patient was rather drowsy before the glucose was given and though he brightened with the injection the effect was not striking.

Five days later glucose was again given intravenously in the same dose and the same dilution as on the day after the convulsion (Figure 9).

Fasting	2'	15'	30'	45'	60'	75'	90'	
71.4	430.1	266.7	216.2	134.7	83.3	74.1	71.4	mgm.per cent.

It will be seen that the fall in the blood-sugar is much slower, indicating that the conversion of glucose into glycogen is proceeding at a more normal pace. Crawford,(108) working on the subject in this department, has come to the conclusion that in the individual the response to intravenous glucose is strikingly consistent. The unusual response noted while the child was in the hypoglycaemic phase would thus appear to be the result of the disturbance in carbohydrate metabolism produced by the convulsion.

Case 64. J.D., a boy of 2 years, had a convulsion lasting three hours, the blood-sugar being 232.6 mgm. per cent. just after it had stopped. Next morning the blood-sugar had fallen very low till about twelve hours after the convulsion the value was 39.7 mgm. per cent. At this point he was given 6 gms. glucose intravenously in a twenty per cent. solution in normal saline.

Before	2'	15'	30'	45'	60'	75'	90'	
39.7	363.6	138.8	88.5	61.0	32.7	29.8	26.3	mgm.per cent.

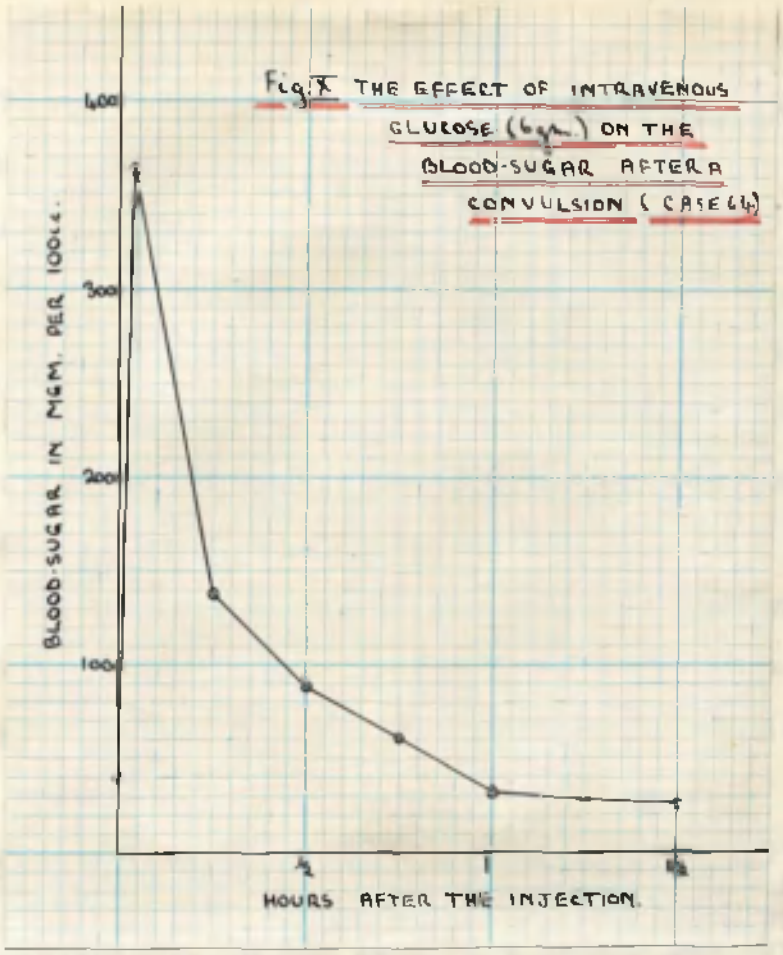


Fig. X THE EFFECT OF INTRAVENOUS GLUCOSE (6 gm.) ON THE BLOOD-SUGAR AFTER A CONVULSION (CASE 44)

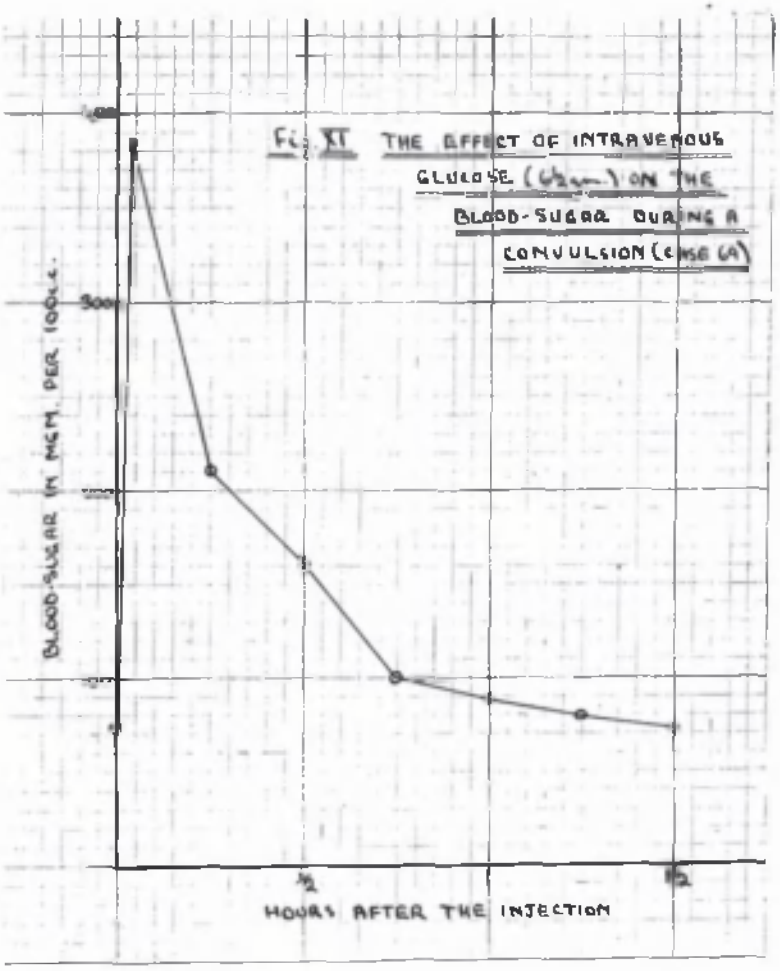
It will be seen that in this case the fall in blood-sugar was even more sudden, implying a rapid absorption of glucose and conversion into glycogen, but in addition the blood-sugar reverted to hypoglycaemic levels one hour after the injection, presumably because of the unopposed action of insulin. The compensatory secretion of adrenaline which is recognised[109] to occur in insulin hypoglycaemia appears in this case to have been lacking. Before the injection the boy

was very drowsy and could only be roused with difficulty. The improvement produced by the glucose was very transient and throughout the day he remained sleepy and dull. At 9.30 a.m. the next morning the blood-sugar was 57.8 mgm. per cent., but a day later it had reached the normal figure of 71.4 mgm. per cent. Unfortunately it was not possible to ascertain the effect of intravenous glucose when the boy had recovered completely from the convulsion, since he had to be dismissed early because of infection in the ward.

Finally glucose was administered intravenously to a child during a generalised convulsion to determine whether it would have any effect in controlling the convulsion.

Case 69, D.McR., a boy of 3 years, had a blood-sugar value of 73.0 mgm. per cent. two hours after the onset of a convulsion. At this point he was given 6½ gms. glucose intravenously in the form of a twenty per cent. solution in normal saline.

Before	2'	15'	30'	45'	60'	75'	90'	
73.0	384.6	210.5	161.5	100.0	88.5	79.4	72.5	mgm. per cent.



It will be seen that the blood-sugar fell in a normal fashion and that there was no tendency for low levels to supervene. The glucose, however, had no effect on the twitching movements which continued for some hours.

It would appear that the administration of glucose intravenously has little to recommend it in the treatment of the hypoglycaemia which may follow a convulsion. Glucose by the mouth in conjunction with small and repeated doses of adrenaline where necessary, constitute the best form of treatment.

FINAL CONCLUSIONS.

(1) Except in cases of true hyperinsulinism, hypoglycaemia plays no part in the causation of the convulsions of infancy and childhood.

(2) In over fifty per cent. of all cases of convulsions in children there is a profound disturbance of the blood-sugar.

(3) The disturbance in blood-sugar is due to a sudden discharge of adrenaline from the adrenal glands, followed by a temporary state of exhaustion of these glands.

(4) A similar sequence of events occurs in traumatic shock from head injuries, fractures and burns.

SUMMARIES OF CASE HISTORIES.

Case 1.

M.G., male, aet. 10 months. Admitted 10.4.36. A full-time healthy child delivered by Caesarean section. Breast-fed till admission and throve well.

Three weeks before admission the child developed a cold and coughed a great deal. He became cross and restless, and this was attributed to teething. He was taken to the out-patient dispensary, and while there took a generalised convulsion which persisted for one-and-a-half hours.

On admission he was found to be a well-nourished child in a convulsion and quite unconscious. T. 100°F., P. 160, R. 64. The twitching affected mainly the right side of the body. There was marked nuchal rigidity and a positive Kernig's sign. The fontanelle was tense. Examination of the chest revealed a rather defective respiratory murmur over the right base and occasional rhonchi at the left base. The spleen was just palpable. The knee-jerks could not be elicited but the other reflexes were present and moderately active. Lumbar puncture was performed under local anaesthesia and about 30 c.c. of slightly turbid cerebro-spinal fluid removed. This contained 330 cells per c.mm., of which 68 per cent. were lymphocytes and 32 per cent. polymorphonuclears.

Blood-Sugar:-

←-----10/4/36----------→<11/4/36>

Time	1 p.m.	2 p.m.	4.30 p.m.	5.30 p.m.	8 p.m.	12 midnight	10.30 a.m.
Blood-Sugar. mgm.%	250.0	144.9	90.1	81.9	93.4	98.8	112.3
No.of hours after convulsion.	1	2	4½	5½	8	12	22½

Nothing abnormal was detected in the urine.

The Mantoux tuberculin test gave a strongly positive reaction at 48 hours.

15.4.36. Lumbar puncture repeated. Tubercle bacilli were seen in the pellicle.

17.4.36. Died.

Autopsy revealed a typical tuberculous meningitis.

Case 2.

H.K., a male aet 2 1/12 years. Admitted 14.12.35. A full-time healthy child. Throve well and developed normally.

At 1 2/12 years took measles followed by chicken-pox. Three months before admission took a fit during which he lay rigid and which lasted about two minutes.

On the day before admission was listless. At 10.30 p.m. he took a convulsion, during which there was generalised rigidity but no twitching. This lasted for a few minutes and was followed by a period of what appeared to be natural sleep, but at 12 midnight he took another spasm which continued till admission at 1.20 a.m.

On examination he was found to be a moderately well-nourished boy in a semi-comatose condition. T. 103.2°F., P. 138, R. 40. The pupils were dilated but reacted to light. There was some degree of nuchal rigidity. None of the superficial reflexes were obtained and the plantar responses were doubtful. The tonsils were enlarged but no abnormality was found in the heart, lungs, or abdomen. The urine obtained a few hours after admission contained sugar and a small amount of acetone.

Blood-Sugar:-

←----------14/12/35----------→ ←15/12/35-→

Time.	1.30 a.m.	2 a.m.	2.30 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	12 noon
Blood-Sugar. mgm.%	317.4	322.6	253.2	89.3	106.4	111.1	87.7	69.5
No. of hours after convulsion.	3	3½	4	11	12	13	14	37½

By next day the temperature had fallen and remained normal thereafter. Repeated examination of the urine showed no further glycosuria. The boy made a rapid recovery and a careful examination of the nervous system revealed no abnormality.

27.12.35. He was dismissed well and had had no further convulsions.

Case 3.

E.F., a girl aet. $1^{6}/12$ years. Admitted 25.3.36. A premature child delivered by Caesarean section because of placenta praevia in the mother. Fairly healthy infant. Breast fed for two months then whole milk and did well. The child was developing normally but could not walk at the time of admission.

Six weeks before admission she had chicken-pox and made a good recovery.

She was well till the night before admission when she vomited a little and did not sleep well. At 6 a.m. on 25.3.36 she was found unconscious in bed and from then until her admission to hospital at 9 a.m. had frequent attacks of generalised rigidity each lasting for about five minutes.

On examination she was found to be a small, pale child, unconscious and breathing stertorously. T. 101.8°F., P. 124, R. 40. There was some stiffness of the lower limbs but no nuchal rigidity. The pupils were equal and active, the knee-jerks very easily elicited and the plantar responses extensor. Otherwise the examination was completely negative. She recovered consciousness at noon but remained drowsy all day. Lumbar puncture was performed but the cerebro-spinal fluid was normal in every respect. There was no abnormality in the urine obtained at 10 a.m., beyond a faint trace of acetone.

Blood-Sugar:-

Time.	8.45 a.m.	9.15 a.m.	9.45 a.m.	10.15 a.m.	10.45 a.m.	11.15 a.m.	11.45 a.m.	12.15 p.m.
Blood-Sugar. mgm.%	232.6	158.7	144.9	102.0	92.6	88.5	74.6	108.7
No.of hours after convulsion.	$2\frac{3}{4}$	$3\frac{1}{4}$	$3\frac{3}{4}$	$4\frac{1}{4}$	$4\frac{3}{4}$	$5\frac{1}{4}$	$5\frac{3}{4}$	$6\frac{1}{4}$

Time.	12.45 p.m.	1.15 p.m.	2.15 p.m.	2.45 p.m.	3.15 p.m.	4 p.m.	4.30 p.m.	5 p.m.
Blood-Sugar. mgm.%	113.6	113.6	84.7	71.9	58.1	49.5	62.5	42.8
No.of hours after convulsion.	$6\frac{3}{4}$	$7\frac{1}{4}$	$8\frac{1}{4}$	$8\frac{3}{4}$	$9\frac{1}{4}$	10	$10\frac{1}{2}$	11

Case 3 (contd.).

Serum Calcium 10.6 mgm. per cent.
Serum Phosphorus 3.3 mgm. per cent.
Plasma Phosphatase 13.9 units.

At 5 p.m. when the blood-sugar was at 42.8 mgm. per cent. 0.25 c.c. 1/1,000 adrenaline was injected subcutaneously with the following result:-

Time.	5 p.m.	5.25 p.m.	5.40 p.m.	6.10 p.m.	6.40 p.m.	7.10 p.m.	7.40 p.m.	8.10 p.m.
Blood-Sugar. mgm.%	42.8	47.6	59.1	61.8	68.5	81.3	85.4	51.5

The child was given nothing to eat or drink between 5 p.m. and 8.10 p.m.

26.3.36. Further blood-sugar readings were as follows:-

	←----------26/3/36----------→					<27/3/36>
Time.	2 a.m.	9.15 a.m.	10.15 a.m.	11.15 a.m.	12.15 p.m.	9.15 a.m.
Blood-Sugar. mgm.%	52.1	65.8	59.1	57.8	73.5	89.3

The patient was still drowsy during the day. The urine contained acetone but no other abnormality.

27.3.36. The child was quite bright and alert and the temperature had settled.

31.3.36. Dismissed well. There have been no further convulsions.

Case 4.

W. McK., male aet. 19/52. Admitted 3.2.36. Full-time child slightly cyanosed at birth. Artificially fed and throve well till 8 p.m. on the night of admission when he took a generalised convulsion lasting about five minutes. Two further similar convulsions occurred before admission at 11.30 p.m.

On examination he was found to be a small, poorly-nourished child acutely ill. T. 101°F., P. 120, R. 60. The fontanelle was bulging and there was nuchal rigidity. The tongue was furred and the throat red but nothing abnormal was made out elsewhere. The urine contained a trace of albumin. Lumbar puncture was performed and 20 c.c. of clear cerebro-spinal fluid were withdrawn but it was found to be quite normal.

Blood-Sugar:-

Time.	12.30 a.m.	1.30 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	3 p.m.
Blood-Sugar. mgm.%	158.7	105.2	105.2	80.6	96.1	114.9	121.9
No.of hours after convulsion.	4½	5½	13½	14½	15½	16½	19

6.2.36. The child developed a haemorrhagic nasal discharge from which B. diphtheriae was cultured.

About 11 p.m. the child's condition became suddenly worse.

7.2.36. Died. At autopsy a patch of broncho-pneumonia was observed in the right upper lobe. There was no evidence of membrane formation in the trachea or oesophagus, larynx or pharynx. No lesion was noted in the suprarenals.

Case 5.

B.T., a girl aet. 1½ days. Admitted 13.12.35. A full-time baby, easy breech delivery. Cyanosed at birth and breathing was not properly established for about three hours.

Thirty hours after delivery she began to have little twitching movements of the limbs and face and her eyes rolled. These attacks lasted for two or three minutes and recurred about every ten minutes.

On examination she was found to be a healthy, well-nourished baby. T. 99.4°F., P. 90, R. 48. There was slight icterus of the skin. Over the left temporal bone there was a soft fluctuant swelling. There was an Erb's paralysis of the left arm and a drop wrist. Nothing abnormal was detected in the heart, lungs or abdomen. The urine contained albumin and a trace of sugar.

Blood-Sugar:-

	←----------13/12/35----------→								←-14/12/35-→	
	9.45 a.m.	10.45 a.m.	11.45 a.m.	12.45 p.m.	1.45 p.m.	2.45 p.m.	3.45 p.m.	5.30 p.m.	1 a.m.	9.15 a.m.
Blood-Sugar. mgm.%	39.8	45.8	55.8	60.6	62.2	54.6	48.7	49.7	67.1	90.0
No. of hours after convulsion.	12	13	14	15	16	17	18	19¾	27¼	35½

14.12.35. The twitching had stopped and the baby seemed quite well. She was discharged home to be breast-fed.

Case 6.

T.B., a boy aet. 10/12 year. Admitted 24.4.36. A full-time healthy baby who throve well till three weeks before admission when a swelling appeared on the left side of the neck. A few days before admission he began to vomit his feeds and had a slight cough. He became constipated and drowsy.

On admission he was found to be a well-nourished child acutely ill and breathing stertorously. T. 105.4°F., P. 160, R. 60. There was a swelling in the left submaxillary region. The throat was very red and the tonsils enlarged and red, especially on the left side. There were some rhonchi on both sides of the chest. The liver was slightly enlarged and the left kidney was palpable. Nothing abnormal was detected in the nervous system and the cerebro-spinal fluid was clear.

26.4.36. The child had a convulsion at 12 midnight.

Blood-Sugar:-

←------27/4/36------------------→

Time.	1 a.m.	10 a.m.	12 noon.	3 p.m.
Blood-Sugar. mgm.%	149.7	88.5	81.3	136.9
No. of hours after convulsion.	1	10	12	15

The urine was not obtained.

27.4.36. The child was running high fever and was very drowsy. The swelling in the left side of the neck was larger and very soft.

28.4.36. An area of dulness could be made out at the left base where the respiratory murmur was tubular. There was much râle at both bases.

29.4.36. Died at 6.20 a.m.

At autopsy there was found extensive broncho-pneumonia particularly marked at the left base.

Case 7.

A.M., a boy aet. $5^{8}/12$ years. Admitted 21.10.35. A normal full-time healthy child who was breast-fed for nine months and developed normally. He had whooping-cough at two years and measles at four years. Since the former illness he had always been pale and of a faintly yellow colour. He was occasionally listless but otherwise well.

On examination he was found to be a well-nourished boy with marked pallor of the skin and mucous membranes. There were no purpuric spots or haemorrhages but the spleen was just palpable. Examination of the blood showed that he had a severe microcytic, hypochromic anaemia and treatment with ferrous sulphate was immediately begun. There was a rapid response and the child's general condition was improving markedly.

28.11.35. At 1 a.m. the boy took a left-sided convulsion which lasted for half-an-hour. This was followed by a deep coma during which none of the superficial or deep reflexes could be elicited. T. 99.4°F., P. 150, R. 30. By 10 a.m. the boy seemed quite well and nothing could be found in a careful examination of the nervous or other systems to account for the fit.

Blood-Sugar:-

Time.	1.30 a.m.	2 a.m.	9.50 a.m.
Blood-Sugar. mgm.%	277.7	238.1	152.6
No. of hours after convulsion.	½	1	8 hrs.50'

The urine at 9 a.m. contained sugar but no acetone.

The boy made an uninterrupted recovery.

7.12.35. Dismissed well.

There have been no further convulsions reported.

Case 8.

M.McL., a girl aet. $4^{8}/12$ years. Admitted 12.12.35. Full-time healthy child. Throve well and developed normally. Said to have had bronchitis at one year and again at $2^{8}/12$ years. Measles at two years.

Two weeks before admission seemed listless and had a hard cough. One week later she became very drowsy and complained of headache. The drowsiness progressed steadily but occasionally she had bouts of screaming. On admission she had a convulsion lasting about one hour.

On examination she was found to be a well-developed girl comatose and very ill. T. 100°F., P. 142, R. 24. Nuchal rigidity and Kernig's sign were very marked. The pupils were dilated and inactive and no superficial or deep reflexes could be elicited. The heart, lungs and abdomen presented no abnormality. The urine contained a trace of albumin, a moderate amount of sugar and a trace of acetone. Microscopically there were a few pus cells. Lumbar puncture revealed a clear cerebro-spinal fluid under considerably increased pressure, the Pandy test positive and the cell count 371 per c.mm. The cellular increase was almost entirely lymphocytic. Tubercle bacilli were later found in the pellicle which formed in the fluid on standing.

An X-ray of the chest showed enlarged hilum shadows.

Blood-Sugar:-

	←13.12.35→												←14.12.35→	
Time.	12.15 a.m.	12.45 a.m.	1.15 a.m.	1.45 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	1.30 p.m.	2.30 p.m.	3.30 p.m.	5.30 p.m.	1.15 a.m.	9.30 a.m.
Blood-Sugar. mgm.%	303.0	270.0	476.1	312.5	91.8	96.1	104.1	112.3	104.1	88.5	75.2	85.4	144.9	91.8
No.of hours after convulsion.	1	$1\frac{1}{2}$	2	$2\frac{1}{2}$	$10\frac{1}{4}$	$11\frac{1}{4}$	$12\frac{1}{4}$	$13\frac{1}{4}$	$14\frac{1}{4}$	$15\frac{1}{4}$	$16\frac{1}{4}$	$18\frac{1}{4}$	26	$34\frac{1}{4}$

The child's general condition deteriorated gradually.

19.12.35. Died.

At autopsy there was found a small tuberculoma in the right lobe of the cerebellum and a thick tuberculous exudate over the base of the brain. A primary lung focus was found in the right upper lobe. The suprarenals appeared normal.

Case 9.

G.W., a boy aet. 1 1/12 years. Admitted 14.2.36. A healthy full-time baby. Breast-fed for eight months and throve well. He cut his first tooth at 9/12 year and began to walk at one year. He was never given cod-liver oil.

The child was well till three weeks before admission when he began to have attacks of dyspnoea and crowing which were worse at nights. At 9 a.m. on the day of admission he took a short generalised convulsion which recurred at 12.30 p.m. and again at 2.30 p.m.

On examination he was found to be a rather fat well-nourished child, cross and vigorous. The radial epiphyses were enlarged and there was a fairly well-marked rachitic rosary. It was not possible to elicit Chvostek's or Trousseau's signs. Examination of the heart, lungs, abdomen and nervous system revealed no abnormality. The urine contained acetone but was otherwise normal.

Blood-Sugar:-

Time.	5 p.m.	6 p.m.	10 a.m.	11.30 a.m.	12 noon	12.30 p.m.	1 p.m.
	←------14/2/36→		←----------15/2/36----------→				
Blood-Sugar. mgm.%	172.3	172.3	71.9	78.1	75.2	55.5	49.7
No.of hours after convulsion.	8	9	25	26½	27	27½	28

15.2.36. X-ray of wrist "old rickets healed".

Serum Calcium 10.7 mgm. per cent.
Serum Phosphorus 4.5 mgm. per cent.
Plasma Phosphatase 14.0 units.

The convulsions were thought to be due to tetany but it was not till a few days later that this was proved.

20.11.36. Chvostek's sign very marked. The child was heart to crow distinctly.

Serum Calcium 8.9 mgm. per cent.
He made a good recovery with chloral and adexolin.

6.3.36. Dismissed well after a stay in the country branch.

Case 10.

A.P., a boy aet. 18/52 year. Admitted 9.3.36. A full-time healthy baby. Breast-fed for six weeks, then whole milk and sugar and throve well.

Two days before admission the child became very cross and had some flatulence. At 6.30 a.m. on the day of admission he had a 'turn' during which he became stiff and rolled his eyes. His legs were pulled up and his breathing gasping. During the day he had three stools, the first being normal and the others pale, loose and slimy. Shortly after admission he had another convulsion.

On examination he was found to be a poorly-nourished child, cyanosed, dehydrated and semi-comatose. The eyes were sunken and the fontanelle depressed. The pupils were equal and reacted sluggishly to light. The reflexes with the exception of the abdominals were present. Nothing abnormal was detected in heart, lungs or abdomen. The urine was not obtained.

Blood-Sugar:-

Time.	3.30 p.m.	4.30 p.m.
Blood-Sugar. mgm.%	21.0	3.7
No. of hours after convulsion.	9	10

At 5.45 p.m. 75 c.c. of 10 per cent. glucose in normal saline were introduced into the longitudinal sinus but though the blood-sugar rose the convulsion continued and the child died at 7.30 p.m.

Blood-Sugar:-

Time.	4.30 p.m.	6 p.m.	6.30 p.m.	7 p.m.	7.30 p.m.
Blood-Sugar.	3.7	256.4	208.4	163.9	84.0*

* Blood taken from the heart immediately post-mortem.

At autopsy the child was found to have a slight acute gastro-enteritis and a terminal broncho-pneumonia. The liver and kidneys showed evidence of cloudy swelling.

Case 11.

D.R., a boy aet. $8^{8}/12$ years. Admitted 18.10.35. At the age of three weeks he developed pyloric stenosis which was cured by Rammstedt's operation. He throve well after this and appeared to develop normally, but at two months he began to take fits. These became progressively worse during the years and affected always the right side. On admission he had a convulsion lasting fifteen minutes.

On examination he was found to be a well-nourished boy, quite conscious but unable to articulate properly. T. 99.4°F., P. 144, R. 30. The pupils were equal and active, the abdominal reflexes were not elicited and the knee-jerks were active. There was definite weakness of the right hand and the child walked with the right leg dragging slightly. The right plantar response was extensor and the left flexor. The lungs and abdomen showed no abnormality but there was a systolic murmur at the base of the heart.

The urine contained no abnormal constituents.

Blood-Sugar:-

←----------18/10/35----------→ <19/10/35>

Time.	12.20 p.m.	12.50 p.m.	1.20 p.m.	1.50 p.m.	2.20 p.m.	5 p.m.	12 mid-night	10 a.m.
Blood-Sugar. mgm.%	129.8	147.6	156.2	137.0	116.2	90.9	71.2	82.2
No. of hours after convulsion.	2	$2\frac{1}{2}$	3	$3\frac{1}{2}$	4	6.40'	13.40'	23.40'

The fundi were normal and a skiagram of the skull showed nothing unusual.

25.10.35. The blood-sugar readings were repeated at the same times and on the same diet. The child was now very well and having no convulsions.

Time.	12.20 p.m.	12.50 p.m.	1.50 p.m.	2.20 p.m.	5 p.m.	12 mid-night	10 a.m.
Blood-Sugar. mgm.%	90.9	81.3	76.3	77.5	75.7	84.7	86.2

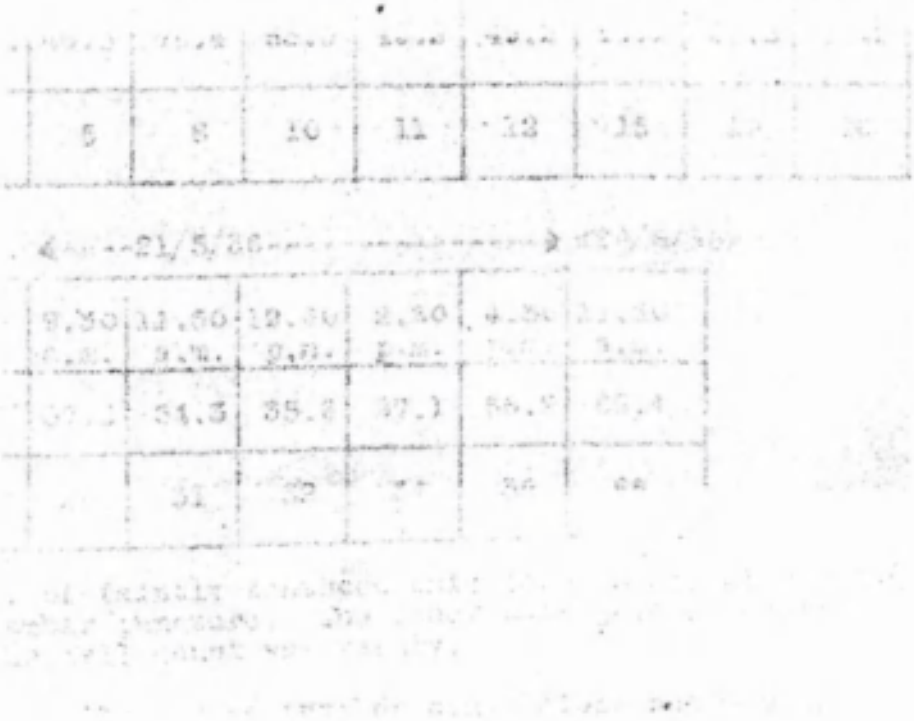
Case 11 (contd.)

The child was seen by a neurological surgeon who decided to operate.

4.11.35. At operation a small area of the cerebral cortex in the region of the centre for the right hand, was found to be thickened. This area was ablated with the electric cautery.

The boy made a good recovery from the operation and had no more fits.

Six months later he reported as an out-patient, having had only five fits in that time.



Case 12.

M.C., a girl aet. 12 days. Admitted 20.5.36. A full-time baby delivered by instruments and required resuscitation. Breast-fed for ten days then put on whole milk, water and sugar.

The baby seemed quite well till 4.30 a.m. on the day of admission when she took a series of short fits during which the limbs twitched, the eyes rolled and the breathing was heavy.

On examination the patient was found to be a small under-nourished baby, cyanosed and ill. T. 103°F., P. 138, R. 40. There was no nuchal rigidity and the fontanelle was flat. The pupils were equal and active and all reflexes physiological. Nothing abnormal was detected in the examination of heart, lungs or abdomen. The urine contained no abnormal constituent.

Blood-Sugar:-

←----------20/5/36----------→

Time.	9.30 a.m.	12.30 p.m.	2.30 p.m.	3.30 p.m.	4.30 p.m.	5.30 p.m.	7.30 p.m.	8.30 p.m.	9.30 p.m.
Blood-Sugar. mgm.%	88.5	70.4	63.3	49.2	49.2	49.2	47.8	38.2	24.8
No.of hours after convulsion.	5	8	10	11	12	13	15	16	17

←----21/5/36----------→ <22/5/36>

Time.	9.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	4.30 p.m.	11.30 a.m.
Blood-Sugar. mgm.%	57.8	54.3	35.6	27.1	56.2	69.4
No.of hours after convulsion.	29	31	32	34	36	55

About 14 c.c. of faintly xanthochromic cerebro-spinal fluid were removed by lumbar puncture. The Pandy test gave a positive reaction and the cell count was twenty.

23.5.36. The child had no further convulsions but remained rather limp.

Dismissed much improved.

Case 13.

C.McA., a girl aet. 9/52 year. Admitted 24.4.36. A premature child, healthy at birth, and appeared to thrive at first.

At about five weeks she was noticed to be pale but seemed healthy and active. There were no haemorrhages.

On examination she was found to be a moderately well-nourished child showing extreme pallor of the skin and mucous surfaces. There was a slight icteric tinge of the skin, but no conjunctival icterus. There was no purpura. Examination of the heart, lungs and nervous system revealed no abnormality. The liver and spleen were both palpably enlarged. Nothing abnormal was detected in the urine.

Blood Examination:-

Haemoglobin 19 per cent.
R.B.C. 1,070,000 per c.mm.
C.I. 0.90.
Leucocytes 6,600 per c.mm.

25.4.36. Blood transfusion 60 c.c.

27.4.36. Blood transfusion 45 c.c.

28.4.36. Blood transfusion 30 c.c.

29.4.36. Dismissed home.

The child was seen several times as an out-patient and the condition of the blood was much improved.

8.5.36. R.B.C. 3,300,000 per c.mm.
W.B.C. 7,100 per c.mm.
Hb. 51 per cent.
C.I. 0.77.

26.5.36. The child was not thriving and was readmitted. She began to vomit and the stools were loose and undigested.

2.6.36. The child took a convulsion at 11.20 a.m. This persisted for two hours and affected the left side. The general condition rapidly became worse and she died at 10 p.m. Urine obtained at 1.30 p.m. contained abundant sugar.

Blood-Sugar:-

Case 13 (contd.).

Time.	11.40 a.m.	12.10 p.m.	12.40 p.m.	1.10 p.m.	1.40 p.m.	2.40 p.m.	3.40 p.m.	4.40 p.m.
Blood-Sugar. mgm.%	204.1	178.6	156.2	162.6	196.1	204.1	196.1	147.1
No.of hours after convulsion.	20'	50'	1 hr. 20'	1 hr. 50'	2 hrs. 20'	3 hrs 20'	4 hrs. 20'	5 hrs. 20'

Time.	5.40 p.m.	7.40 p.m.	8.40 p.m.	9.40 p.m.
Blood-Sugar. mgm.%	128.2	106.4	72.9	135.1
No.of hours after convulsion.	6 hrs 20'	8 hrs 20'	9 hrs. 20'	10 hrs. 20'

At autopsy the organs showed a uniform pallor. The liver gave a very marked iron reaction and in the kidneys and spleen this was less marked. A slight pachymeningitis interna haemorrhagica was present over the vertex of the skull but there was no recent effusion of blood. There was an early longitudinal sinus thrombosis.

Case 14.

D.F., a boy aet. $2^{8}/12$ years. Admitted 25.7.36. A full-time healthy infant who throve well and developed normally.

The child had occasional bouts of diarrhoea in the three months before admission.

Five weeks before admission he fell from a lorry and sustained a deep wound in the left frontal region. He recovered from this but two weeks later he began to vomit a little and the diarrhoea recurred. One week before admission he had a convulsion and after this he became listless and quiet. At 10.45 p.m. on the night of admission he had a convulsion involving the right side of the body and this lasted for two hours.

On examination he was found to be a well-nourished child, still having twitching movements of the right side of the face, right arm and leg. T. 101.8°F., P. 140, R. 40. The pupils were dilated and fixed and there was well-marked nuchal rigidity. The knee-jerks were active but the abdominal and cremasteric reflexes were elicited with difficulty. There was no abnormality in the heart, lungs or abdomen. The cerebro-spinal fluid obtained by lumbar puncture was found to contain 300 cells per c.mm., of which 80 per cent. were lymphocytes. The Pandy test was positive and tubercle bacilli were later found in the pellicle. The urine contained nothing abnormal.

Blood-Sugar:-

←----------26/7/36----------→ <27/7/36>

Time.	1 a.m.	2 a.m.	3 a.m.	4 a.m.	11.30 a.m.	1 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	125.0	102.0	89.3	86.9	85.4	93.4	135.1
No.of hours after convulsion.	$2\frac{1}{4}$	$3\frac{1}{4}$	$4\frac{1}{4}$	$5\frac{1}{4}$	$12\frac{3}{4}$	$14\frac{1}{4}$	$34\frac{3}{4}$

27.7.36. X-ray of chest showed areas of consolidation in both lungs.

31.7.36. Died. At autopsy there was found tuberculous meningitis and cervical gland tuberculosis.

Case 15.

A.G., a boy aet. 3/365 year. Admitted 10/2/36. The mother was in labour for three days owing to some mal-presentation. The child was finally delivered by instruments but was cyanosed at birth and required resuscitation. Soon after birth he began to take generalised convulsions and these continued till admission.

On examination the infant was found to be well-developed but cyanosed and gasping. T. 98.4°F., P. 108, R. 48. He was having slow twitching movements of the arms and legs. There was a large caput over the posterior half of the head and forceps marks in the left temporal region. The air-entry in the right lung was very poor but seemed normal in the left lung. The heart sounds were of fairly good quality.

Blood-Sugar:-

←----10/2/36--→ ←----------11/2/36----------→ <12/2/36>

Time.	10 p.m.	11 p.m.	12 mid-night	11 a.m.	12 noon	2 p.m.	3 p.m.	4 p.m.	5 p.m.	10 p.m.	7 a.m.
Blood-Sugar. mgm.%	42.0	44.4	50.2	77.5	82.6	129.8	91.7	97.1	97.1	101.0	85.4

The blood-sugar estimations were not begun till about two days after the onset of the convulsions so that it seems unnecessary to tabulate the actual numbers of hours.

The urine was not obtained.

12.2.36. The child died at 7 a.m. At autopsy there was found a large sub-dural haemorrhage with partial destruction of the posterior part of the brain.

Case 16.

D.McL., a boy aet. $5^{9}/12$ years. Admitted 18.12.35. A full-time healthy child. Breast-fed for one month then whole milk and Sister Laura's Food. Cut his first tooth at six months but has never been able to walk or talk properly.

Whooping-cough at $^{6}/12$ year and measles at $1^{5}/12$ years. At $1^{6}/12$ the child developed pain and stiffness of the left arm and was admitted to this hospital, where he was found to have a tuberculous elbow joint. A few weeks later he developed a tuberculous cervical adenitis and after surgical treatment of both these conditions he was removed to a sanatorium where he spent $2^{7}/12$ years and apparently did well.

Six months before admission the child had been discharged from the sanatorium and had remained well till in the last two weeks he developed a cold with rhinitis and cough. At 4.30 p.m. on the day of admission he was found in a generalised convulsion which lasted for four hours.

On admission he was found to be a much emaciated child giving the appearance of great neglect and still having twitching movements of all the limbs. T. 101.8°F., P. 150, R. 48. There were numerous pustular sores all over his body and some oedema of both feet. Examination of the heart, lungs and abdomen revealed no abnormality. When the effects of the convulsion had passed off, the central nervous system appeared normal. The urine contained albumin, a moderate amount of acetone and abundant pus. The cerebro-spinal fluid was normal in all respects.

Blood-Sugar:-

←----18/2/35--------→ ←----------19/12/35 ----------→ ←20/12/35

Time	7.30 a.m.	8.30 a.m.	9.30 a.m.	10.30 a.m.	10 a.m.	11 a.m.	12 noon	1 p.m.	3 p.m.	4 p.m.	5 p.m.	10 a.m.
Blood-Sugar. mgm.%	188.7	50.0	44.0	57.4	63.8	66.6	54.3	46.9	77.5	52.6	44.6	47.6

The child remained drowsy for several days and thereafter slowly improved. The pyuria persisted and was thought to be tuberculous with a superimposed pyogenic infection. The tuberculin skin tests were strongly positive. The child was dismissed after a stay of a month in hospital, the general condition being much improved.

23.2.36. Five weeks later he was readmitted in a similar convulsion. The history was that on the day before admission he vomited

Case 16 (contd.).

once and became dull and listless. At 6 p.m. on the day of admission he suddenly took a generalised convulsion which lasted for three hours.

The general condition on examination was very similar to that of the first admission. T. 105°F., P. 160, R. 28. The urine contained abundant pus.

Blood-Sugar:-

Time.	12 noon	1 p.m.	3 p.m.	4 p.m.
Blood-Sugar. mgm.%	36.5	46.3	47.6	55.5
No.of hours after convulsion.	18	19	21	22

The day after the fit when the blood-sugar readings were made the boy was drowsy and disinclined to speak.

At 4 p.m. 0.5 c.c. 1/1,000 adrenaline was injected hypodermically with the following results:-

Time.	4.5 p.m.	4.15 p.m.	4.45 p.m.	5.45 p.m.	7.45 p.m.	10.30 p.m.
Blood-Sugar. mgm.%	57.7	70.9	82.6	169.5	112.3	88.5
Time after injection.	5'	15'	45'	1 hr. 45'	3 hrs. 45'	6 hrs. 30'

Next day the blood-sugar was still low but rose during the afternoon.

Time.	9.30 a.m.	11.30 a.m.	2.30 p.m.
Blood-Sugar. mgm.%	45.4	44.4	128.2

Case 16 (contd.).

6.3.36. A glucose tolerance curve was performed. The boy was given 11 gm. glucose in 110 c.c. of water.

	Fasting	½ hour	1 hour	1½ hours	2 hours
Blood-Sugar. mgm.%	59.1	69.9	80.0	69.1	77.5

A skiagram of the skull showed no abnormality.

An intravenous pyelogram showed both ureters and particularly the left to be tortuous and dilated. A laparotomy was performed and the left kidney found to be the seat of advanced tuberculous disease, while the bladder showed a congenital abnormality in the form of a diverticulum which completely obliterated the recto-vesical pouch of Douglas.

The boy was dismissed improved and has since been fairly well.

Case 17.

J.McL., a boy aet. 11/52 year. Admitted 20/3/36. A premature child healthy at birth. Fed on whole milk and sugar in inadequate amount.

The child was, however, well till six hours before admission when he began to have short generalised convulsions. Between the fits, of which the infant had nine, he appeared to be unconscious.

On examination he was found to be a small undernourished child, cross but not acutely ill. T. 97°F., P. 130, R. 36. There was craniotabes on the right side of the occiput but no frontal bossing. Neither Chvostek's nor Trousseau's signs could be elicited. The pupils were equal and active and the reflexes physiological. Nothing abnormal could be detected in the heart, lungs or abdomen. The urine contained only a trace of albumin.

Serum Calcium 6.7 mgm. per cent.
Serum Phosphorus 4.0 mgm. per cent.
Plasma Phosphatase 13.2 units.

Blood-Sugar:-

Time.	10 a.m.	11 a.m.	12 noon	2 p.m.	3 p.m.
Blood-Sugar. mgm.%	126.5	90.9	99.5	119.0	135.1
No. of hours after convulsion.	13	14	15	17	18

Calcium chloride grs. 10 t.i.d. and adexolin m. 5 t.i.d. were administered but the convulsions continued.

21.3.36. 10 c.c. calcium gluconate intravenously at 1 p.m.

Case 17 (contd.).

←----------21/3/36----------→ 22/3/36

Time.	12.15 p.m.	12.45 p.m.	1.15 p.m.	2.15 p.m.	2.45 p.m.	3.15 p.m.	3.45 p.m.	4.15 p.m.	11 a.m.
Blood-Sugar. mgm.%	89.3	62.1	81.3	156.2	204.1	149.2	106.4	66.6	79.4

Calcium gluconate was given intravenously for the next five days and this finally controlled the convulsions.

24.3.36. Serum Calcium 8.0 mgm. per cent.

28.3.36. Serum Calcium 10.9 mgm. per cent.

Dismissed well.

Case 18.

J.H., a boy aet. 5/52 year. Admitted 3.1.36. A full-time healthy baby who was breast-fed for three weeks and then put on milk and water.

Fifteen hours before admission the child took a convulsion affecting the left side of the body. Thereafter he had a long series of convulsions recurring about every hour and lasting a few minutes.

On examination the child was found to be rather small but quite well nourished and quite normal between the attacks. T. 98°F., P. 134, R. 44. The head was rather small for his age. Nothing abnormal was detected in examination of the heart, lungs, abdomen or nervous system. The urine contained a trace of albumin.

Blood-Sugar

←----------3/1/36----------→ ←4/1/36→

Time	11.30 a.m.	12 noon	12.30 p.m.	1 p.m.	2.30 p.m.	3 p.m.	3.30 p.m.	4 p.m.	4.30 p.m.	5 p.m.	5.30 p.m.	9.30 a.m.	11.30 a.m.
Blood-Sugar mgm.%	81.3	56.2	94.3	77.5	79.7	73.0	69.4	46.9	46.9	43.1	40.9	23.6	73.3
No. of hours after convulsion.	21	21½	22	22½	24	24½	25	25½	26	26½	27	43	45

The convulsions were gradually controlled by means of chloral. Lumbar puncture revealed a normal cerebro-spinal fluid.

5.1.36. The child developed severe gastro-enteritis which persisted for about ten days.

20.1.36. The stools were now much better but the baby looked dehydrated and ill.

21.1.36. Died 1.30 a.m.

At autopsy there was found patchy, irregular congestion in the small and large intestine. There was slight broncho-pneumonia. The suprarenals appeared to be normal.

Case 19.

E.G., a girl aet. $1^{11}/12$ years. Admitted 10.7.35. A full-time healthy child.

She was well till three days before admission when she became irritable and constipated. At 3 p.m. on the day of admission she became suddenly unconscious, fell to the left side and when picked up was rigid with staring eyes and frothing lips. Shortly after this she vomited.

On admission two hours after the onset of the convulsion she was found to be a moderately well-nourished child, deeply cyanosed and unconscious. T. 103°F., P. 132, R. 28. The right arm and leg were rigid and showed occasional twitching. The pupils were equal but dilated and fixed. The knee-jerks were active but the abdominal and plantar reflexes could not be elicited. There was no facial phenomenon. Nothing abnormal was detected in examination of the heart, lungs or abdomen. The urine contained neither albumin nor sugar. Lumbar puncture revealed a clear cerebro-spinal fluid under increased pressure but with no increase in the cell count or the protein content.

Blood-Sugar:-

	←----------10/7/35----------→						←---11/7/35------→		
Time.	5 p.m.	6 p.m.	7 p.m.	8 p.m.	9 p.m.	11 p.m.	12 mid-night	9 a.m.	12 noon	5 p.m.
Blood-Sugar. mgm.%	250.0	204.1	125.0	71.2	56.2	52.3	52.0	42.6	55.5	36.7
No.of hours after convul-sion.	2	3	4	5	6	8	9	18	21	26

The next morning the child appeared quite well though irritable. Acetone was detected in the urine at 1 a.m., i.e. ten hours after the convulsion.

Forty-two hours after the seizure the fasting blood-sugar was normal but the blood-sugar curve after 9 gms. glucose showed rather a high rise and a delayed fall.

Fasting	½ hour.	1 hour.	1½ hours.	2 hours.
82.6	188.6	153.8	126.5	101.0 mgm. per 100 c.c.

Case 19 (contd.).

Nothing abnormal was detected in the subsequent careful examination of the child's nervous system. A skiagram of the skull showed a suggestion of 'paw marking' in the parietal and occipital regions. The sutures did not appear to be separated.

5.8.35. Dismissed well.

8.11.35. Readmitted. The child remained well till four days before admission when she began to have diarrhoea. This continued for three days and was followed by constipation. At 11.30 a.m. on the day of admission she became rigid and this lasted for two hours. At 4 p.m. she began to have twitching movements of the left side of the body.

On admission at 6 p.m. she was unconscious and cyanosed, and had occasional twitching of the left side of the face, left arm and leg. The pupils were equal and reacted sluggishly to light. The plantar responses were doubtful and all other reflexes were present on the right side and absent on the left side. Nothing abnormal was discovered in examination of heart, lungs or abdomen. By 7.30 p.m. the child was regaining consciousness and there was a definite left-sided paresis with absent abdominal reflex and extensor plantar response. The urine contained a trace of sugar and was loaded with acetone.

Blood-Sugar:-

Time.	6.30 p.m.	7.30 p.m.	9.30 p.m.	10.30 p.m.	11.30 p.m.	10.30 a.m.
Blood-Sugar. mgm.%	49.5	52.3	59.5	59.5	53.7	40.9
No. of hours after convulsion.	7	8	10	11	12	23

10.11.35. The spastic paresis was passing off and in the course of one week had quite disappeared. The urine contained no sugar but still a large amount of acetone.

27.11.35. Sugar tolerance curve. 10 gms. glucose in 100 c.c.water.

Fasting.	½ hour.	1 hour.	1½ hours.	2 hours.
84.7	94.3	99.0	86.2	77.5 mgm.per 100 c.c.

6.12.35. Blood-sugar curve following 0.5 c.c. 1/1,000 adrenaline hypodermically:-

Case 19 (contd.).

Fasting	½ hr.	1 hr.	1½ hrs.	2 hrs.	2½ hrs.	3 hrs.
58.8	113.6	172.4	217.3	256.4	147.1	96.1

7.1.36. Dismissed well.

4.2.36. Sugar tolerance curve. 10 gms. glucose in 100 c.c. water.

Fasting	½ hour.	1 hour.	1½ hours.	2 hours.
87.7	125.0	147.0	135.1	70.4 mgm. per 100 c.c.

25.2.36. The child was fatally burned in her own home. At autopsy the suprarenal glands showed no lesion except some vacuolation of the cortical cells which was possibly due to toxaemia associated with the mode of death. The pituitary and pancreas appeared to be healthy and the thyroid gland was of the infantile type showing little colloid in the vesicles.

Case 20.

C.N., a boy aet. $1^{10}/12$ years. Admitted 26.8.36. A normal full-time healthy child. Throve well and developed normally. The only previous illness was measles at $1^{2}/12$ years.

On the morning of the day of admission the boy was drowsy and had repeated loose stools. At 3.20 p.m. he took a generalised convulsion which lasted fifteen minutes and which was followed by a period of unconsciousness.

On examination the patient was found to be a well-nourished child, drowsy and acutely ill. T. 101.8°F., P. 120, R. 26. There was no nuchal rigidity or Kernig's sign. The knee-jerks were not elicited but all other reflexes were active. The pupils were equal and active. The urine contained acetone and a faint haze of albumin.

Blood-Sugar:-

←-----26/8/36------→ ←------27/8/36----------→

Time.	3.50 p.m.	4.50 p.m.	7 p.m.	10 p.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30
Blood-Sugar. mgm.%	109.9	85.4	54.6	44.4	27.0	23.3	22.3	24.6
No.of hours after convulsion.	½ hr.	1½ hrs.	3hrs. 40'	6hrs. 40'	18hrs 10'	19hrs 10'	20 hrs. 10'	21 hrs. 10'

During this period the child was given five ounces of whole milk and one teaspoonful of sugar every four hours. He had one loose motion.

27.8.36. The boy was irritable and drowsy. At 12.45 noon when the blood-sugar was 24.6 mgm. per cent., 0.5 c.c. adrenaline 1/1,000 was injected hypodermically with the following result:-

←----------27/8/36----------→ ←---28/8/36--→

Time.	12.30 p.m.	1 p.m.	1.15 p.m.	1.45 p.m.	2.15 p.m.	2.45 p.m.	3.45 p.m.	4.45 p.m.	2.30 a.m.	9.30 a.m.
Blood-Sugar. mgm.%	24.6	63.5	96.6	128.2	65.3	42.5	34.8	23.6	25.0	32.8

28.8.36. The boy was removed from hospital against advice.

Case 21.

J.W., a girl aet. 2 years. Admitted 28.5.36. A healthy full-time infant. Breast-fed till $1^{6}/12$ years and throve well.

Eight weeks before admission the child took measles and was severely ill. She appeared to recover satisfactorily but one week before admission she developed a slight cough, vomited a great deal and became drowsy. The general condition steadily deteriorated and headache became severe.

On admission she was found to be a rather wasted child, very drowsy and ill. T. 99°F., P. 88, R. 32. Nuchal rigidity was very marked. The pupils were unequal and very sluggish. Nothing abnormal could be detected in the heart, lungs or abdomen. The knee-jerks could not be elicited but all other reflexes were normal. The Mantoux test gave a strongly positive result at 48 hours.

29.5.36. Lumbar puncture under chloroform anaesthesia revealed a clear fluid under greatly increased pressure. The cells were increased to 110 per c.mm. and chiefly lymphocytes. The Pandy test was positive.

3.6.36. The child had a convulsion at 8.45 a.m. and died four hours later.

Blood-Sugar:-

Time.	9.15 a.m.	9.45 a.m.	10.45 a.m.	11.45 a.m.	12.45 p.m.
Blood-Sugar. mgm.%	250.0	250.0	169.5	188.7	185.2*
No.of hours after convulsion.	½	1	2	3	4

* Blood removed from heart immediately after death.

The urine at 11.45 a.m. contained abundant sugar.

Permission for an autopsy was not obtained.

Case 22.

T.M., a boy aet. 3/12 year. Admitted 19.5.36. A healthy full-time child who was breast-fed and throve well. On the day before admission he became cross and irritable and vomited several times. At 6.30 a.m. on the day of admission he had a short right-sided convulsion and this recurred several times during the morning.

On admission he was found to be a well-nourished infant, irritable but not acutely ill. T. 99°F., P. 134, R. 40. The fontanelle was flat and there was no nuchal rigidity. There was no craniotabes or other signs of rickets. Nothing abnormal was detected in the heart, lungs, abdomen or nervous system. The urine contained neither sugar nor acetone.

Blood-Sugar:-

←----------19/5/36----------→ <20/5/36>

Time.	3 p.m.	4 p.m.	5 p.m.	6 p.m.	7 p.m.	8 p.m.	9 p.m.	12 mid-night	9.30 a.m.
Blood-Sugar. mgm.%	90.1	72.4	55.8	68.7	71.9	90.1	78.1	86.5	60.9
No.of hours after convulsion.	8½	9½	10½	11½	12½	13½	14½	17½	27

Lumbar puncture revealed a normal cerebro-spinal fluid. Serum Calcium 8.5 mgm. *per cent.*

20.5.36. The child had a short convulsion at 2.30 p.m. and again at 4.30 p.m.

Blood-Sugar:-

←----------20/5/36----------→ <21/5/36>

Time	2.30 p.m.	3.30 p.m.	4.30 p.m.	5.30 p.m.	7.30 p.m.	8.30 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	56.5	93.4	102.0	84.0	81.3	85.5	61.7
No.of hours after convulsion.	32	33	34	35	37	38	51

Case 22 (contd.).

21.5.36. There were no further convulsions but the child looked pale.

Haemoglobin 65 per cent.
R.B.C. 3,400,000.
C.I. 0.96.

22.5.36. Blood transfusion - 140 c.c. of the father's blood.

23.5.36. Hb. 90 per cent.
R.B.C. 4,700,000.
C.I. 0.95.
W.B.C. 11,000.

29.5.36. Dismissed well.

Case 23.

A.B., aet. $2^{3}/12$ years. Admitted 17.6.36. A full-time healthy child who throve well and developed normally.

At $1^{3}/12$ years had bronchitis which recurred about 4 months before admission and lasted about 4 weeks.

Five days before admission the child became fevered, cross and drowsy and apparently had severe, colicky, abdominal pains. Five hours before admission he had a convulsion which lasted for about two hours.

On admission the child appeared to be acutely ill and very drowsy. Apart from nuchal rigidity and an absent knee-jerk on the left side there was little to be made out in the nervous system. There were some rhonchi on both sides of the chest but no abnormality in the heart. The spleen was palpable. The Mantoux intradermal tuberculin test was very positive at 48 hours.

20.6.36. At 8 p.m. the child had a severe generalised convulsion which continued in spite of chloral till 10.30 p.m. Urine obtained about 10 p.m. by catheterisation contained a moderate amount of sugar and acetone. Lumbar puncture was performed and 25 c.c. of slightly turbid fluid obtained under increased pressure. The Pandy test was positive and the cell count 587 per c.mm., of which sixty-five per cent. were lymphocytes. Tubercle bacilli were found in the pellicle which formed later.

Blood-Sugar:-

←----------20/6/36----------→ ←--21/6/36--→

Time.	8.15 p.m.	8.30 p.m.	9 p.m.	10 p.m.	11 p.m.	1 a.m.	2 p.m.
Blood-Sugar. mgm.%	256.4	434.8	264.7	200.0	145.1	71.9	73.2
No.of hours after convulsion.	¼	½	1	2	3	5	18

22.6.36. An X-ray of the chest showed areas of consolidation in both lungs.

The child's general condition grew steadily worse.

27.6.36. Died. Permission for an autopsy was not granted.

Case 24.

C.T., a girl of 4 years. Admitted 20.7.36. She was a normal full-time baby and appeared to thrive for the first eight months. Soon after this, however, she began to take fits and it was noticed that she was unable to sit up without support. There was marked hypotonia of the limbs, and though this improved with massage, her mental condition failed to improve. It became obvious that she was definitely backward. The convulsions, which were very short, became more frequent and were controlled by luminal. In the summer of 1936 the fits became more severe and prolonged in character and the child was admitted for investigation.

During examination the child had a convulsion affecting the left side of the body and lasting about half a minute. She became cyanosed and the respirations were stertorous for another half-minute, but thereafter the child seemed quite well. She was moderately well-nourished, of good colour, cheerful, but quite insane. There was marked hypotonia of the limbs and she could neither stand nor walk alone. The plantar responses were extensor but otherwise there was nothing remarkable in the nervous system. The heart, lungs and abdomen appeared healthy. The urine contained nothing abnormal.

Blood-Sugar:-

Time.	9.30 a.m.	10 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	3.30 p.m.	4.30 p.m.
Blood-Sugar. mgm.%	65.3	68.5	65.3	54.3	65.3	66.6	73.0	65.3
No.of hours after convulsion.	5'	35'	1 hr. 5'	2hrs. 5'	3hrs. 5'	5hrs 5'	6 hrs 5'	7 hrs. 5'

Lumbar puncture was done and 20 c.c. clear fluid removed. This contained three cells per c.mm. and Pandy's test and the Wassermann reaction were both negative.

Case 25.

E.O'N., a girl aet. $5^{9}/12$ years. Admitted 27.3.37. She was a normal full-time healthy child and was first admitted to hospital at the age of $1^{4}/12$ years with a history of having fallen on the back of her head and four hours later developed a severe generalised convulsion which lasted about ten hours. The condition was diagnosed as encephalitis and the left arm and leg were left in a state of spastic paralysis. This gradually improved with massage but the child continued to take bouts of tonic fits every few months. These came on every ten minutes and she often had as many as twenty in a day.

At 2 a.m. on 13.7.35 she had a minor fit and at 2.30 p.m. she had a severe clonic spasm which lasted two-and-a-half hours. Lumbar puncture at this time revealed no abnormality but the urine obtained some hours after the convulsion contained abundant sugar but no acetone. The phenyl-hydrazine test showed definite glucosazone crystals.

Two months later the child was readmitted with a history of having had one to three minor fits per week. At 9 a.m. on 19.9.35 and again at 2 p.m. she had a severe fit lasting about two hours in each case. The convulsions appeared to affect chiefly the right arm and leg. The right knee-jerk could not be elicited while the left was exaggerated. Examination of the cerebro-spinal fluid was again negative. The urine contained sugar and on this occasion a moderate amount of acetone.

The child was not seen again till 27.3.37 when she was admitted after a severe fit starting at 11.45 the previous night and which lasted about one hour. On examination she was found to be a moderately well-nourished girl, dull but not acutely ill. Apart from enlarged and septic tonsils, nothing abnormal was found in the alimentary, cardiac or respiratory systems. The right knee-jerk was sluggish and the left very active. The left plantar response was extensor and the right flexor.

Blood-Sugar:-

←----------27/3/37----------→ <28th> <29th>

Time.	9.45 a.m.	10.45 a.m.	11.45 a.m.	12.45 p.m.	1.45 p.m.	2.45 p.m.	3.45 p.m.	4.15 p.m.	4.45 p.m.	12 mid-night	4 a.m.	8 a.m.	9.30 a.m.
Blood-Sugar mgm.%	113.6	117.6	80.6	55.2	59.1	52.9	43.5	52.1	50.2	66.6	42.2	45.2	77.5
No.of hours after convulsion.	10	11	12	13	14	15	16	$16\frac{1}{2}$	17	$24\frac{1}{4}$	$28\frac{1}{4}$	$32\frac{1}{4}$	$57\frac{3}{4}$

The urine at 9 a.m. on 27.3.37 contained sugar and a large amount of acetone.

Case 25 (contd.).

30.3.37. Sugar tolerance curve. 14 gms. glucose in 140 c.c.water.

Fasting.	½ hour.	1 hour.	1½ hours.	2 hours.
71.9	169.5	120.5	86.2	61.7 mgm. per 100 c.c.

31.3.37. Dismissed well.

Case 26.

J.A., a girl aet. 1 year. Admitted 10.6.37. She was a full-time healthy infant who throve well and was developing normally. Four hours before admission she had a generalised convulsion which lasted off and on until she was brought to hospital.

On examination she was found to be a pale, well-nourished child conscious but acutely ill. T. 98.4°F., P. 120, R. 30. The throat was very red and the tonsils enlarged. The heart, lungs and abdomen seemed normal and there were no signs of rickets. Lumbar puncture revealed a clear fluid normal in every way.

Serum Calcium 11.3 mgm. per cent.
Serum Phosphorus 3.2 mgm. per cent.
Plasma Phosphatase 19.7 units.

The urine contained no abnormal constituent.

Blood-Sugar:-

←---------10/6/37----------→ ←-11/6/37→

Time.	2.30 a.m.	9.30 a.m.	11.30 a.m.	12.30 p.m.	3 p.m.	5 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	101.0	69.1	54.9	65.8	59.2	78.1	88.5
No. of hours after convulsion.	7	14	16	17	19½	21½	38

11.6.37. The child seemed very well but pale.

14.6.37. Blood-sugar at 9.30 a.m. - 70.9 mgm. per cent.

19.6.37. Dismissed well.

Case 27.

E.M., a girl of $2^{5}/12$ years. Admitted 30.1.36. A full-time healthy baby who throve well until five months before admission when she took whooping-cough followed by broncho-pneumonia. Two weeks before admission the ears began to discharge and a week later the child took a fit and was admitted to a surgical ward unconscious. A lumbar puncture was done and the cerebro-spinal fluid found to be under greatly increased pressure and containing seventy cells per c.mm., of which about eighty per cent. were polymorphs. Pandy's test was markedly positive. A skiagram of the chest revealed the 'snowstorm' appearance of subacute miliary tuberculosis. The Mantoux intradermal tuberculin test gave a markedly positive reaction.

On admission to the medical ward the patient was found to be a rather poorly-nourished child, drowsy and irritable, and with double otorrhea. T. 98°F., P. 110, R.28. There was marked nuchal rigidity and Kernig's sign, but no paralysis or other disturbance of the nervous system. Nothing abnormal was detected in examination of the heart, lungs or abdomen. Ophthalmoscopic examination revealed normal eye grounds.

31.1.36. Lumbar puncture repeated. The cerebro-spinal fluid on this occasion contained only 20 cells per c.mm. and these were mainly lymphocytic in type. Pandy's test was still markedly positive.

The child remained very ill.

1.2.36. Sugar tolerance curve after 9 gms. glucose in 90 c.c. water.

Fasting.	½ hr.	1 hr.	1½ hrs.	2 hrs.
51.6	89.3	62.9	70.4	62.1 mgm. per 100 c.c.

10.2.36. The child's general condition was rather better. She could sit up and feed herself. Repeated examination of the cerebro-spinal fluid, the aural discharge and the sputum, for tubercle bacilli proved unsuccessful.

20.2.36. Lumbar puncture repeated. About 6 c.c. of clear cerebro-spinal fluid were removed. The fluid contained only 6 cells per c.mm., but the protein was still markedly increased. The Wassermann reaction of both blood and cerebro-spinal fluid was negative.

5.3.36. The child developed dactylitis of the left ring finger and later several small cold abscesses in the arms and legs.

16.3.36. At 3.30 p.m. the child suddenly went into a convulsion

Case 27 (contd.).

involving the whole body, but chiefly the right arm and right side of face. T. 102°F., P. 144, R. 40. The convulsion lasted for five hours. Lumbar puncture revealed no change in the cerebro-spinal fluid from the previous examination.

Blood-Sugar:-

←----------16/3/36----------→

Time.	4 p.m.	4.30 p.m.	5 p.m.	5.30 p.m.	6 p.m.	6.30 p.m.	7 p.m.	8 p.m.	9 p.m.
Blood-Sugar. mgm.%	80.6	88.5	119.0	156.2	188.6	196.1	121.9	232.5	126.5
No.of hours after convulsion.	½	1	1½	2	2½	3	3½	4½	5½

←----------17/3/36----------→

Time.	4 a.m.	9.30 a.m.	10.30 a.m.	11 a.m.	11.30 a.m.	12 noon
Blood-Sugar. mgm.%	138.8	86.9	103.1	101.0	90.1	59.1
No.of hours after convulsion.	12½	18	19	19½	20	20½

The sugar content of the cerebro-spinal fluid at 5 p.m. was 62.8 mgm. per 100 c.c.

Urine obtained at 6.30 p.m. contained sugar but no acetone.

17.3.36. The child was very ill, but there was no residual paralysis and nothing abnormal was detected in the nervous system. There were some moist râles at both lung bases. A skiagram of the skull showed no abnormality.

19.3.36. At 7 a.m. the child took another convulsion which continued for two hours.

Blood-Sugar:-

Case 27 (contd.).

Time.	8.30 a.m.	9.30 a.m.	10 a.m.	10.30 a.m.	11 a.m.	11.30 a.m.	12 noon	12.30 p.m.	1 p.m.	2 p.m.	3 p.m.
Blood-Sugar. mgm.%	135.1	188.7	96.1	144.9	126.5	126.5	60.6	66.6	64.9	120.4	82.6
No.of hours after convul-sion.	1½	2½	3	3½	4	4½	5	5½	6	7	8

24.3.36. The general condition was gradually deteriorating.

31.3.36. Some thick creamy pus was aspirated from a cold abscess in the arm and microscopic examination revealed scanty tubercle bacilli.

1.4.36. The child was wasted and comatose.

2.4.36. Died at 8.30 a.m. Permission for an autopsy was refused.

Case 28.

W.M., a boy aet. $2^{3}/12$ years. Admitted 2.11.35. A full-time baby delivered as a breech by instruments. He was cyanosed at birth and required resuscitation, but thereafter throve well, and was healthy till three days before admission when he developed rhinitis. At 8.20 a.m. on the day of admission he took a convulsion lasting two hours and affecting chiefly the left side of the body.

On admission the boy was still in the convulsion. T. 98°F., P. 120, R. 18. The head and eyes were deviated to the right, and the left angle of the mouth, the left arm and leg were twitching. The respirations were deep and stertorous. The pupils were dilated and fixed, the right being larger than the left. The knee-jerks were elicited with difficulty, but the abdominal and cremasteric reflexes were absent. The right plantar response was flexor and the left doubtful. The boy was well-nourished, and nothing abnormal was detected in the other systems except a slight reddening of the throat. The urine contained a trace of sugar and acetone.

Blood-Sugar:-

←----------11/11/35----------→ <12/11/35>

Time.	9.20 a.m.	9.50 a.m.	10.20 a.m.	10.50 a.m.	11.20 a.m.	11.50 a.m.	12.20 p.m.	12.50 p.m.	1.50 p.m.	2.50 p.m.	3.50 p.m.	5.30 p.m.	10.30 a.m.
Blood-Sugar. mgm.%	85.4	133.3	100.0	109.8	91.6	77.5	65.3	72.4	73.5	51.5	40.9	36.3	66.6
No. of hours after convulsion.	1	$1\frac{1}{2}$	2	$2\frac{1}{2}$	3	$3\frac{1}{2}$	4	$4\frac{1}{2}$	$5\frac{1}{2}$	$6\frac{1}{2}$	$7\frac{1}{2}$	9hrs 10'	26hrs. 10'

12.11.35. The child was fairly bright and seemed comfortable. There was no paresis of limbs and all reflexes were elicited except the cremasterics. The cerebrospinal fluid obtained by lumbar puncture was normal, and the Wassermann reaction was negative.

20.11.35. The child had no further convulsions and was dismissed well.

26.4.37. The child was readmitted at 11 a.m. He had been well for six months after dismissal, after which he began to take fits every few weeks. These were apparently only tonic, as there were never any twitching movements. They usually lasted half-an-hour and the child was unconscious during this time. He was well

Case 28 (contd.).

and free from fits for eight months before readmission. On the morning of the day of admission he complained of pain in the right side of the head and at 9.30 a.m. he took a convulsion lasting about fifteen minutes, after which he was unconscious for about one hour.

On admission the child was semi-comatose and breathing stertorously. The only abnormality in the nervous system was that the abdominal and cremasteric reflexes could not be elicited. The other systems presented nothing abnormal. The urine obtained at 11.45 a.m. contained neither sugar nor acetone, while that passed at 4 p.m. contained a moderate amount of acetone.

Blood-Sugar:-

28/11/37

←----------26/4/37----------→ ←-----27/4/37---------→

Time.	11.30 a.m.	11.35 a.m.	12.5 p.m.	12.35 p.m.	2.35 p.m.	3.35 p.m.	5 p.m.	9.30 a.m.	10.30 a.m.	11.45 a.m.	3.30 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	133.8	88.5	81.3	88.5	87.7	75.2	149.2	56.2	59.3	42.9	57.1	82.6
Lactic Acid. mgm.%			16.6				23.8			17.3		
No. of hours after convul-sion.	2	2hrs. 5'	2hrs. 35'	3hrs. 5'	5hrs. 5'	6hrs. 5'	7½	24	25	26¼	30	48

27.4.37. The boy seemed well and quite bright. A skiagram of the skull showed no abnormality.

Sugar tolerance curve after 15 gms. glucose in 150 c.c. water.

Fasting.	½ hour.	1 hour.	1½ hours.	2 hours.
90.1	116.5	106.4	100.0	71.4 mgm. per cent.

30.4.37. Dismissed well.

Case 29.

A.M., a girl aet. $3^{2}/12$ years. Admitted 16.3.36. A healthy full-time infant who throve well and developed normally.

Ten days before admission began to vomit and this persisted. A few days later complained of headache and became drowsy and listless. Her appetite was poor and she appeared to lose weight.

On examination the patient was found to be an emaciated child barely conscious and resenting interference. There was marked rigidity of the neck, spine and hamstring muscles and retraction of the abdomen. The pupils were unequal and sluggish in action. The heart and lungs presented no abnormality. About 30 c.c. clear cerebro-spinal fluid were withdrawn by lumbar puncture. The Pandy test was strongly positive and the cells numbered 141 per c.mm. No organisms were detected on films.

17.3.36. At 2.20 p.m. the child took a convulsion. This lasted only a few minutes, but an hour later the convulsion recurred and continued intermittently for some hours.

Blood-Sugar:-

Time.	2.50 p.m.	3.20 p.m.	3.50 p.m.	4.20 p.m.	4.50 p.m.	5.20 p.m.
Blood-Sugar. mgm.%	144.9	144.9	250.0	303.0	119.0	135.7
No.of hours after convulsion.	½	1	1½	2	2½	3

The urine was not obtained.

18.3.36. The child died at 2.45 a.m. and permission for an autopsy was refused.

Case 30.

C.S., a girl aet. $1^{10}/12$ years. Admitted 20.11.35. A full-time healthy baby. Breast-fed for three months then whole-milk and throve.

The child had been very well till one day before admission when she was noticed to be listless and had a poor appetite. Next day she vomited everything and at 8 p.m. she took a generalised convulsion which lasted half-an-hour and was followed by a period of drowsiness.

On examination she was found to be a well-nourished child, flushed and very drowsy. T. 100.6°F., P. 166, R. 38. There was no nuchal rigidity and Kernig's sign was absent. The pupils were moderately dilated and active and the reflexes were physiological.

Examination of the heart, lungs and abdomen revealed no abnormality. The tongue was dirty and the throat red, the tonsils being noticeably enlarged. During examination the child passed a loose, green stool containing mucus and streaks of unaltered blood. There was nothing abnormal in the urine.

Blood-Sugar:-

	←20/11/35→	←21/11/35→						
Time.	10.30 p.m.	11.30 p.m.	12.30 a.m.	1.30 a.m.	2.30 a.m.	3.30 a.m.	9.30 a.m.	12 noon
Blood-Sugar. mgm.%	86.2	77.5	76.3	81.3	86.9	84.2	82.6	66.6
No.of hours after convulsion.	$2\frac{1}{2}$	$3\frac{1}{2}$	$4\frac{1}{2}$	$5\frac{1}{2}$	$6\frac{1}{2}$	$7\frac{1}{2}$	$13\frac{1}{2}$	16

23.11.35. The child continued to have stools containing blood and mucus, and B. dysenteriae (Flexner) was cultured from one of these. There were no further convulsions.

28.11.35. The general condition was much improved and the stools now almost normal.

2.12.35. Dismissed well.

Case 31.

D.M., a boy aet. $1^{11}/12$ years. Admitted 11.1.36. A healthy baby who throve well. He had pneumonia at eleven months, but recovered completely. Three days before admission he took a cold and at 10 p.m. on the night of admission he took a convulsion.

On examination he was found to be a well-nourished child having severe twitching movements of the face and mouth. He was cyanosed and breathing stertorously. T. 99°F., P. 130, R. 40. A few hours later he was completely conscious, but restless and flushed. The tongue was dirty, the throat red, and the tonsils much enlarged. The anterior cervical glands on both sides were enlarged. He had slight rickets. Examination of the heart, lungs, abdomen and nervous system revealed no abnormality. Lumbar puncture was performed on admission but the cerebro-spinal fluid found to be quite normal. The sugar content of the fluid was 142.8 mgm. per cent. The urine contained a trace of sugar but no acetone.

Blood-Sugar:-

←----------12/1/36----------→

Time.	2.30 a.m.	3 a.m.	3.30 a.m.	12.30 noon	2.15 p.m.	3.15 p.m.	4.15 p.m.	5.15 p.m.	7.15 p.m.	9.15 p.m.
Blood-Sugar. mgm.%	93.4	96.1	100.0	75.2	70.4	140.8	125.0	81.9	68.2	66.2
No.of hours after convulsion.	$4\frac{1}{2}$	5	$5\frac{1}{2}$	$14\frac{1}{2}$	$16\frac{1}{4}$	$17\frac{1}{4}$	$18\frac{1}{4}$	$19\frac{1}{4}$	$21\frac{1}{4}$	$23\frac{1}{4}$

←--------13/1/36----------→

Time.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.
Blood-Sugar. mgm.%	93.4	113.6	98.0	91.7
No.of hours after convulsion.	$35\frac{1}{2}$	$36\frac{1}{2}$	$37\frac{1}{2}$	$38\frac{1}{2}$

13.1.36. The urine contained nothing abnormal.

Case 31 (contd.).

13.1.36. At 9 p.m. he had another convulsion.

Blood-Sugar:-

←-13.1.36-→ ←----------14/1/36-------→ ←15.1.36→

Time.	10 p.m.	12 mid-night	1 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	9.30 a.m.
Blood-Sugar. mgm.%	142.8	111.1	91.7	81.8	78.7	81.9	81.9
No.of hours after convulsion.	1	3	4	12½	13½	14½	36½

14.1.36. Tonsils much enlarged and red. Blood Wassermann reaction negative.

19.1.36. Dismissed.

One month later the child was readmitted and the tonsils and adenoids removed under ethyl-chloride anaesthesia.

Case 32.

J.K., a boy aet. 4 9/12 years. Admitted 31.1.37. A breech birth. Healthy child, bruised about the head. Throve well and developed normally. Pneumonia at one year. The boy had had difficulty in breathing for two years before admission and this was said to be due to enlarged tonsils and adenoids.

He was well till about 4 p.m. on the day of admission when he stopped playing and seemed inclined to lie down. A few hours later he complained of occipital pain which was later succeeded by pain in the right side of the neck.

At 10 p.m. he had a sudden attack of unconsciousness followed by twitching of the left side of the body.

On examination he was found to be a well-nourished child, semi-conscious and cyanosed. T. 98°F., P. 100, R. 22. The pupils were dilated but reacted to light; the reflexes with the exception of the abdominals were present. There was no nuchal rigidity and nothing abnormal was detected in the heart, lungs or abdomen. The cervical glands were palpably enlarged on both sides and the tonsils large and red. The urine at 12 midnight on 31.1.37 contained a trace of sugar and a moderate amount of acetone. Lumbar puncture revealed a normal cerebro-spinal fluid.

Blood-Sugar:-

31/1/37 ←---------1/2/37----------→

Time.	12 mid-night	10 a.m.	11 a.m.	12 noon	1 p.m.	2.30 p.m.
Blood-Sugar. mgm.%	144.9	52.1	42.0	36.5	49.5	47.1
No.of hours after convulsion.	2	12	13	14	15	16½

1.2.37. The boy seemed fairly well though a little drowsy. The urine contained no sugar but a large amount of acetone.

Since the blood-sugar was at a low level it was decided to watch the effect of giving glucose intravenously.

At 2.30 p.m. 9 gms. glucose were given intravenously in the form of twenty per cent. solution in normal saline.

Case 32 (contd.).

	Before	2' after	15' after	30' after	45' after	60' after	75' after	90' after
Blood-Sugar. mgm.%	47.1	333.3	131.7	111.1	74.1	83.3	76.8	78.1

4.2.37. Sugar tolerance curve - 15 gms. glucose in 150 c.c.water.

	F.	½ hr.	1 hr.	1½ hrs.	2 hrs.
Blood-Sugar. mgm.%	56.5	133.3	116.3	116.3	54.7

6.2.37. Intravenous glucose curve repeated.

	F.	2'	15'	30'	45'	60'	75'	90'
Blood-Sugar. mgm.%	71.4	430.1	266.7	216.2	134.7	83.3	74.1	71.4

11.2.37. Tonsils and adenoids removed under ethyl chloride anaesthesia.

19.2.37. Irregularly dismissed.

Case 33.

C.C., a girl aet. 11 years. Admitted 21.2.36. She was a full-time child, cyanosed at birth. Apart from whooping-cough and measles she was healthy till the age of five, when she began to have twitching movements of the head and eyes. These gradually became worse until finally she was taking definite convulsions. The fits lasted about ten minutes and occurred almost every day. She was unable to attend school for a time, but remained mentally alert. At the age of eight years, under homoeopathic treatment she became free from fits for a period of about five months, but thereafter they became very frequent again. Just before admission she had about twenty, each one lasting for about two minutes and being followed by a period of unconsciousness lasting a few minutes.

On examination she was found to be a rather dull-looking, well-nourished child. T. 99.4°F., P. 86, R. 20. Nothing abnormal was detected in the examination of the heart, lungs, abdomen or nervous system. The Mantoux tuberculin test gave a very positive reaction.

27.2.36. At 11.55 a.m. the girl took a short fit followed by a period of unconsciousness, the whole episode being over in the space of a few minutes. The urine contained nothing abnormal.

Blood-Sugar:-

Time.	12 noon	12.30 p.m.	2.30 p.m.	3.30 p.m.	4.30 p.m.
Blood-Sugar. mgm.%	101.0	95.2	102.2	97.1	91.7
No.of hours after convulsion.	5'	35'	2 hrs. 35'	3 hrs. 35'	4 hrs. 35'

28.2.36. Luminal gr. ½ t.i.d. This lessened the frequency of the fits but did not stop them.

18.3.36. Luminal gr. ½ was given five times a day and this finally controlled the fits.

24.4.36. Dismissed home.

Case 34.

E.S., a boy aet. 8/52 year. Admitted 3.2.36. Normal full-time healthy child. Breast-fed for six weeks then milk, water and sugar. He throve well till the night before admission when he took a generalised convulsion at 10 p.m. This lasted only a few minutes but was followed by three similar fits before midnight.

On admission the child was found to be well-nourished and of good colour and not acutely ill. T. 98.4°F., P. 140, R. 50. A well-marked facial phenomenon could be elicited, but Trousseau's sign was negative. Nothing abnormal was detected in examination of the various systems.

Serum Calcium 8.0 mgm. per cent.
Plasma Phosphatase 17.8 units.

Blood-Sugar:-

Time.	10 a.m.	4.40 p.m.	5.10 p.m.	5.40 p.m.	6.10 p.m.	7.40 p.m.	8.40 p.m.
Blood-Sugar. mgm.%	63.3	87.7	98.0	89.3	125.0	109.9	133.3
No.of hours after convulsion.	12	18 hrs. 40'	19hrs. 10'	19hrs. 40'	20hrs. 10'	21 hrs. 40'	22 hrs. 40'

↑ convulsion

At 4.30 p.m. the child took a convulsion lasting a few minutes and at 6 p.m. received calcium chloride grs. x.

4.2.36. The convulsions continued but the calcium chloride was stopped since the child was vomiting. Chloral gr. i every three hours was instituted instead.

13.2.36. In spite of chloral the convulsions persisted.

14.2.36. Chloral stopped. Luminal gr. ¼ t.i.d.

The fits were finally controlled by luminal, but the child developed gastro-enteritis, which necessitated the administration of parenteral fluid.

25.3.36. Dismissed home much improved.

Case 35.

H.McV., a girl aet. 1 6/12 years. Admitted 14.9.36. A healthy baby six weeks premature. She throve well at first, but at ten months she began to take short fits lasting only a few minutes and occurring at intervals of two or three hours. There were periods of several days or weeks when there were no fits, but they always recurred. On the day of admission she had many short generalised convulsions.

On examination the child was found to be large, fat and healthy-looking. The fontanelle was still patent, but there were no signs of rickets. The child was cheerful, but looked mentally deficient. Nothing abnormal was detected in the heart, lungs, abdomen or nervous system.

17.9.36. At 10.45 a.m. the child had a convulsion lasting about one minute. T. 98.2°F., P. 140, R.34.

Serum Calcium 10.9 mgm. per cent.
Serum Phosphorus 4.0 mgm. per cent.
Plasma Phosphatase 13.5 units.

Blood-Sugar:-

Time.	10.50 a.m.	11.15 a.m.	11.45 a.m.	12.45 p.m.	2.45 p.m.	3.45 p.m.	4.45 p.m.
Blood-Sugar. mgm.%	72.4	69.4	78.1	59.5	86.9	78.1	69.9
No.of hours after convulsion.	5'	½ hr.	1	2	4	5	6

The urine contained nothing abnormal.

22.9.36. The child's general condition remained good and there were no further convulsions, but she was obviously very backward. A skiagram of the skull revealed no abnormality.

24.9.36. Dismissed well.

Case 36.

J.G., a boy aet. 2 years. Admitted 11.1.36. He was a normal full-time baby, healthy at birth and throve well. At one month both ears began to discharge and this continued at intervals till admission. At one year he had a convulsion and made a good recovery. On the night before admission he vomited, and at 7.15 a.m. the next day he went into a convulsion. The twitching movements were generalised and continued till admission.

On examination he was found to be a well-nourished child, almost moribund, the skin ashen-grey and moist. There were slow convulsive movements of the arms, and the head and eyes were deviated to the left. The deep and superficial reflexes were present but sluggish. The heart and abdomen seemed normal. All over both lungs there were moist râles. The left ear was discharging. The urine contained albumin.

Blood-Sugar:-

Time.	3.15 p.m.	4.15 p.m.	5.15 p.m.	6.15 p.m.	7.30 p.m.
Blood-Sugar. mgm.%	25.0	31.2	28.8	14.5	11.2*
No.of hours after convulsion.	8	9	10	11	12¼

* Specimen of blood obtained from the heart after death.

The child died at 7.30 p.m. At autopsy he was found to have acute broncho-pneumonia involving chiefly the right side, otitis media and longitudinal sinus thrombosis. The liver showed fatty degeneration, but the suprarenals and pancreas were normal.

A.H., a boy aet. 11 6/12 years. Admitted 2.3.36. Healthy baby. Measles at one year and whooping-cough at three years. At three years he developed a septic infection of the right hand and elbow joint which necessitated a stay of a year in hospital.

Four months before admission he took a fit, during which he is said to have spun round in a circle for a few minutes, and then to have fallen unconscious. The whole thing lasted about ten minutes and the boy was dazed for a few minutes longer. At first the fits occurred about once a month, but just before admission he was having about ten in a week.

On examination he was found to be a fair-sized thin boy, pale and rather unwell. T. 99.4°F., P. 100, R. 22. The right elbow and thumb were ankylosed and there were various septic areas on his chest and abdomen. Examination of the heart, lungs and abdomen was negative. There was ptosis of both eyes and a latent squint, but the pupils were equal and active. The superficial and deep reflexes were easily elicited and the plantar responses were flexor. There was no paresis and the boy walked well along a line. Romberg's sign was negative. On ophthalmoscopic examination the disc margins were seen to be slightly indistinct. The Wassermann reaction of the blood was negative. At 48 hours the Mantoux tuberculin test was strongly positive and at 96 hours even more so. The urine contained nothing abnormal.

He continued to have one or two short fits every day. Luminal diminished their severity but not their frequency.

17.3.36. Just after dinner he had a short convulsion which lasted about two minutes.

Blood-Sugar:-

Time.	12.45 p.m.	1.45 p.m.	2.45 p.m.
Blood-Sugar. mgm.%	140.8	125.0	95.2
No. of hours after convulsion.	5 mins.	1 hr. 5'	2 hrs.5'

18.4.36. The boy's mental condition appeared to be deteriorating. He could not be kept in bed and took to running about the ward naked. Chloral had little effect and he became quite unmanageable.

20.4.36. Dismissed home.

Case 38.

A.McM., a boy aet. 3/12 year. Admitted 17.2.36. A full-time, healthy baby who was breast-fed for one month and then put on a feed of milk, sugar and water.

The child throve well till five weeks before admission when he began to have short convulsions, which occurred two or three times daily. On the night of admission he had a long series of these between 7.30 p.m. and 10 p.m.

On admission the patient was found to be a pale, quiet, under-nourished child. T. 98.4°F., P. 130, R. 36. There was no nuchal rigidity and no Kernig's sign, and examination of the heart, lungs, abdomen and nervous system was completely negative. Neither Chvostek's nor Trousseau's sign could be elicited. The urine showed no abnormality.

Serum Calcium 8.1 mgm. per cent.
Serum Phosphorus 5.6 mgm. per cent.
Plasma Phosphatase 14.4 units.

Blood-Sugar:-

<18/2/36> <19.2.36>

Time.	12.30 a.m.	1 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	1.30 p.m.	2.30 p.m.	3.30 p.m.	4.30 p.m.	5.30 p.m.	8.30 p.m.	11.30 p.m.	10.30 a.m.
Blood-Sugar. mgm.%	64.9	60.9	56.2	62.1	74.6	70.9	70.9	60.9	144.9	119.0	97.1	107.5	99.0	109.9
No.of hours after convulsion.	5	5½	14	15	16	17	18	19	20	21	22	25	28	39

There were no further convulsions after admission.

21.2.36. Dismissed well.

Case 39.

J.R., a girl aet. 7/12 year. Admitted 26.4.36. A healthy, full-time baby who was fed on whole milk, then Ostermilk, but did not thrive well.

On the morning of the day of admission she was noticed to be breathing heavily, had a nasal discharge and a slight cough. She vomited once and rapidly became very ill. The motions appeared to be normal.

On admission the child was moribund and at 12 noon she took a convulsion. She was pale, cyanosed and twitching, and showed evidence of gross neglect. There were some râles at both bases, but otherwise the examination of the systems was negative. Lumbar puncture revealed a clear fluid containing 63 cells per c.mm., but with a negative Pandy test. The urine was not obtained.

Time.	12 noon.	3 p.m.	4 p.m.
Blood-Sugar. mgm.%	103.1	33.0	12.9*
No.of hours after convulsion.	-	3	4

* Blood taken from the heart immediately post-mortem.

The child died at 4 p.m., and at autopsy was found to have acute ileo-colitis. There was no lesion in the suprarenal glands.

Case 40.

J.C., a boy aet. 1 3/12 years. Admitted 29.3.36. A full-time, healthy baby. Throve well and developed normally.

Two weeks before admission became listless and began to vomit. The vomiting persisted and he became very drowsy. The bowels became very constipated.

On admission he was found to be a well-nourished child, acutely ill and comatose. T. 98°F., P. 120, R. 26. There was well-marked nuchal rigidity, but Kernig's sign was not elicited. The heart, lungs and abdomen revealed no abnormality. Lumbar puncture gave a slightly turbid fluid under normal pressure. The cell count was 198 c.mm., of which eighty per cent. were lymphocytes. The protein was much increased. The urine exhibited no abnormality.

30.3.36. The child had a short right-sided convulsion at 10.15 a.m. After this his condition gradually gre worse and he died at 8.30 p.m.

Blood-Sugar:-

Time.	10.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	3.30 p.m.	4.30 p.m.
Blood-Sugar. mgm.%	131.6	151.5	158.7	112.3	102.0	120.5
No.of hours after convulsion.	15'	1 hr.15'	2hrs.15'	4 hrs. 15'	5 hrs. 15'	6 hrs. 15'

Permission was not obtained for an autopsy.

Case 41.

R.D., a boy aet. 6½ years. Admitted 14.4.36. A full-time, healthy child who developed normally. At the age of 11/12 year and 1 11/12 years he had short fits. Two years before admission, after a severe scald, he had a series of short fits. One year later he developed pneumonia and made a good recovery. He was well thereafter till two days before admission when he developed headache and vomited. At 2.30 p.m. and again at 4 p.m. on the day of admission he had a convulsion lasting about 10 minutes.

On examination the boy was found to be flushed but not acutely ill. T. 104°F., P. 140, R. 40. Nothing abnormal was detected in the heart, lungs, abdomen or nervous system. The urine contained acetone but no sugar.

Blood-Sugar:-

	←14/4/36→	←15/4/36→						
Time.	4.30 p.m.	6.30 p.m.	12.30 a.m.	10.30 a.m.	1.30 p.m.	2.45 p.m.	4 p.m.	5 p.m.
Blood-Sugar. mgm.%	96.1	95.2	95.2	78.1	105.3	95.2	109.2	80.0
No. of hours after convulsion.	2	4	10	20	23	24¼	25½	26½

15.4.36. The temperature subsided and the boy seemed very well apart from redness of the throat.

21.4.36. Dismissed well.

Case 42.

T.W., a boy aet. 8/12 year. Admitted 14.12.36. A full-time, healthy child. Seven months before admission an elder sister, J.W. (Case 21), had died of tuberculous meningitis.

The boy throve well till six days before admission when he became fevered, vomited and developed a cough. The breathing became laboured and the child rapidly became very ill. He had a generalised convulsion at 7 a.m. which lasted an hour.

On admission he was found to be a pale, poorly-nourished boy, gravely ill and drowsy. T. 99.2°F., P. 136, R. 50. The fontanelle was bulging, but there was no nuchal rigidity. The abdomen was scaphoid and the liver and spleen were much enlarged. There was dulness to percussion at the right apex, in front and behind, and the respiratory murmur was tubular at this area. No superficial or deep reflexes could be elicited. Lumbar puncture gave about 20 c.c. of turbid fluid under increased pressure. The Pandy test was markedly positive and the cells increased to 165 per c.mm. Films showed abundant cocci and a heavy pneumococcal growth resulted on culture. Nothing abnormal was observed in the urine.

Blood-Sugar:-

Time.	9.50 a.m.	10.50 a.m.	11.50 a.m.
Blood-Sugar. mgm.%	149.2	59.5	50.5
No. of hours after convulsion.	2 hrs. 50'	3 hrs. 50'	4 hrs. 50'

The child died at 12.50 p.m. on the day of admission. At autopsy he was found to have tuberculosis of the lungs, generalised miliary tuberculosis, acute suppurative broncho-pneumonia and pneumococcal meningitis.

Case 43.

J.J., a boy aet. 6/52 year. Admitted 11.1.37. A full-time baby delivered by instruments. He was artificially fed from the beginning and throve. At two weeks he developed a nasal discharge, which persisted till admission. Four days before admission he had two short convulsions and the next day had two more. At 8 p.m. on the day before admission he took a convulsion and from then till 5.30 a.m. the convulsive movements kept recurring.

On examination the baby was found to be fair-sized and moderately well nourished. T. 98.8°F., P. 130, R. 32. The twitching movements were much more marked on the left side. The fontanelle was flat and there was no nuchal rigidity. There was some rhinitis, but the mouth and throat were clean. The liver and spleen were palpably enlarged. Chvostek's sign was positive on the right side, but Trousseau's sign could not be elicited. The heart, lungs and nervous system appeared to be normal. The urine contained a moderate amount of acetone.

Blood-Sugar:-

	←----11/1/37------→				←----------12/1/37----------→					
Time.	2 p.m.	3 p.m.	4 p.m.	5 p.m.	5 a.m.	6.30 a.m.	10 a.m.	11 a.m.	1 p.m.	2 p.m.
Blood-Sugar. mgm.%	59.1	47.1	44.2	66.6	83.3	59.0	76.7	62.8	72.4	65.7
No.of hours after convulsion.	18	19	20	21	33	34½	38	39	41	42

12.1.37. Serum Calcium 6.5 mgm. per cent.
Serum Phosphorus 4.0 mgm. per cent.
Plasma Phosphatase 23.6 units.

In spite of calcium gluconate intravenously and intramuscularly the convulsions continued. Chloral also failed to control them but luminal in ¼ gr. doses every six hours for thirty-six hours finally did so.

14.1.37. Serum Calcium 7.2 mgm. per cent.

21.1.37. Serum Calcium 8.0 mgm. per cent.

25.1.37. Serum Calcium 8.8 mgm. per cent.

25.1.37. The child was dismissed home well.

Adexolin was prescribed in three-minim doses.

Case 44.

J.W., a boy aet. $6^{10}/12$ years. Admitted 22.3.36. Fairly healthy baby. Throve well and developed normally. Diphtheria followed by scarlet fever at $3^{6}/12$ years. Whooping-cough and pneumonia at 6 years.

He took a generalised convulsion at the age of one year, but had no more till a year later, when he began to take fits very frequently. For a while they had occurred every two weeks but of late they had come on about every three months.

At 7 a.m. on the day of admission the boy took a generalised convulsion which lasted till 10.30 a.m., after which he fell into a deep sleep.

On examination he was found to be a well-nourished boy of good colour but semi-comatose. T. 98.8°F., P. 110, R. 28. The pupils were, however, active and the superficial and deep reflexes were easily elicited. There was no abnormality of any system and a few hours later the boy seemed perfectly well and mentally alert. The urine was obtained at 3.30 p.m. and found to contain abundant sugar but no acetone. At 6 p.m. and again at 9 p.m. there was no sugar in the urine.

Blood-Sugar:-

←----------22/3/36----------→

Time.	12 noon	12.30 p.m.	1 p.m	1.30 p.m.	2 p.m.	2.30 p.m.	3 p.m.	3.30 p.m.	4 p.m.	5 p.m.	10 p.m.
Blood-Sugar. mgm.%	68.9	90.1	120.5	90.1	82.8	82.8	78.1	90.1	120.5	111.1	89.3
No.of hrs. after convulsion	5	5½	6	6½	7	7½	8	8½	9	10	15

←---------23/3/36---------→

Time.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	3.30 p.m.
Blood-Sugar. mgm.%	112.3	89.3	102.0	85.5	91.7
No.of hrs. after convulsion	26½	27½	28½	29½	32½

23.3.36. No glycosuria throughout the day.

26.3.36. Lumbar puncture revealed normal cerebro-spinal fluid. There was no sugar observed in the urine during the rest of the boy's stay in hospital.

31.3.36. Dismissed well.

Case 45.

M.G., a girl aet. 5 years. Admitted 15.3.37. A healthy baby who throve well and developed normally. One month before admission she had pneumonia. One week before admission she complained of pain in her right ear, which later began to discharge. A swelling appeared behind the right ear and this gradually increased in size pushing the ear down and out.

On admission a curved incision was made behind the right ear and thick greenish pus found in the superficial tissues. The opening was extended into the mastoid cells but no further pus found.

20.3.37. The temperature was unsettled, but the child appeared well. The ear and the wound were discharging profusely.

22.3.37. The child had a generalised convulsion at 6 p.m., with a marked rise in temperature and pulse. T. 102.2°F., P. 128, R. 34.

The patient remained ill, but the wound continued to discharge freely.

31.3.37. She had a generalised convulsion at 2.45 p.m. lasting 30 minutes. There was marked nuchal rigidity and Kernig's sign was positive. A lumbar puncture was done and 6 c.c. of straw-coloured fluid withdrawn. This showed a moderate cellular increase and fairly abundant streptococci. The child's general condition rapidly became worse.

Blood-Sugar:-

	←------31/3/37---------→			←---1/4/37---→	
Time.	3.15 p.m.	4.15 p.m.	5.15 p.m.	10 a.m.	3 p.m.
Blood-Sugar. mgm.%	218.5	169.5	156.3	76.4	72.5
No. of hours after convulsion.	½	1½	2½	19¼	24¼

The urine at 9 a.m. on 1.4.37 contained no sugar but abundant acetone.

2.4.37. Died. Permission for an autopsy was refused.

Case 46.

J.C., a boy aet. 2 days. Admitted 28.11.35. A full-time child rather cyanosed at birth but breathed well within a few minutes.

At 8 p.m. on the night before admission the infant began to have twitching movements of the face and hands. Later these movements became more severe, but the constitutional disturbance was not great.

On examination the baby was found to be well-developed and healthy-looking. T. 99°F., P. 138, R. 40. There was no external evidence of injury to the head or other parts. Nothing abnormal was detected in the heart, lungs, abdomen or nervous system. The urine contained no unusual constituent.

Blood-Sugar:-

	←28/11/35→	←30/11/35→	←1/12/35→	←2/12/35→
Time.	2 p.m.	2 p.m.	2 p.m.	2 p.m.
Blood-Sugar. mgm.%	46.5	112.3	90.9	119.0
No.of hours after convulsion.	18	-	-	-

The convulsions were treated with chloral, and finally responsed to this. The child did not suck well.

3.12.36. Dismissed well.

Case 47.

C.S., a boy aet. $1^{3}/12$ years. Admitted 13.12.35. A healthy baby till the age of six months when he had tetany. He made a good recovery and was well till about two months before admission when he became restless at night and developed a cough. He had frequent attacks of rhinitis. At 7 a.m. on the day of admission he took a convulsion which lasted ten minutes.

On examination he was found to be a large fat child with grunting respirations and frequent loose cough. T. 100°F., P. 150, R. 46. The tonsils were enlarged and red. No dulness could be made out but there were abundant rhonchi and râles all over both sides of the chest. Nothing abnormal was detected in the heart, abdomen or nervous system. The urine contained no abnormal constituent.

Serum Calcium 11.6 mgm. per cent.

Serum Phosphorus 3.3 mgm. per cent.

Plasma Phosphatase 11.4 units.

Blood-Sugar:-

←--------13/12/35----------→ <14/12/35>

Time.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	1.30 p.m.	2.30 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	78.1	80.0	93.4	102.0	100.0	87.7	79.4
No.of hours after convulsion.	$2\frac{1}{2}$	$3\frac{1}{2}$	$4\frac{1}{2}$	$5\frac{1}{2}$	$6\frac{1}{2}$	$7\frac{1}{2}$	$26\frac{1}{2}$

14.12.35. The child seemed well though the temperature was high. There were still a great many rhonchi in the chest.

17.12.35. Both membrana tympani were found to be bulging and red and were incised. The ears discharged for a few days and then dried up.

24.12.35. The child was very well except for a persistent cough. The chest was much clearer.

25.12.35. Dismissed.

Case 48.

O.M., a boy aet. 1/12 year. Admitted 22.5.36. A full-time baby born after a long labour. Cyanosed at birth but appeared to recover rapidly. He was breast-fed till four days before admission but never sucked well.

About 11 a.m. on the day of admission he began to have twitching movements of the right side of the body.

On examination the patient was found to be a small, thin child, fairly vigorous, but slightly dehydrated. T.97.6°F., P. 120, R. 46. Nothing abnormal could be elicited in the heart, lungs, abdomen or nervous system. The urine was in no way abnormal.

Blood-Sugar:-

←-----22/5/36------→ <23/5/36>

Time.	4 p.m.	5 p.m.	7.30 p.m.	10 a.m.
Blood-Sugar. mgm.%	24.7	23.7	50.0	66.6
No.of hours after convulsion.	5	6	8½	23

23.5.36. The child continued to have short convulsions.

24.5.36. No further convulsions.

Dismissed well.

Case 49.

P.C., a boy aet. 7/52 year. Admitted 20.4.36. A healthy full-time baby. Apart from some feeding difficulty the child appeared to thrive until the night before admission when he vomited his 10 p.m. feed. At 3.45 a.m. he began to have short convulsions which recurred frequently between then and 6.30 a.m.

On admission the child was found to be thin but vigorous. T. 98°F., P. 136, R. 40. Nothing abnormal was detected in the examination of the various systems. There was no craniotabes and neither Chvostek's nor Trousseau's sign was elicited. The urine contained no abnormal constituent.

Blood-Sugar:-

←----------20/4/36----------→ <21/4/36>

	9.45 a.m.	10.15 a.m.	10.45 a.m.	11.15 a.m.	11.45 a.m.	12.15 p.m.	1.15 p.m.	3.15 p.m.	4.15 p.m.	5.15 p.m.	12 mid-night	9.30 a.m.	11.30 a.m.
Blood-Sugar. mgm.%	91.3	67.1	61.7	72.5	75.2	76.3	95.2	84.0	119.0	94.3	90.1	79.4	108.7
No. of hours after convulsion.	6	6½	7	7½	8	8½	9½	11½	12½	13½	20¾	29¾	31¾

Serum Calcium 9.6 mgm. per cent.

Serum Phosphorus 3.6 mgm. per cent.

Plasma Phosphatase 13.0 units.

The child had no further convulsions after admission.

23.4.36. Dismissed well.

Case 50.

M.G., a boy aet. 4 years. Admitted 23/7/36. A full-time, healthy baby who throve well and developed normally.

The child had had no illnesses till the morning of the day before admission when he came in from play and vomited. He then began to have loose brown slimy motions. The vomiting and diarrhoea persisted throughout the day and night. The child became dull and listless and about 2.30 a.m. began to have convulsions.

On examination the patient was found to be a moderately well-nourished child practically moribund and having frequent convulsive seizures. T. 104°F., P. 170, R. 74. There was marked cyanosis and rigidity. Nothing abnormal could be detected in the heart, lungs, abdomen or nervous system. The urine was not obtained. The child died at 8 a.m.

Blood-Sugar:-

Time.	6.30 a.m.	8 a.m.
Blood-Sugar. mgm.%	8.5	11.7*
No.of hours after convulsion.	4	5½

* Blood taken from the heart immediately after death.

At autopsy the cause of death was found to be acute ileo-colitis (B. dysenteriae: Flexner). The lower ileum and whole of the colon were much congested and coated with fragments of inflammatory exudate. The pancreas and suprarenals appeared to be healthy.

Case 51.

D.W., a girl aet. $2^{3}/12$ years. Admitted 23.9.36. A healthy baby who throve well till two months before admission when she became cross and irritable, went off her food and slept badly. One month later she developed a right-sided cervical adenitis which was surgically treated by incision and scraping. Her general condition remained poor after the operation and at 8 p.m. on the night of admission she felt sick and vomited several times. She then took a generalised convulsion which lasted till admission at 1 a.m. The head and eyes were deviated to the right.

On examination the patient was found to be a well-nourished girl, conscious but rather drowsy. T. 100°F., P. 136, R. 40. There was a right-sided malar flush and spasticity of the right arm and leg, while the right hand was held in the claw position. The knee-jerks were easily elicited on both sides and the plantar responses flexor. The abdominal reflexes were active on the left side and sluggish on the right, the lower ones being absent. There was no nuchal rigidity. Nothing abnormal was detected in the examination of heart, lungs or abdomen. The tongue was furred, and the tonsils enlarged and red. The cervical glands were enlarged on both sides. The urine contained nothing abnormal.

Blood-Sugar:-

	←24/9/36→											25th	26th
Time.	1.30 a.m.	3 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	3.30 p.m.	5 p.m.	6 p.m.	8 p.m.	12 mid-night	9.30 a.m.	9.30 a.m.
Blood-Sugar. mgm.%	88.5	75.7	58.1	55.8	56.8	63.6	60.6	46.5	45.0	52.9	54.1	55.2	61.7	52.6
No.of hours after convulsion.	5	6½	13	14	15	16	18	19	20½	21½	23½	27½	37	61

24.9.35. Serum Calcium 11.4 mgm. per cent.
Serum Phosphorus 2.5 mgm. per cent.
Plasma Phosphatase 13.1 units.

25.9.35. The Mantoux tuberculin skin test gave a markedly positive reaction. A skiagram of the skull was normal and one of the chest showed veiling of both lungs.

29.9.35. The child was now very well. The spasticity of the right arm and leg had passed off. Ophthalmoscopic examination showed a tuberculoma of the choroid on the right side. The cerebro-spinal fluid was normal.

7.10.35. Dismissed home.

Case 52.

D.McL., a boy aet. $1^{1}/12$ years. Admitted 7.6.36. Healthy baby, though one month premature. Throve well on whole milk. Gastro-enteritis at $^{3}/12$ year.

Two-and-a-half hours before admission the child had a generalised convulsion which lasted only a few minutes, but which recurred four times in two hours.

On examination the child was found to be well-nourished, active and bright. T. 98.4°F., P. 120, R. 28. Nothing abnormal could be detected in the examination of the heart, lungs or abdomen. There was no nuchal rigidity or Kernig's sign and the reflexes were physiological. Chvostek's sign could not be elicited and there was no carpo-pedal spasm. The urine contained a trace of acetone but no other abnormal constituent.

Serum Calcium 12.0 mgm. per cent.

Serum Phosphorus 5.0 mgm. per cent.

Plasma Phosphatase 12.2 units.

Blood-Sugar:-

Time.	12.30 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.
Blood-Sugar. mgm.%	81.3	59.5	106.4	81.9	112.3	89.3
No.of hours after convulsion.	2½	11½	12½	13½	14½	16½

9.6.37. The child was now very well and was dismissed.

Case 53.

M.H., a girl aet. 11 years. Admitted 6.11.35. She was a healthy baby who throve well and developed normally. At the age of nine years she developed acute rheumatism and carditis. Two years later she was admitted to hospital on 8.5.35 with a history of fatigue and breathlessness of six weeks' duration. On examination there was nothing abnormal to be found except a loud systolic murmur at the apex conducted to the axilla. The Mantoux skin test, however, gave a markedly positive result and a skiagram of her chest revealed enlarged hilum and mediastinal glands and a possible Ghon's focus. Later some adventitiae appeared in the chest, but her general condition improved and she was sent to the country. A few days later she suddenly took a fit with rapid breathing, cyanosis and rigidity of the body. She was unconscious for about ten minutes then slowly recovered. On readmission she was dazed but fairly well and there was no abnormality of her nervous system. She was dismissed home on 16.8.35.

The child was readmitted on 6.11.35 with a history of having been well till ten days before when she took a fainting turn. At 5.15 p.m. on the day of admission she suddenly screamed and became rigid. There were no clonic movements. The pupils were dilated and fixed. The girl was deeply unconscious but recovered completely in about fifteen minutes. The urine contained neither sugar nor acetone.

Blood-Sugar:-

←----------6/11/35----------→ ←7/11/35→

Time.	5.30 p.m.	6 p.m.	6.30 p.m.	7 p.m.	9 p.m.	12 mid-night	9 a.m.
Blood-Sugar. mgm.%	86.9	87.9	71.9	102.0	78.1	94.7	74.0
No. of hours after convulsion.	$\frac{1}{4}$	$\frac{3}{4}$	$1\frac{1}{4}$	$1\frac{3}{4}$	$3\frac{3}{4}$	$6\frac{3}{4}$	$15\frac{3}{4}$

The next morning the child seemed perfectly well and nothing abnormal could be detected in the nervous system. A akiagram of the skull showed no abnormality.

15.11.35. The child was sent to the country once more. She had no more fits and remained well.

17.1.36. Dismissed well.

Case 54.

T.H., a boy aet. $1^{2}/12$ years. Admitted 31.5.37. A healthy baby who throve well till this illness. On the day before admission he developed a cough. In the evening he had two attacks of sudden pallor. At 1.30 a.m. on the day of admission he took a generalised convulsion lasting about an hour.

On admission the patient was found to be a well-nourished child, convulsing and cyanosed. T. 101.4°., P. 140, R. 30. When the convulsion had ceased, examination of the nervous system revealed no abnormality. There was no nuchal rigidity and Kernig's sign was absent. The heart, lungs and abdomen seemed normal. The tongue was furred and dirty and the tonsils much enlarged and red. The urine contained a trace of sugar and a trace of acetone.

Blood-Sugar:-

←----------31/5/37----------→ d1/6/37>

Time.	2.30 a.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	3 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	154.3	91.7	91.7	91.7	78.1	62.9	73.7
No.of hours after convulsion.	1	8	9	10	11	13½	32

1.6.37. Throat swab - negative for B. diphtheriae.

3.6.37. The child was drowsy and refusing to drink, but nothing fresh could be made out on examination.

5.6.37. X-ray of chest - "Areas of consolidation both lungs: ++ hilum shadows, large mediastinal glands."

The Mantoux tuberculin skin test gave a positive reaction.

7.6.37. The child was very drowsy and had nuchal rigidity. Lumbar puncture was done and 30 c.c. clear fluid removed. This contained 73 cells per c.mm., of which sixty-nine per cent. were lymphocytes. The protein was increased. No tubercle bacilli were found in the pellicle which formed.

The child's condition gradually deteriorated. Repeated examination of the cerebro-spinal fluid failed to discover tubercle bacilli.

19.6.37. The child died at 11.40 a.m. Permission for an autopsy was refused.

Case 55.

M.McA., a boy aet. 9/52 year. Admitted 29.3.36. A full-time healthy baby who throve fairly well on whole milk and sugar.

The child was well till 8 a.m. on the day of admission when he began to have twitching of the limbs which continued for about three hours. There was no vomiting and a normal stool was passed.

On examination the patient was found to be a fairly well-nourished child in a generalised convulsion. T. 99°F., P. 130, R. 40. There was marked cyanosis and dyspnoea and the pupils were dilated and fixed. It was impossible to elicit any superficial or deep reflexes. Nothing abnormal was detected in the heart, lungs or abdomen. During examination the child had two very loose, watery, yellow stools. Lumbar puncture revealed a normal cerebro-spinal fluid. The urine was not obtained.

Blood-Sugar:-

Time.	12.30 p.m.	2.45 p.m.
Blood-Sugar. mgm.%	40.1	23.9
No. of hours after convulsion.	$4\frac{1}{2}$	$6\frac{3}{4}$

Blood N.P.N. 21.6 mgm. per cent.

Blood Cholesterol 120.7 mgm. per cent.

Serum Calcium 11.3 mgm. per cent.

Serum Phosphorus 4.5 mgm. per cent.

The child died at 3.15 p.m. At autopsy he was found to have acute gastro-enteritis and terminal broncho-pneumonia.

Case 56.

M.M., a girl aet. $8^{6}/12$ years. Admitted 4.1.36. She was a healthy girl who was noticed to be puffy about the face eight days before admission. Five days later the oedema became generalised and there was vomiting, oliguria and anorexia. The urine was dark brown in colour.

On admission the patient was found to be a fairly well-nourished girl with marked oedema of the face, hands, feet and legs. Nothing abnormal was detected in the various systems. The urine contained albumin and blood. A few days after admission she developed ascites. The red blood cells disappeared from the urine within a few days but albumin persisted in large amounts. The oedema and ascites persisted unchanged for several months in spite of various lines of treatment.

1.6.36. The child took a convulsion at 3.45 p.m. lasting about five minutes.

Blood-Sugar:-

Time.	4 p.m.	4.15 p.m.	4.30 p.m.	5 p.m.	11.30 p.m.	9.30 a.m.	10.30 a.m.
Blood-Sugar. mgm.%	76.3	78.1	88.5	94.3	133.3	63.7	105.2
No.of hours after convulsion.	$\frac{1}{4}$	$\frac{1}{2}$	$\frac{3}{4}$	$1\frac{1}{4}$	$7\frac{3}{4}$	$17\frac{3}{4}$	$18\frac{3}{4}$

The urine was not obtained.

The child remained unconscious for several days after the convulsion.

9.6.37. Died. Permission for an autopsy was refused.

Case 57.

E.W., a girl aet. 4/12 year. Admitted 8.11.35. She was a healthy child till two weeks before admission when she became listless and vomited occasionally. Later she became cross and fevered and two days before admission she had three convulsions.

On admission the baby was seen to be acutely ill. T. 99°F., P. 130, R. 50. The fontanelle was bulging and there was marked nuchal rigidity. The pupils were equal but fixed. The knee-jerks were present but the abdominal reflexes were not elicited. The plantar responses were extensor. Nothing abnormal was detected in the heart, lungs or abdomen. Lumbar puncture revealed a clear fluid under increased pressure. The Pandy test was strongly positive and there were 88 cells per c.mm. Tubercle bacilli were found in the pellicle.

10.11.35. The child had a short convulsion at 10.25 a.m.

Blood-Sugar:-

Time.	10.30 a.m.	11 a.m.	11.30 a.m.	1 p.m.	2 p.m.
Blood-Sugar. mgm.%	96.1	98.0	90.9	97.1	90.0
No.of hours after convulsion.	5'	35'	1hr.5'	2 hrs. 35'	3 hrs. 35'

The urine was not obtained.

The child became unconscious and had frequent convulsions during the next few days.

17.11.35. Died. At autopsy there was found miliary tuberculosis with tuberculous meningitis.

Case 58.

J.S., a girl aet. $6^{4}/12$ years. Admitted 8.1.37. A full-time, healthy child. Throve well and developed normally. Measles at 2 years. Bronchitis at $6^{1}/12$ years.

The child had always had a winter cough and this had become rather worse in the last few months. One week before admission she became drowsy and irritable and developed a sore throat and headache, but these appeared to clear up within a few days. On the day before admission the headache recurred and next day she took a generalised convulsion which lasted for about ten minutes.

On admission she was found to be a rather thin child acutely ill, drowsy and irritable. T. 101°F., P. 132, R. 28. There was some rigidity of the back and limbs and a slight Kernig's sign on the left side. The pupils were equal and reacted sluggishly to light. The abdominal reflexes and knee-jerks could not be elicited and the plantar responses were doubtful. There was nothing abnormal to be made out in examination of the chest, heart, or abdomen. Lumbar puncture revealed a cerebro-spinal fluid containing 50 cells per c.mm. The urine contained a large amount of acetone and a deposit of amorphous urates.

11.1.37. The child had a generalised convulsion lasting about five minutes.

Blood-Sugar:-

Time.	10.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	3.30 p.m.	4.30 p.m.
Blood-Sugar. mgm.%	106.4	96.1	96.1	82.6	120.5	109.9
No.of hours after convulsion.	5	6	7	9	10	11

The urine contained acetone but no sugar.

The child was obviously gravely ill and for the first time an area of dulness and tubularity was found in the left chest.

X-ray of the chest - Consolidation left lung.

13.1.37. By now the signs of consolidation were classical.

16.1.37. The child died at 7.35 p.m.

Case 59.

M.B., a girl aet. $2^{5}/12$ years. Admitted 13.1.37. A full-time, healthy child. Throve well and developed normally. At $11/12$ year the child had a convulsion lasting fifteen minutes.

She was well till six months before admission when she took a convulsion which lasted six hours. She was unable to speak for four weeks thereafter and the right arm and leg were completely paralysed for ten days. In about two months she could walk well.

On the day before admission she was noticed to be listless and heavy, and about 5.15 p.m. she struck her head against the fireguard while playing and appeared to faint. There were no convulsive movements of the limbs and she was quite well again in a few minutes. Next morning she seemed fairly well and ate her breakfast. At 1.45 p.m., however, she took a convulsion, at first localised to the right hand and later generalised. This lasted till admission 3 hours later.

On examination the patient was found to be a well-nourished girl, drowsy and ill. T. 100.2°F., P. 138, R. 36. There was no nuchal rigidity or Kernig's sign and no paresis of limbs. The knee-jerks were active and the plantar responses flexor, while the abdominal reflexes could not be elicited. The pupils were equal and active. Nothing abnormal was observed in the heart, lungs or abdomen. The urine at 5 p.m. on 13.1.37 contained abundant sugar and a moderate amount of acetone. At 8.15 p.m. the urine contained less sugar but more acetone.

Blood-Sugar:-

Time.	4.45 p.m.	5.15 p.m.	5.45 p.m.	6.15 p.m.	6.45 p.m.	7.45 p.m.	8.25 p.m.	8.35 p.m.
Blood-Sugar. mgm.%	312.5	259.7	231.0	188.7	105.2	63.2	77.5	111.1
No.of hours after convulsion.	3	3½	4	4½	5	6	6 hrs. 40'	6 hrs. 50'

At this point 7 gms. of glucose were given intravenously in the form of a twenty per cent. solution in normal saline.

Blood-Sugar. mgm.%	111.1	556.0	400.0	285.7	212.8	181.8	148.1	126.6
	Before	2'	15'	30'	45'	60'	75'	90'

14.1.37

Time.	9.45 a.m.	12.15 p.m.	2.45 p.m.	9.45 a.m.
Blood-Sugar. mgm.%	72.4	67.5	84.0	70.4

The child was very irritable when disturbed and still had slight fever.

15.1.37. The urine contained no sugar but a large amount of acetone.

18.1.37. Sugar tolerance curve.

9 gms. glucose in 90 c.c. water.

	F.	½ hr.	1 hr.	1½hrs	2 hrs.
Blood-Sugar. mgm.%	57.5	99.0	78.1	57.5	55.8

Lumbar puncture revealed a normal cerebro-spinal fluid. The Wassermann reaction of the blood and cerebro-spinal fluid was negative.

20.1.37. Blood-sugar curve after 7 gms. glucose intravenously as on 13.1.37.

	F.	2'	15'	30'	45'	60'	75'	90'
Blood-Sugar. mgm.%	61.7	444.0	333.3	256.6	217.4	166.7	153.8	125.0

The child had no further convulsions and seemed quite well though very cross. It was thought that she might be mentally defective.

22.1.37. Dismissed well.

Case 60.

D.N., a boy aet. 3 days. Admitted 26.3.36. A full-time baby born after a prolonged labour. Very cyanosed but revived fairly well.

The child seemed quite healthy for the first two days, sucking the breast well and having no vomiting. About 7 a.m. on the day of admission, however, he began to have twitching movements of the face and mouth, coming on very frequently and lasting about two minutes. During the attacks he became very blue.

On examination he was found to be a moderately well-nourished infant having occasional convulsive movements of the head and arms. T. 97°F., P. 140, R. 46. There was no evidence of external injury to the head and no paresis of limbs. The pupils were equal and active but the abdominal reflexes and knee-jerks could not be elicited. Nothing abnormal was detected in heart, lungs or abdomen. About 2 c.c. clear cerebro-spinal fluid were withdrawn by lumbar puncture. The globulin was slightly increased but the cell count was 4 c.mm. The urine contained no abnormal constituent.

Blood-Sugar:-

	<27/3/36>	<28/3/36>	<30/3/36>
Time.	10 a.m.	10 a.m.	4 p.m.
Blood-Sugar. mgm.%	25.2	56.5	90.9
No.of hours after convulsion.	27	-	-

27.3.36. The child had to be fed by gavage, but the convulsive movements had stopped.

28.3.36. Dismissed.

Case 61.

A.B., a girl aet. 1 5/12 years. Admitted 19.6.36. A full-time, healthy baby who throve well and developed normally.

Three weeks before admission she developed whooping-cough and later became very listless. Ulcers appeared on the lips and tongue, and a few days before admission she began to have attacks of vomiting. She had four short generalised convulsions on the night before admission.

On admission the patient was found to be a thin child, comatose and cyanosed. T. 103°F., P. 180, R. 40. The respirations were rapid and noisy and the nostrils were blocked by crusted secretions. No area of dulness was made out in the chest but the respiratory murmur was obscured all over the left back by abundant loose râles. The heart was enlarged to the left and there was a soft systolic murmur at the base. The liver was enlarged to four fingers' breadth below the costal margin and the spleen was just palpable. The mouth was very dirty and the tongue coated with thick white sordes. Nothing abnormal was detected in examination of the nervous system. The cerebro-spinal fluid obtained by lumbar puncture was found to be normal.

At 1 a.m. on 20.6.36 the child had two short convulsions. The urine was not obtained.

Time.	.3 a.m.	9.30 a.m.	11.15 a.m.
Blood-Sugar. mgm.%	70.9	30.8	15.1*
No. of hours after convulsion.	2	8½	10¼

* Specimen of blood obtained immediately post-mortem.

The child died at 11.15 a.m. the same day.

At autopsy, both lungs were found to be the seat of diffuse broncho-pneumonic consolidation with foci of suppurative softening. Nothing abnormal was detected in the suprarenals. Both middle ears were filled with pus.

Case 62.

C.McG., a girl aet. $1^{5}/12$ years. A full-time healthy child. Throve well and developed normally. Apart from measles at $10/12$ year she had been very healthy.

At 11 p.m. on the night before admission she was heard to make a gurgling noise and was found lying staring and unconscious. Later she moved her arms and head about a great deal.

On admission she was found to be a rather spare child, drowsy and irritable, but not acutely ill. To. 98°F., P. 120, R. 30. There was no nuchal rigidity and the nervous system presented no abnormality. The radial epiphyses were enlarged and there was a rachitic rosary. Neither Chvostek's nor Trousseau's sign could be elicited. The throat was very red and the tonsils slightly enlarged, but apart from this no abnormality could be detected in examination of the various systems. The urine contained acetone.

Blood-Sugar:-

Time.	10 a.m.	11 a.m.	12 noon.
Blood-Sugar. mgm.%	67.1	60.2	54.0
No.of hours after convulsion.	11	12	13

11.1.37. Serum Calcium 12.5 mgm. per cent.

Serum Phosphorus 3.7 mgm. per cent.

Plasma Phosphatase 9.2 units.

The child had no further convulsions and improved rapidly within the next few days.

15.1.37. Dismissed well.

Case 63.

M.G., a girl aet. 4/12 year. Admitted 19.5.36. A full-time, healthy baby. Breast fed for a short time and then milk, water and sugar.

The child throve well till six weeks before admission when she became irritable and listless. She refused feeds and lost weight, and on the day before admission became definitely drowsy.

18.5.36. At 1 p.m. began to have short generalised convulsions which recurred very frequently till admission at 2.30 p.m. on 19.5.36.

On examination the patient was found to be a small under-nourished child, acutely ill and drowsy. T. 99°F., P. 140, R. 44. There was marked nuchal rigidity and spasticity of all the limbs. Nothing abnormal was detected in the nervous system, heart, lungs or abdomen. The urine contained no abnormal constituent. Lumbar puncture revealed a slightly opalescent fluid which gave a positive Pandy test and had 140 cells per c.mm., of which fifty-five per cent. were lymphocytes and forty-five per cent. polymorphs. No organisms were seen in films.

Blood-Sugar:-

	←----------19/5/36----------→								←-20/5/36---→		
Time.	3 p.m.	4 p.m.	5 p.m.	6 p.m.	7 p.m.	8 p.m.	9 p.m.	12 mid-night	9.30 a.m.	12.30 p.m.	3.30 p.m
Blood-Sugar. mgm.%	88.5	55.8	55.8	66.0	58.5	66.6	65.3	111.1	58.5	71.9	75.2
No.of hours after convulsion.	26	27	28	29	30	31	32	35	44½	47½	50½

20.5.36. X-ray of chest showed "snowstorm" lungs.
X-ray of skull - " ? starting of lambdoid suture."

25.5.36. Died.

At autopsy the child was found to have tuberculosis of the lungs, generalised miliary tuberculosis and tuberculous meningitis.

Case 64.

J.D., a boy aet. 2 years. Admitted 7.4.37. A full-time, healthy baby. Three months before admission he had a convulsion lasting fifteen minutes and was thereafter drowsy for one day. He was noticed to be ailing on the day before admission, but there were no definite symptoms till 11 p.m. when he had a generalised convulsion, most marked on the right side, and lasting about three hours.

On examination he was found to be a well-nourished boy, drowsy and cross when disturbed. T. 97.8°F., P. 120, R. 30. There were small papulo-vesicular spots resembling scabies on the legs and lower trunk. The tonsils were enlarged and red, but the cervical glands were not much enlarged. The pupils were equal and active and the knee-jerks active. The left plantar response was flexor and the right doubtfully extensor. The abdominal and cremasteric reflexes were present. The urine obtained in the early morning contained abundant sugar but no acetone.

Blood-Sugar, etc.:-

Time.	2 a.m.	9.30 a.m.	10 a.m.	11 a.m.
Blood-Sugar. mgm.%	232.6	52.9	41.8	39.7
Lactic Acid. mgm.%	-	-	-	24.3
No.of hrs. after convulsion.	3	10½	11	12

At 11 a.m. the boy was given 6 gms. glucose intravenously in the form of twenty per cent. solution in normal saline.

Blood-Sugar. mgm.%	363.6	138.8	88.5	61.0	32.7	29.8	26.3
Length of time after injection.	2'	15'	30'	45'	60'	75'	90'

The child remained drowsy throughout the day.

Blood-Sugar 4.30 p.m. 66.2 mgm. per cent.

Lactic Acid 4.30 p.m. 15.2 mgm. per cent.

8.4.37. Next morning the child appeared quite well and the nervous system showed nothing abnormal.

Serum Calcium 13.6 mgm. per cent.
Serum Phosphorus 4.1 mgm. per cent.
Plasma Phosphatase 11.7 units.

10.4.37. The child was dismissed because of scarlet fever in the ward.

Case 65.

M.McL., a girl aet. 3/12 year. Admitted 12.9.36. A twin, the other died of pneumonia at 10 weeks. Breast-fed for three weeks and then Ostermilk. The child had never thriven well.

Three weeks before admission she had gastro-enteritis which, however, responded to treatment. She was apparently well except for a cough on the day before admission, but overnight she became ill with frequent panting respirations. At 7 a.m. on the morning of admission she had a generalised convulsion.

On examination she was found to be a small, marantic infant, cyanosed, convulsing and moribund. T. 96.8°F., P. 130, R. 40. The fontanelle was depressed and the skin loose and inelastic. Nothing abnormal was detected in the heart, lungs or nervous system. The abdomen was flaccid and the liver a little enlarged. The urine contained a trace of albumin and a moderate amount of acetone. Lumbar puncture was performed and 10 c.c. clear cerebro-spinal fluid obtained. This contained 36 cells per c.mm., and the globulin was much increased.

Blood-Sugar at 10.30 a.m. = 19.7 mgm. per cent., i.e. 3½ hours after the convulsion.

The child died at 7.35 p.m.

At autopsy there was found irregular, patchy congestion of the small intestine, with oedema of the mucosa. No lesion was noted in the large bowel, suprarenals or pancreas. The kidneys and liver showed marked fatty degeneration.

Case 66.

W.M., a boy aet. 27/52 year. Admitted 13.2.36. A full-time, healthy baby, cyanosed at birth. Throve well. On the night before admission the child was cross and next morning at 11 o'clock he had a generalised convulsion. During the day he had attacks of screaming and at 3 p.m. he had another short fit.

On admission he was found to be a large, fat, healthy-looking baby, not acutely ill. T. 98.4°F., P. 100, R. 40. Nothing abnormal was detected in the examination of heart, lungs, abdomen or nervous system. Neither Trousseau's nor Chvostek's sign could be elicited. There was slight enlargement of the radial epiphyses. The urine contained a trace of albumin and a fair amount of acetone.

Blood-Sugar:-

	←-------13/2/36--------→		←--14/2/36--→
Time.	5.30 p.m.	7.30 p.m.	12 noon.
Blood-Sugar. mgm.%	92.6	104.1	112.3
No.of hours after convulsion.	6½	8½	25

14.2.36. Serum Calcium 6.5 mgm. per cent.
Serum Phosphorus 4.8 mgm. per cent.
Plasma Phosphatase 13.7 units.

15.2.36. Calcium gluconate 4 c.c. intramuscularly.
Adexolin m. 3 t.i.d.

17.2.36. The child was heard to be crowing. Trousseau's sign was positive and Chvostek's facial phenomenon was elicited on the right side. He had no further convulsions.

20.2.36. Dismissed home well.

Case 67.

O.McC., a boy aet. $3^{11}/12$ years. Admitted 18.1.36. A healthy, full-time baby who throve well and developed normally. He had chicken-pox at $1^{6}/12$ years, measles at two years and whooping-cough at $2^{2}/12$ years. For three or four weeks before admission the child was not eating well and vomited occasionally. At 1 p.m. on the day before admission he had a convulsion lasting five minutes. There were seven similar convulsions before midnight.

On admission the patient was found to be a well-developed, well-nourished boy, not acutely ill. T. 99°F., P. 102, R. 20. There were numerous red petechial spots on the trunk and neck. The throat was rather red. Nothing abnormal was detected in the heart, lungs, abdomen or nervous system. The urine contained acetone.

Blood-Sugar:-

	←----------18/1/36----------→									←-19/1/36--→	
Time.	11 a.m.	1 p.m.	1.30 p.m.	2 p.m.	2.30 p.m.	3 p.m.	4 p.m.	5 p.m.	7 p.m.	2 a.m.	11.45 a.m.
Blood-Sugar. mgm.%	81.9	87.7	92.6	90.9	89.2	142.8	70.4	75.2	130.0	80.7	75.7
No. of hours after convulsion.	22	24	$24\frac{1}{2}$	25	$25\frac{1}{2}$	26	27	28	30	37	$46\frac{3}{4}$

On ophthalmoscopic examination both fundi were normal. Lumbar puncture was done under local anaesthesia and 10 c.c. of clear cerebro-spinal fluid removed. This contained 27 cells per c.mm., mainly lymphocytes. Pandy's test was faintly positive.

20.1.36. The boy had several short convulsions in hospital, but apart from these, seemed moderately well. The blood Wassermann reaction was negative.

Serum Calcium 11.5 mgm. per cent.
Serum Phosphorus 3.2 mgm. per cent.
Plasma Phosphatase 10.1 units.

21.1.36. Lumbar puncture repeated: 4 c.c. clear fluid removed under normal pressure. There were 278 cells per c.mm. and Pandy's test was still only faintly positive.

25.1.36. The child developed a bright red scarlatiniform rash on the thighs, buttocks, lower back and elbows. This was diagnosed as German measles and the child was transferred to an infectious diseases hospital, where he had no further convulsions and lumbar puncture was not repeated.

Case 68.

V.C., a boy aet. 6/52 year. Admitted 2.1.36. A full-time, healthy infant, breast-fed till admission.

Five days before admission the child became very constipated and the bowels did not move for three days. After a dose of castor oil the stools became loose and green and on the day of admission were brownish and contained mucus.

On examination the patient was found to be a rather poorly-nourished infant, dehydrated and ill. T. 101°F., P. 140, R. 40. The fontanelle was depressed and there was no nuchal rigidity. Nothing abnormal was noted in the heart, abdomen or nervous system. There were some fine râles at both bases. The urine was not obtained.

4.1.36. Between 12 midnight and 1 a.m. the child had two convulsions and was thereafter drowsy. Lumbar puncture was performed and 4 c.c. of blood-stained cerebro-spinal fluid removed.

Blood-Sugar:-

Time.	9.30 a.m.	11.30 a.m.
Blood-Sugar. mgm.%	18.8	21.0
No.of hours after convulsion.	9½	11½

The child died at 4.30 p.m.

At autopsy patches of irregular congestion were found throughout the small and large intestine. The liver was much enlarged and of a bright yellowish colour with numerous haemorrhagic areas scattered throughout it; this appearance was very suggestive of acute yellow atrophy. The suprarenals and pancreas appeared to be normal. There were numerous small petechial haemorrhages in the disc on the left side of the skull.

Case 69.

D.McR., a boy aet. 3 years. Admitted 21.5.37. He was a full-time, healthy baby who appeared to thrive for the first year. It became evident that he was not developing normally. He never learned to feed himself or walk and was unclean in his habits. One week before admission he had an attack of diarrhoea and vomiting. Three days later he took a convulsion affecting chiefly the left side of the body and this was repeated several times each day till admission.

On examination the child was found to be pale but not acutely ill, and obviously an idiot. T. 99.4°F., P. 120, R. 24. He was quite unable to sit up or walk. The right arm and leg showed aimless movements, while the left arm and leg were rigid and motionless. There was paresis of the left side of the face. The muscle jerks were much exaggerated on the left side. The left plantar response was extensor and the right flexor, while the left abdominal reflex could not be elicited. The pupils were equal and reacted sluggishly to light. Nothing abnormal was detected in the heart, lungs or abdomen.

25.5.37. The child took a convulsion at 8.50 a.m.

Blood-Sugar, etc.:-

Time.	9.30 a.m.	10 a.m.	10.50 a.m.	10.55 a.m.
Blood-Sugar. mgm.%	90.9	90.9	78.1	73.0
Lactic Acid. mgm.%	-	-	-	53.0
No.of hours after convulsion.	40'	1hr.10'	2	2 hrs. 5'

At 10.55 a.m. the child was given 6½ gms. glucose intravenously in a twenty per cent. solution of normal saline.

Blood-Sugar. mgm.%	384.6	210.5	161.2	100.0	88.5	79.4	72.5
No.of minutes after injection.	2'	15'	30'	45'	60'	75'	90'

Case 69 (contd.).

The convulsive movements continued in spite of the intravenous glucose until about 1 p.m.

←----------25/5/37----------→ (26/5/37) (28/5/37)

Time.	1.30 p.m.	2.30 p.m.	5 p.m.	9.30 a.m.	12 noon
Blood-Sugar. mgm.%	66.2	64.1	129.8	106.3	91.7
Lactic Acid. mgm.%	-	-	33.5	-	42.1

The cerebro-spinal fluid obtained by lumbar puncture was quite normal and the Wassermann reaction was negative.

4.6.37. Sugar tolerance curve 12 gms. glucose in 120 c.c. water.

Fasting.	½ hr.	1 hr.	1½ hrs.	2 hrs.
64.9	59.5	75.7	75.7	121.9 mgm. per cent.

10.6.37. Lactic acid 18.0 mgm. per cent.

2.7.37. Dismissed home. I.S.Q.

Case 70.

M.O'H., a girl aet. 10 years. Admitted 19.1.36. She had been a healthy baby and apart from measles and pneumonia at two years had had no serious illnesses. Four days before admission she went off her food and vomited. Two days later she had severe abdominal pain and shortness of breath. At 6 a.m. on the day of admission she had a convulsion lasting ten minutes. During the day she had several similar fits.

On admission she was found to be a well-nourished child with oedema of the face, lumbo-sacral region, ankles and feet. T. 99.6°F., P. 104, R. 28. She was conscious but rather dull. The tonsils were enlarged and septic and the tongue furred and dry. There was an impaired percussion note at the left base and moist râles in this region. Nothing abnormal was detected in the heart or abdomen.

The blood-pressure was 148/120 mm. Hg. The urine contained albumin, blood, red blood corpuscles and casts, but no sugar.

Blood-Sugar:-

<19/1/36> ←----------20/1/36----------→ <21/1/36>

Time.	12 mid-night	1 a.m.	1.30 a.m.	2 a.m.	9.30 a.m.	10 a.m.	10.30 a.m.	11 a.m.	12 noon	1 p.m.	2 p.m.	3 p.m.	4 p.m.	5 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	78.7	69.4	72.4	71.9	69.4	64.1	69.9	64.1	62.5	58.5	62.8	97.1	90.1	85.5	102.0
No.of hours after convulsion.	18	19	19½	20	27½	28	28½	29	30	31	32	33	34	35	51½

The general condition improved rapidly in hospital.

28.1.36. The urine contained occasional red blood cells. B.P. 104/64.

12.2.36. Dismissed home as a chicken-pox contact.

Case 71.

M.B., aet. 9 years. Admitted 3.6.37. A full-time child. Noticed to have a swelling on the right side of the back which was said to be due to a spina bifida. At the age of 10/12 year this was removed, but the right leg has remained paralysed since and a scoliosis developed. The child learned to walk, however, with the aid of crutches. She developed cystitis at an early age.

Five weeks before admission the girl had an attack of tonsillitis which cleared up in a few days.

Two weeks later she developed albuminuria and oedema of the feet, which quickly became generalised and extensive.

At 7.30 a.m. on the day of admission she began to have generalised convulsions which persisted till admission at 10.15 a.m.

On examination she was found to be a deformed and grossly oedematous child, having twitching movements of all the limbs. She was cyanosed and breathing stertorously. There was a well-marked scoliosis, also kyphosis and lordosis, and the right lower limb was much wasted. The heart sounds were of poor quality and the blood-pressure was 150/120 mm. Hg.

Some coarse râles were audible on both sides of the chest and the abdomen showed evidence of free fluid. A specimen of urine obtained by catheterisation was found to be loaded with albumin, but microscopically there were only a few granular casts and no pus cells. Sugar was also present in the urine and a moderate amount of acetone. Cerebro-spinal fluid obtained by cisternal puncture was under normal pressure, contained 44 cells per c.mm. and an increased quantity of proteins as evidenced by a positive Pandy test.

The child died at 11.30 a.m. Permission for an autopsy was not granted.

The blood-sugar at 10.30 a.m. was 256.4 mgm. per cent.

Blood was obtained by cardiac puncture after death and the following estimations were made:-

Non-protein nitrogen - 78.1 mgm. per cent.
Total proteins - 8.6 mgm. per cent.
Non-protein nitrogen - 78.1 mgm. per cent.
Total proteins - 8.6 mgm. per cent.
Chloride - 82.1 ml. N/10 per cent.
Plasma chlorides - 90.1 ml. N/10 per cent.

Case 72.

I.G., a boy aet. $5^{3}/12$ years. Admitted 8.1.37. A full-time, healthy child who throve well and developed normally. Measles at $2^{6}/12$ years. Pneumonia at 4 years. Chickenpox at $4^{3}/12$ years.

On the morning of the day of admission the boy refused his breakfast and about 4 p.m. he took a convulsion which lasted about one hour.

On examination he was found to be a well-nourished child, flushed and restless, but not acutely ill. T. 100.4°F., P. 148, R. 46. Nothing abnormal could be detected in the nervous system, heart, lungs or abdomen. His throat was very red and the tonsils moderately enlarged and inflamed. The urine was not obtained till 9 o'clock the next morning and it contained no sugar but a fair amount of acetone.

Blood-Sugar:-

Time.	10 a.m.	11 a.m.	12 noon
Blood-Sugar. mgm.%	47.1	57.8	54.0
No. of hours after convulsion.	18	19	20

12.1.37. The child was quite well and had had no further convulsions. The tonsils were still large and inflamed. The boy was always very quiet and seemed rather backward.

21.1.37. Sugar tolerance curve after 16 gms. glucose in 160 c.c. water.

	F.	½	1 hr.	1½	2.
Blood-Sugar. mgm.%	65.8	101.0	123.5	133.3	86.3

28.1.37. Tonsils and adenoids removed under ethyl-chloride anaesthesia.

5.2.37. Dismissed home well.

Case 73.

W.McD., a boy aet. 6/12 year. Admitted 15.1.37. A full-time, healthy baby who was breast-fed for three months and then given Nestle's milk, Farola and saps. The right ear had been discharging for three months. At 10 a.m. on 14/1/37 the child fell out of bed and knocked his forehead. At 10 p.m. he had a generalised convulsion which lasted about one minute. During the night he had six similar convulsions.

On admission he was found to be a well-nourished child, not acutely ill. T. 98.6°F., P. 130, R. 36. Nothing abnormal was detected in the examination of heart, lungs, abdomen or nervous system. There was slight enlargement of the radial epiphyses, but no rachitic rosary. Chvostek's sign was not elicited. The urine contained no abnormal constituent.

Serum Calcium 6.5 mgm. per cent.
Serum Phosphorus 3.6 mgm. per cent.
Plasma Phosphatase 20.6 units.

Blood-Sugar:-

Time.	1 p.m.	2 p.m.	3 p.m.	4 p.m.	5 p.m.
Blood-Sugar. mgm.%	75.7	69.9	63.3	90.9	91.7
No.of hours after convulsion.	13	14	15	16	17

The child was given 10 c.c. of ten per cent. calcium gluconate intramuscularly and the convulsions ceased.

Three minims of adexolin were given three times a day.

21.1.37. Dismissed well.

Case 74.

E.D., a girl aet. 6/52 year. Admitted 27.4.37. A full-time, healthy baby. At four days began to take convulsions which persisted intermittently till the ninth day. No cause was found for these and they finally cleared up completely.

The child was thereafter well and thriving till twelve hours before admission when she began to take short fits about every half-hour.

On examination she was found to be a healthy-looking child, not acutely ill. T. 97.6°F., P. 136, R. 42. The fontanelle was neither tense nor depressed and there was no nuchal rigidity. Examination of the heart, lungs and abdomen revealed no abnormality. Neither Chvostek's nor Trousseau's sign could be elicited. The superficial and deep reflexes were physiological. The urine contained nothing abnormal.

Serum Calcium 7.2 mgm. per cent.
Serum Phosphorus 3.9 mgm. per cent.
Plasma Phosphatase 18.2 units.
Lactic Acid 17.0 mgm. per cent.

Blood-Sugar:-

Time.	12.45 p.m.	2.15 p.m.	3.30 p.m.	3.40 p.m.	5 p.m.
Blood-Sugar. mgm.%	54.3	57.5	51.5	50.7	61.7
No. of hours after convulsion.	12	13½	14¾	14 hrs. 55'	16¼

28.4.37. Calcium gluconate 5 c.c. intramuscularly. Chvostek's sign was detected.

29.4.37. No further fits.

30.4.37. Dismissed well.

SUMMARIES OF SURGICAL CASE HISTORIES.

Case 1.

H.S., a girl aet. 4 years. Admitted 23.10.35. At 10 a.m. on the day of admission the child was struck on the head by a falling ladder. She lost consciousness for a short time and was thereafter dazed.

On examination the child was found to be shocked and drowsy. T. 97°F., P. 140, R. 36. There was a large diffuse haematoma in the left parietal region, but no evidence of any pressure symptoms. An X-ray plate of the skull showed multiple linear fractures of the vault. The urine contained sugar but no acetone.

Blood-Sugar:-

←----------23/10/35----------→ ←----24/10/35----→

Time.	1 p.m.	2 p.m.	2.30 p.m.	3 p.m.	3.30 p.m.	5 p.m.	8 p.m.	12 mid-night	9 a.m.	3 p.m.	4 p.m.	5 p.m.
Blood-Sugar. mgm.%	250.0	139.8	136.9	125.7	108.1	80.3	72.4	65.3	60.9	78.1	75.7	62.4
No. of hours after injury.	3	4	4½	5	5½	7	10	14	23	29	30	31

The child made a good recovery and was dismissed well on 20.11.35.

Case 2.

G.C., a boy aet. 6 years. Admitted 25.5.36. The child fell over a bannister down one flight of stairs. He was unconscious when picked up, and vomited several times. There was no bleeding from the nose or ears.

On admission the child was found to be dazed but only slightly shocked. T. 98.8°F., P. 100, R. 20. There was a swelling over the occiput and a skiagram showed a fracture in this region. There was no abnormality of the nervous system. The urine contained neither sugar nor acetone.

Case 2 (contd.).

Blood-Sugar:-

	←25/5/36→					←26/5/37→			
Time.	4.30 p.m.	5.30 p.m.	7.30 p.m.	8.30 p.m.	9.30 p.m.	9.30 a.m.	2.30 p.m.	3.30 p.m.	4.30 p.m.
Blood-Sugar. mgm.%	86.9	80.6	80.6	72.9	94.5	56.5	45.8	107.5	104.1
No.of hours after Injury.	3½	4½	6½	7½	8½	20½	25½	26½	27½

The boy's condition rapidly improved and he was dismissed well on 17.6.36.

Case 3.

W.O., a boy aet. 6 years. Admitted 20.5.36. At 6 p.m. on the day of admission he fell over a bannister a distance of nine feet. He did not lose consciousness.

On examination the boy was found to be pale and shocked. T. 97°F., P. 104, R. 26. There was a large haematoma in the occipital region and an X-ray plate showed fracture of the occipital bone. The pupils were equal and active and all reflexes were physiological. The urine contained neither sugar nor acetone.

Blood-Sugar:-

	←20/5/36→	←21/5/36→						←22/5/36→			
Time.	9 p.m.	10 p.m.	9.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	4.30 p.m.	5.30 p.m.	9.30 a.m.	11.30 a.m.	3.30 p.m.	5.30 p.m.
Blood-Sugar. mgm.%	106.4	102.0	89.3	58.8	77.5	53.8	57.5	57.5	48.8	45.5	64.9	57.5
No.of hours after injury.	3	4	15½	17½	18½	20½	22½	23½	39½	41½	45½	47½

The boy made good progress and was dismissed well on 9.6.36.

Case 4.

J.G., a boy aet. 3 years. Admitted 28.9.36. About noon on the day of admission the boy fell from a wall and was picked up unconscious. There was no haemorrhage and no vomiting.

On admission the boy was found to be in a state of severe shock and semi-conscious. T. 96.2°F., P. 150, R. 26. There was a depressed fracture of the skull in the right fronto-parietal region and excoriation of the skin on the right side of the face. The pupils were equal and reacted sluggishly to light. The left leg was spastic and the left knee-jerk much exaggerated. Blood-sugar estimations were not made till the following day and urine on the morning of 29.9.36 contained no sugar but a large amount of acetone.

←------29/9/36----→ ←---30/9/36----→ <1/10/36>

Time.	12 noon	3 p.m.	4 p.m.	5 p.m.	9.30 a.m.	12.30 p.m.	3.30 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	54.3	39.7	44.6	44.4	40.0	43.6	42.7	55.2
No.of hours after injury.	24	27	28	29	45½	48½	51½	69½

The boy never regained consciousness and died at 10.20 a.m. on 2.10.36.

At autopsy there was found a depressed fracture of the anterior part of the right parietal bone and a linear fracture running down from this and involving the temporal and petrous bones. The brain substance in relation to the fracture showed contusion.

Case 5.

R.S., a boy aet. 5 5/12 years. Admitted 19.9.36. The child was knocked down by a motor car.

On admission he was extremely shocked. T. 99.6°F. P. 128, R. 32. There was a large haematoma on the forehead and a wound in the occipital region. There was bleeding from the nose. The urine was not obtained. The blood-sugar at 3 p.m. on 21.9.36 was 61.7 mgm. per cent. Three days later the child was still unconscious and the blood-sugar at 5p.m. was 66.6 mgm. per cent.

At 6 p.m. on 24.9.36 the child had a convulsion, after which the temperature rose to 104°F., and the pulse to 164. Urine obtained on the morning of 25.9.36 contained sugar and a trace of acetone.

25.9.36. Blood-Sugar:- 1 p.m. 112.3 mgm.%. 4 p.m. 112.3 mgm.%
The child died at 11.50 p.m. At autopsy he was found to have a fracture of the occiput and meningeal haemorrhage.

Case 6.

M.A., a girl aet. 7 years. Admitted 15.5.37. At 2.30 p.m. on the day of admission the child was knocked down by a motor-car and received head injuries; when picked up she was unconscious.

On admission the girl was found to be cyanosed and very shocked. T. 97.6°F., P. 110, R. 26. There was an irregular, lacerated wound in the right frontal region and haemorrhage from the nose. The pupils were equal, the right reacting to light, but the left fixed. There was superficial bruising of the abdominal wall. The urine contained sugar.

The blood-sugar at 7.30 p.m. was 208.3 mgm. per cent.

16.5.37. Under a local anaesthetic the edges of the scalp wound were excised and a linear fracture of the frontal bone exposed. The wound was sutured. There was marked ecchymosis of the right eyelid. The girl was still unconscious and gravely ill.

300 c.c. ten per cent. glucose in saline intravenously.

17.5.37. Blood-Sugar at 3 p.m. = 67.5 mgm. per cent.

18.5.37. The child died at 1 a.m.

Case 7.

W.McI., a boy aet. 12 years. Admitted 8.12.36. At 9 p.m. on the night of admission a chimney-can fell on the boy's head. He lost consciousness but regained it before admission.

On admission the boy was found to be conscious but drowsy and shocked. T. 97°F., P. 120, R. 24. There was bleeding from the nose and a large fluctuant haematoma on the forehead. The nervous system seemed normal. The urine contained sugar but no acetone.

Blood-Sugar:-

‹8/12/36› ‹----9/12/36-----› ‹10/12/36›

Time.	9.30 p.m.	12 noon	2 p.m.	4 p.m.	12 noon.
Blood-Sugar. mgm.%	167.1	135.1	86.9	69.9	50.5
No.of hours after injury.	½	15	17	19	39

A skiagram of the skull showed a depressed fracture of the frontal bone. The child made a good recovery and was dismissed well on 30/12/36.

Case 8.

G.K., a boy aet. 5 years. Admitted 23.2.37. At noon on the day of admission the boy was knocked down by a tramcar but was not rendered unconscious.

On admission he was found to be slightly shocked. T. 97.6°F., P. 120, R. 22. There was a large haematoma of the forehead and many abrasions. The nose was much swollen and bleeding. The nervous system seemed normal. The urine contained nothing abnormal.

Blood-Sugar:-

	←------23/2/37--------→			←-24/2/37-→
Time.	3 p.m.	4 p.m.	5 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	89.5	65.8	79.4	51.3
No.of hours after injury.	3	4	5	21½

An X-ray plate of the skull showed a fracture of the nasal bones.

The child made a good recovery and was dismissed well on 2.3.37.

Case 9.

T.G., a boy aet. 5/12 year. Admitted 29.10.36. The child fell from his sister's arms and struck the corner of the table about 10.45 a.m. on the day of admission.

On examination the child was found to be cross, but not apparently shocked. T. 97°F., P. 120, R. 24. There was a depression in the right parietal region and an X-ray plate showed a depressed fracture. There was no evidence of intracranial disturbance. The urine was not obtained.

The blood-sugar at 2.45 p.m., i.e. four hours after the injury, was 142.8 mgm. per cent. Just after this the child was given a general anaesthetic and the depressed piece of bone elevated into position.

Convalescence was uneventful and the child was dismissed well on 20.11.36.

Case 10.

M.A., a girl aet. 11 years. Admitted 24.10.35. The girl was knocked down by a tramcar at 4 p.m. She did not lose consciousness but complained of feeling slightly dizzy.

On examination there was found a slight haematoma on the left side of the skull. The child was not shocked. T. 98°F., P. 96, R. 20. The urine contained nothing abnormal.

Blood-Sugar:-

	←--------24/10/35-------→				←-----25/10/35----------→					
Time.	4.30 p.m.	5 p.m.	5.30 p.m.	7 p.m.	12 mid-night	9 a.m.	11 a.m.	1 p.m.	3 p.m.	5 p.m.
Blood-Sugar. mgm.%	84.0	67.1	78.1	80.0	75.7	65.3	71.6	96.1	63.6	84.0
No.of hours after injury.	½	1	1½	3	8	17	19	21	23	35

The girl made a good recovery and was dismissed well on 26.10.35.

Case 11.

J.C., a boy aet. 12 years. Admitted 3.12.36. About noon the boy fell from a lorry and was picked up unconscious.

On examination he was dazed and irritable and answered questions slowly. T. 96.2°F., P. 90, R. 22. The pupils were equal and reacted to light and all the reflexes were physiological. There was a lump on the left parietal region, but a skiagram of the skull showed no fracture. The urine contained nothing abnormal.

Blood-Sugar:-

	←-------3.12.36-------→			(4.12.36)
Time.	2.45 p.m.	3.45 p.m.	4.45 p.m.	9.45 a.m.
Blood-Sugar. mgm.%	82.6	82.6	81.3	51.2
No.of hours after injury.	2¾	3¾	4¾	21¾

The child recovered rapidly and was allowed home on 11.12.36.

Case 12.

H.C., a boy aet. 6 years. Admitted 9.2.37. At 3.15 p.m. the boy was knocked down by a motor car.

On admission he was found to be shocked and drowsy. T. 97.8°F., P. 92, R. 26. There were superficial wounds all over the head, face, and neck, and abrasions on the left arm. There was no bleeding from the nose or ears. The pupils were equal and reacted to light. The urine contained nothing abnormal.

Blood-Sugar:-

	←----9/2/37---→		←-10/2/37-→
Time.	4 p.m.	5 p.m.	12 noon.
Blood-Sugar. mgm.%	156.2	112.3	65.7
No.of hours after injury.	¾	1¾	20¾

A skiagram of the skull showed no fracture.

The facial cuts were sutured and the child made a good recovery and was dismissed well on 16.2.37.

Case 13.

J.G., a boy aet. 11 years. Admitted 1.6.36. At 10.30 p.m. on the night of admission the boy fell over a bannister on to his head. He was unconscious when picked up, but improved soon, though he vomited several times. There was bleeding from the nose.

On examination the boy was found to be shocked but conscious. T. 96°F., P. 120, R. 24. There was an abrasion in the right frontal region. The pupils were equal and active and there was no paralysis. The urine contained nothing abnormal.

Blood-Sugar:-

	←----------2/6/36----------→								←-3/6/36-→	
Time.	9.30 a.m.	11.30 a.m.	12.30 p.m.	3.30 p.m.	4.30 p.m.	5.30 p.m.	7.30 p.m.	9.30 p.m.	9.30 a.m.	11.30 a.m.
Blood-Sugar. mgm.%	87.7	84.0	81.3	76.9	67.1	61.7	75.2	62.9	75.7	75.7
No.of hours after injury.	11	13	14	17	18	19	21	23	35	37

An X-ray plate of the skull showed no fracture and the boy was dismissed well on 24.6.36.

Case 14.

A.McD., a girl aet. 5 years. Admitted 25.2.36. At 12.30 noon on the day of admission the child was knocked down by a motor 'bus and her head struck the kerb. She did not lose consciousness but vomited and became very drowsy.

On examination the patient was found to be markedly shocked and drowsy. T. 97.6°F., P. 122, R. 28. There was bruising of the right eye and a contusion on the left side of the head posteriorly. The urine contained neither sugar nor acetone.

Blood-Sugar:-

←----25/2/36------→					←--------26/2/36----------→						←27th→
Time.	2.30 p.m.	3.30 p.m.	4.30 p.m.	5.30 p.m.	9.30 a.m.	10.30 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	4.30 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	149.2	70.9	70.4	70.4	48.4	42.7	59.5	60.2	63.7	68.5	61.7
No.of hours after injury	2	3	4	5	21	22	23	24	26	28	45

A skiagram of the skull showed no fracture.
The child made a rapid recovery and was dismissed well on 28.2.36.

Case 15.

J.G., a boy aet. 7 years. Admitted 27.5.36. At 10 p.m. in the night of admission the boy fell from a washhouse roof, a distance of about 14 feet. He was unconscious for a few minutes but there was no haemorrhage or vomiting.

On admission the boy was found to be slightly shocked but conscious. T. 97.8°F., P. 114, R. 24. He had a small wound on the left upper lip and abrasions of the left eyebrow and chin. The pupils were equal and active and there was no paralysis. The urine contained nothing abnormal.

Blood-Sugar:-

←----------28/5/36----------→						←29/5/36→
Time.	10 a.m.	11 a.m.	12 noon	3 p.m.	4 p.m.	5 p.m.	10 a.m.
Blood-Sugar. mgm.%	100.0	81.3	70.9	89.3	92.6	81.3	69.2
No.of hours after injury.	12	13	14	17	18	19	36

A skiagram of the skull showed no fracture. The boy recovered rapidly and was dismissed well on 1.6.36.

Case 16.

A.M., a boy aet. 8 years. Admitted 13.5.37. At 3 p.m. on the day of admission the boy was knocked down by a motor lorry but did not lose consciousness.

On admission the boy was found to be shocked but conscious. T. 97°F., P. 104, R. 20. There was a haematoma in the right frontal region and haemorrhage from the right nostril. The right pupil was larger than the left, but both reacted to light. The other reflexes were physiological. The urine contained nothing abnormal.

Blood-Sugar:-

←----------14/5/37----------→

Time.	11.30 a.m.	12.30 p.m.	2.30 p.m.
Blood-Sugar. mgm.%	74.7	71.9	67.1
No.of hours after injury.	20½	21½	23½

A skiagram of the skull showed no fracture.
The child made a good recovery and was dismissed well on 1.6.37.

Case 17.

C.P., a boy aet. 4 years. Admitted 28.10.35. At 1.30 p.m. the child fell and injured his right leg.

On examination the child was found to be considerably shocked. T. 98.2°F., P. 120, R. 24. The right thigh was swollen, tender and painful and there was an obvious fracture of the femur at the junction of the middle and lower thirds. The limb was put up in extension.

Blood-Sugar:-

←---------28/10/35----------→ ←-------29/10/35-------→

Time.	3.30 p.m.	4 p.m.	4.30 p.m.	5 p.m.	5.30 p.m.	8 p.m.	12mid-night	9.30 a.m.	11 a.m.	12 noon	1 p.m.	4 p.m.
Blood-Sugar.	270.2	177.0	156.2	153.0	130.7	142.8	116.3	106.3	75.2	83.3	90.9	101.5
No.of hours after injury	2	2½	3	3½	4	6½	10½	20	21½	22½	23½	26½

The urine was not obtained.
The boy made a good recovery and was dismissed well on 4.12.35.

Case 18.

F.T., a boy aet. 10 years. Admitted 30.10.35. At 1 p.m. a wooden barricade fell on the boy's right leg, rendering him unable to stand or walk.

On examination there was found to be a fracture of the right tibia in the middle third. The boy exhibited no signs of shock. T. 98°F., P. 90, R. 20. The urine contained nothing abnormal.

Blood-Sugar:-

	←30/10/35→							←31/10/35→		
Time.	2.30 p.m.	3 p.m.	3.30 p.m.	4 p.m.	4.30 p.m.	5.30 p.m.	10.30 p.m.	1 a.m.	9.30 a.m.	10.30 a.m.
Blood-Sugar. mgm.%	100.0	76.9	74.6	86.2	84.0	92.5	73.5	62.8	82.6	69.9
No.of hours after injury.	1½	2	2½	3	3½	4½	9½	12	20½	21½

Plaster of Paris was applied to the right leg and the child was dismissed on 31/10/35.

Case 19.

T.M., a boy aet. 6 years. Admitted 16.11.36. The boy was knocked down by a tramcar about noon. He sustained an injury to the right leg and a bruise above the right eye, but did not lose consciousness.

On admission there was found a fracture of the right femur at the junction of the upper and middle thirds. The boy was conscious and not shocked. T. 98.8°F., P. 120, R. 20. The limb was put up in extension. Nothing abnormal was observed in the urine.

Blood-Sugar:-

	←10/11/36→	←17/11/36→
Time.	4 p.m.	9.30 a.m.
Blood-Sugar. mgm.%	107.5	84.0
No.of hours after injury.	4	21½

The boy made an uneventful recovery and was dismissed well on 18.12.36.

Case 20.

T.K., a boy aet. $3^{11}/12$ years. Admitted 20.10.35. At 6.30 p.m. on the day of admission the child was scalded by a kettle of boiling water.

On examination the child was found to be considerably shocked. T. 97.4°F., P. 120, R. 28. The scald involved the left buttock and thigh and to a lesser extent the right thigh. It was treated the same evening with tannic acid and flavine. The urine was not obtained.

Blood-Sugar:-

Time.	10.30 a.m.	11 a.m.	11.30 a.m.	12 noon	1 p.m.	2 p.m.
Blood-Sugar. mgm.%	35.8	62.9	64.1	117.0	130.6	142.8
No.of hours after scald.	16	16½	17	17½	18½	19½

The child remained very ill for several days, but thereafter made a good though slow recovery and was dismissed well on 5.2.36.

Case 21.

G.D., a boy aet. $1^{2}/12$ years. Admitted 18.5.37. At 1.30 p.m. the child was scalded with boiling water.

On admission the child was found to be shocked. T. 97.4°F., P. 148, R. 36. There was an extensive scald of the first and second degree affecting the left arm and forearm and the outer aspect of the left thigh. The urine was not examined.

Blood-Sugar:-

	18/5/37	19/5/37
Time.	7.30 p.m.	12 noon.
Blood-Sugar. mgm.%	88.5	107.5
No.of hours after scald.	6	22½

A tannic acid and flavine dressing was applied.
The child developed broncho-pneumonia and died at 12 noon on 21.5.37.

Case 22.

R.P., a boy aet. 8 years. Admitted 31.3.37. At 3 p.m. the boy was scalded severely on the left foot.

On admission he was found to be considerably shocked. T. 97.6°F., P. 128, R. 24. Nothing abnormal was discovered in the urine.

The blood-sugar at 4.30 p.m. was 169.5 mgm. per cent. Immediately after this, an anaesthetic was administered for purposes of treatment and no further estimation of the blood-sugar was made. The patient is still in hospital.

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