IMPORTANT PROCEDURES IN THE CONSERVATIVE TREATMENT OF ECLAMPSIA

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THE good results given by the widely separated methods of the conservative treatment of eclampsia, that is, the use of elimination by Tweedy and the use of sedatives by Stroganoff, indicate that the maternal organism will recover if the disease has not lasted too long and the damage has not been too great. The favorable and unfavorable reports of various clinics based on one or the other methods suggest that each case must be individualized and that both experience and judgment are necessary. Furthermore, obstetricians have recognized as a result of the high maternal mortality that mild cases if treated radically will show an increase in the mortality rate, and severe cases treated conservatively will continue to show a definite mortality if early death of the fetus does not take place or delivery occur. As a result, many have taken the good points of each treatment and by combining them and determining the type of case are beginning to get still more favorable results. They have also realized that certain severe cases must be handled by early delivery. The difficulty still persists, however, in that it is usually impossible to decide as to the severity of the case before too much damage may have occurred.

In our studies of the toxemias of pregnancy we have found that there are a mass of results reported, but that there is no uniformity. Therefore, from the beginning we have been collecting as much data on each case as time permitted and have constantly increased the variety of examination as our accumulated material gave fresh ideas or required additional confirmation. Our results indicate the need for certain specific treatment the use of which enables us to give a prognosis early enough to be of value. Much of this data is not included in this paper but will furnish the basis of an additional report.

Our treatment can be summarized as follows:

1. $MgSO_4$ in 25 per cent solution is given intramuscularly to control convulsions. On admission we inject 10 c.c. and give 5 c.c. after each convulsion until controlled. Our average amount over a period of five years has been 19 c.c. with a maximum of 50 c.c. in only one case. In coma no $MgSO_4$ is used, for we believe that its only action, if given intramuscularly, is as a sedative. If given intravenously it does decrease intracranial pressure, but its depressant action on the respiration and the heart are so marked that they contraindicate its use. Dorsett has given intramuscularly from 15 c.c. to 200 c.c. depending on the severity of the case. In three cases he gave 200 c.c. per twenty-four hours. We attribute our success with the

to 100 per cent, and if the patient is edematous, it actually exceeds the intake. It is therefore evident that the diuresis is a physiologic phenomenon occurring only after delivery or death of the fetus and since its appearance is so intimately associated with clinical improvement, we consider its production of primary importance. Tables II and III are representative of typical changes in the blood and urine in eclampsia, especially after delivery; and Table IV of changes both before and after. In a number of cases we have succeeded in lowering the serum

TABLE I

NUMBER		DATE	HEMOGLOBIN	CELL VOLUME PER CENT	SERUM PROTEIN PER CENT	WEIGHT KILO.
F. F.	1*	7/18/27	113	46.4	6	77.7
11738	2	7/19/27	102	34	3.82	
	3	8/17/27	81	40	6.35	62.3
E. D.	1	9/ 6/27	86	39	5.92	
174	2	9/ 6/27	98	41	5.56	
	3	9/20/27	76	35	5.8	
E. B.	1	10/16/27	91	43	5.03	70.2
440	2	10/21/27	65	30.8	4.42	
	3	10/25/27	74	32	5.12	
L. B.	1	11/24/27	83	40	6.81	
715	2	11/29/27	66	35	6	
	3	12/ 7/27	70	36	5.85	
N. C.	1	2/16/28	101	42	8.42	79
1332	2	2/17/28	72	28.8	4.93	
	3	2/29/28	82	30	7.5	67
M. W.	1	4/10/28	111	42	6.4	166
1760	2	4/12/28	58	27	4.43	
	3	5/ 3/28	83	36	7.23	116
T. H.	1	5/15/28	91	37	6.54	
1992	2	5/15/28	70	28	5.17	
	3	5/24/28	59	26	6.4	
McC.	1	5/24/28	100	44	6.42	
2062	3	6/ 1/28	75	35	6.45	
W. P.	1	8/12/28	63	34	5.13	71.2
2734	a	8/18/28	105	55	5.6	
	2	8/20/28	66	32	4.28	
	3	9/ 1/28	85	34	6.45	60
E. A.	1	10/18/28	85	45	5.55	75
3380	b	10/19/28	119	49	5.52	
	2	10/20/28	105	41	4.15	
	3	11/ 5/28	98	35	6.26	
W. W.	1	10/25/28		49	5.83	65
3437	2	10/30/28		32	4.8	58
	3	11/ 5/28		50	6.88	56.8
F. S.	1	12/ 6/28	117	42	6.43	69.6
3779	2	12/ 8/28	90	36	5.45	
	3	12/10/28	111	38	6.73	
L. W.	1	1/16/29	121	42	5.86	72
4129	2	1/21/29	83	31	4.84	
	3	2/ 8/29	100	40	6.36	49.5
M. B.	1	4/ 2/29	105	40	7.47	69
4640	2	4/ 6/29	77	24	5.64	62
	3	4/22/29	64	27	6.92	

*1. Admission; 2. Greatest Dilution; 3. Discharge; a. Convulsion; b. Aftertreatment.

protein concentration before delivery but we have never succeeded in producing the typical diuresis of 4000 c.c. or more except after delivery or death of the fetus. Table V demonstrates that identical changes can occur before delivery. In this case no fetal movements, were noticed by the mother after July 20, and it is a fair assumption that the fetus died on that day. This is supported by the fact that the urine increased steadily from 400 c.c. on the twentieth, to 2400 c.c. on the twenty-first, and reached a maximum of 6600 c.c. on the twenty-third, and then slowly dropped. No diuresis occurred after delivery on the twenty-seventh.

Our treatment differs from that described by other clinics in that we use large amounts of hypertonic glucose solution and it is to this that we ascribe not only our favorable results but also our ability to control the convulsion with small amounts of $MgSO_4$. Hypertonic glucose solution injected intravenously reduces intracranial pressure, which is usually markedly increased in eclampsia. (Zangemeister, Thies.) Hypertonic salt solution was first used by neurologic surgeons to lower the intracranial pressure but was discarded in favor of glucose because the latter is just as efficient, has no terminal increase in intracranial pressure as NaCl or Ringers has (Peet, Weed and McKibben, and Sachs and Belcher), and can be repeated more frequently because its end-products are CO_2 and water. A 1 per cent solution of $MgSO_4$ is recommended for intravenous injection in nephritic uremia to control the convulsions. It has a dehydrating effect on the brain (Blackfan). We attempted this in one case but stopped the injection because of the effect on the respiration.

It is the general belief that glucose solution if injected intravenously is burned, stored as glycogen and polymerized (Sansum and Woodyatt, and Erlanger and Woodyatt), and if the amount is greater than can be removed by these mechanisms, the excess is excreted in the urine, resulting in a polyuria. Therefore, our purpose was to give enough glucose to produce a glycosuria, thus hoping to initiate a diuresis. We found that in the normal individual 500 c.c. of a 20 per cent solution of glucose (100 gm.) if given over a period of sixty to ninety minutes will not produce a glycosuria; but if given in thirty to fifty minutes will result in 10 to 30 gm. being excreted in the urine. If 200 gm. of glucose are given in thirty to fifty minutes, from 70 to 100 or more grams will be excreted in the urine. We give the eclamptic patient 1000 c.c. of a 20 per cent glucose solution (200 gm.) intravenously over a period of thirty to fifty minutes, two, three, and sometimes four times daily. Twenty-four-hour urine examinations have shown that comparatively little or no glucose is excreted in the urine. In Table VI, we have tabulated those pateints in whom we have twenty-four-hour urines and in only three cases was more than 100 gm. of glucose excreted in the urine per day. Two patients, Cases 3437 and 3380, received injections of

800 c.c. of a 30 per cent instead of 1000 c.c. of a 20 per cent solution, and the excessive glycosuria is apparently due to too great a strain on the tolerance which is apparently approximately 200 gm. We have decreased the amount of 30 per cent to 700 c.c., for although 30 per cent produces a more marked diuresis than 20 per cent, more sugar is excreted in the urine and apparently less toxic material excreted; for it seemed to us that these two cases did not respond as well to injections of 30 per cent as they did to 20 per cent. The glycosuria in the majority of the cases varied from none up to 50 gm. per twenty-four hours. There is no apparent relation between tolerance and weight or edema.

The urine output was increased in all cases to whom the treatment as detailed was given; but it was not due to the glycosuria for either there was none or at most of only a moderate degree. In the latter event the excretion of glucose is so small in proportion to the urine that it is evident that other factors are involved. Once a polyuria has been produced in eclampsia by glucose, it usually continues. This may be due to changes produced by the glucose in the cell or the cell membrane which are changed in pregnancy.

The blood pressure undoubtedly plays a part in the diuresis. Experimentally it has been proved that the urine varies directly as the blood pressure, therefore, in the eclamptics one would expect large amounts of urine but actually an oliguria or anuria exists. The fact that a diuresis can be established so rapidly with glucose indicates that the urinary suppression is due more likely to spasm of the renal capillaries rather than to edema of the kidney. Examination of the capillaries of the nail bed has shown that the circulation improves during glucose administration. In some cases, we have seen the beading disappear. Furthermore, when the serum proteins are high, the osmotic pressure exerted by them in the kidney holds water in the capillaries; but when they are low diuresis is apt to occur. Therefore, in eclampsia after delivery if the capillary spasm could be relieved, a marked diuresis should occur, for the blood pressure is high, the proteins are low and the water content of the blood is increased. A negative water balance after delivery in eclampsia with a urine output of 4 to 5 or even 6 liters is common. Another factor is the increase in P_H which increases the base binding property of the serum proteins and also increases their water binding power.

In eclampsia we find that after glucose injection the chlorides are increased in the urine, and since they are an electrolyte, they will exert a greater osmotic pressure than glucose, thus resulting in a greater diuresis. Their excretion indicates that the kidney is attempting to maintain the osmotic equilibrium of the plasma and since the blood sugar is being constantly increased, this can probably be accomplished more easily by excreting chlorides, which exert a greater

even though there is no marked edema and the early convulsions in the majority of cases are, we believe, due to an edema of the brain which thus explains our control of convulsions with the glucose. We have found that the urine from eclamptics on admission, although concentrated as a rule, is relatively low in chlorides but after delivery or the death of the fetus the chloride concentration increases despite the diuresis which occurs. In the normal individual as the urine becomes more dilute the concentration of chloride decreases, but in the toxemics the reverse is true, and we have been able to cause marked excretions of chlorides in the urine by injecting glucose. In Table X we present data from a case of toxemia of pregnancy. The patient was admitted on August 17, 1928, because of headache and edema of the feet. The blood pressure was 215/120 and the urine contained a faint trace of albumin. On the seventeenth, eighteenth, nineteenth, and twentieth the amount of urine and chloride content varied but was in accord while the concentration was inversely proportional to the amount which is normal. Following the injection of 800 c.c. of a 30 per cent solution of glucose not only was the amount of urine and chloride increased but likewise the concentration of NaCl. This phenomenon occurred on each day that glucose was given. On the twenty-third and twenty-sixth no glucose was given and although the volume of urine remained high the chloride excretion was low. This decrease in chloride content after the washing out by glucose is due to retention by the body to replace tissue chloride. Since glucose replaces electrolyte, especially chloride, in the normal individual both in the blood and tissues, it is evident why the eclamptic patient with a tissue chloride retention will have a greater tolerance for glucose since the replacement is apparently based on the relative osmotic pressure exerted by chloride and glucose and not on their respective molecular concentrations. In some diabetic patients, Peters and coworkers find low serum chlorides and infer that the tissues are likewise deficient in chloride. Experimentally it has been demonstrated that there is an inverse relationship between the concentration of glucose and chloride in the blood after the injection of glucose, and it has been suggested that the chlorides possess the property of shifting to other tissues from the blood in order to preserve the optimal osmotic conditions in the blood (Foshay, Herrick).

A study of our cases has proved that the mild cases recover irrespective of the treatment, providing it is of a conservative nature, but in the severe type recovery is markedly favored by early death of the fetus or delivery. Since the blood dilution with its accompanying phenomena occurs after delivery or after death of the fetus, it is considered of prognostic value. Thus, for example, if, after the patient has been in our hands for eight to twelve hours and has been treated

as outlined, we find that the blood is not diluting, that there is no satisfactory diuresis, that coma is either not clearing up or is developing and that the temperature and pulse are increasing, then it is evident that the case is not only a severe one but delivery must be completed within a short period of time without additional shock. If delivery cannot be completed through the natural passage readily, then we prefer abdominal cesarean section under local anesthesia.

Table XI is self-explanatory. Our series is small but it has been carefully culled. A number of cases in whom the occurrence of convulsions was reported were excluded if the laboratory findings and subsequent clinical course were normal. (None of these died.) In the mild type one would expect no maternal mortality. In the severe type there will always be some maternal mortality and a high fetal mortality.

CASE HISTORIES

TABLE II. M. W. 1760

DAY	CELL VOL. PER CENT	SERUM PROTEIN PER CENT	GLUCOSE GM. INTRA "V"	GLUCOSE GM. URINE	URINE C.C.	NaCl GM.	NaCl PER CENT	WEIGHT KILO.
4–10	41.6	6.4						166
11	46	5.68	385					Delivery
12	27	4.43	200 (11 and 12)	22	7200	12.3	0.17	
13				13	2000	13.2	0.66	
14	28	4.92		0	4000	20.4	0.51	
15			90	0	5100	26.5	0.52	
16				0	4300	15.9	0.37	130
17	30	5.7		0	5500	21.4	0.39	
18					4000	26.8	0.67	
19	30.6	5.38			1400	7.1	0.51	
20					2500	7.3	0.27	
21					3900	9.4	0.24	
22					3000	8.4	0.28	
23	32	6.45			3100	6.2	0.205	121
24					2900	7.85	0.27	
25					2700	8.35	0.31	
26					2300	6.45	0.28	
27	36	7.05			2100	5.65	0.27	120
28					2150	5.57	0.26	
29					1500	4.5	0.30	
30					2600	7.28	0.28	
5– 5	36	7.23						116

M. W., 1760, primipara, thirty-four years old. At term. On April 6, 1928 patient had two convulsions and on April 10 she had four more. Admitted to hospital on April 10, 1928. She was very obese, in coma, and had a general edema. Blood pressure was 230/140. Urine coagulated on heating. During a period of fifteen hours the patient received 385 gm. of glucose intravenously and voided 1100 c.c. She became conscious. In view of the duration of the disease and the fact that the patient was now in the best condition that could be expected, a cesarean section under local anesthesia was done. A living 5110 gm. baby was delivered. Discharged on May 4, 1928. Note: The decrease of serum proteins before delivery. Marked blood dilution, diuresis, and weight loss (50 kilo in three weeks) after delivery.

TABLE III. E. A. 3380

DAY	CELL VOL. PER CENT	SERUM PROTEIN PER CENT	GLUCOSE GM. INTRA "V"	GLUCOSE GM. URINE	URINE C.C.	NaCl GM.	NaCl PER CENT	WEIGHT KILO.
10–18	45	5.55	440	120	2000	1.8	0.09	165
19	49	5.52	880	154	3350	5.3	0.16	Delivery
20	41	4.15	396	98	3060	5.8	0.19	
21	40	4.26	420	239	3810	7.7	0.20	
22	34	4.85	480	126	3450	6.5	0.19	
23	39.5	4.82	430	155	3250	5.8	0.18	
24				0	1300	0.7	0.05	
25				0	2075	0.5	0.02	
26	30	5.15		0	3250	1.0	0.03	
30	24.5	5.1						
11– 5	35	6.26						

E. A., 3380, primipara, sixteen years old. Forty-one weeks gestation. Normal pregnancy. Admitted on October 18, 1928. Had had headaches and dizziness for past two days. Blood pressure 142/75. Edema of ankles and face. Urine contained a large amount of albumin. Phenolsulphonephthalein test, 70 per cent in two hours with total urine output of 550 c.c. Four hours after admission patient had a convulsion and blood pressure rose to 170/110. During the next fifteen hours she had three more convulsions but despite 42 c.c. of 25 per cent $MgSO_4$ solution given during this period together with 2000 c.c. of 20 per cent and 800 c.c. of 30 per cent glucose solution intravenously, patient gradually became comatose, with temperature rising to 39.8° C. and pulse to 132. Patient was having contractions and the cervix admitted 1 finger, but delivery from below could not be completed under twelve to eighteen hours at a minimum, therefore, a cesarean section under local anesthesia was performed. A living 3450 gm. baby was delivered. Discharged November 9, 1928. Note: The steadily increasing blood concentration despite treatment. Marked dilution and diuresis after operation.

TABLE IV. W. P. 2734

DAY	CELL VOL. PER CENT	SERUM PROTEIN PER CENT	GLUCOSE GM. INTRA "V"	GLUCOSE GM. URINE	URINE C.C.	NaCl GM.	NaCl PER CENT	WEIGHT KILO.
8–12	34	5.13	400	7	500	0.85	0.17	
13	32.6	4.5	350	29	1650	0.93	0.06	
14			200		700			71
15				0	400	0.14	0.035	
16				0	500	0.01	0.002	
17	31.6	4.69		0	1630	0.23	0.014	72
18	55.2	5.60	100	12			0.006	
19	46	4.94	350	0	600	0.04	0.006	Delivery
20	32	4.28		0	5900	2.71	0.046	
21				0	5150	10.3	0.2	
22				0	5300	20.14	0.38	60
23				0	3800	13.4	0.35	
24	32	5.28		0	1600	4.4	0.276	

W. P., 2734, primipara, nineteen years old, thirty-six weeks gestation. Admitted on Aug. 12, 1928 on account of headache, edema of ankles and blood pressure of 180/110. Urine coagulated on heating. Received 1000 c.c. of 20 per cent glucose solution intravenously once or twice daily. Phenolsulphonephthalein test on Aug.

S. L., 7315, primipara, twenty-six years old, thirty six weeks gestation. Edema of legs for month. Headache, spots before eyes, pain in epigastrium for last four days. Had 2 convulsions before admission on Oct. 16, 1926. She was in deep coma and very edematous. Urine contained large amount of albumin. Blood pressure 180/125. Had one convulsion shortly after admission. Within twenty-four hours following treatment the patient was conscious and fully coordinated. On October 18 patient began to vomit and abdomen became distended. Blood pressure had risen to 210/135. Urine output on eighteenth was 930 c.c., on nineteenth was 2775, on twentieth was 1800 and on the twenty-first was 1050. On the nineteenth approximately 550 c.c. of blood were removed. On the nineteenth, patient

TABLE VI

NUMBER	WEIGHT KILO.	EDEMA	DAY	GLUCOSE GM. INTRA "V"	GLUCOSE GM. URINE	URINE C.C.
M. W.	87.9		22	400	19	720
8392			23	500	28	4900
		Ankles	24	400	28	4040
			25	200	12	2420
			26	200	0	1800
	78.2		27	200	0	2650
F. F.	77.6		18	575		600
11,738	76	Legs	19	200	7	900
	62.5		20	400	1	400
E. B.	70.2		16	400	1	830
440		General	17	750	32	3100
			18	200	57	4500
N. C.	78.9		16	320	23	1400
1332		Ankles	17	100	0	2500
			18	100	0	4000
	67.5		19	200	0	2100
M. W.	166		11–12	585	22	7200
1760	116	General	13		13	2000
			14	200	0	4000
			15	90	0	5100
W. P.	71		12	200	7	500
2734	72.3	Legs	13	350	29	1650
	60		14	200	0	700
			18	200	0	400
			19	150	12	500
L. W.	72		17	290	35	1500
4129	49.5	General	18	180	36	2000
	47		19	240	65	1600
W. B.	69		2	500	9	1775
4640		Ankles	3	200	62	500
	61.8		4		10	1700
F. S.	69.6		6	400	77	2950
3779		Ankles	7	370	120	2850
			8	200		3650
W. W.	65		25	400	58	1310
3437		Ankles	26	400	118	2100
			27	480	91	2400
	58.2		28		68	2600
E. A.	75		18	440	120	2000
3380		Ankles	19	880	154	3350
			20	400	98	3060
			21	420	239	3810
			22	480	126	3450
			23	440	155	3250

TABLE X. O. H. 2796

DAY	GLUCOSE GM. INTRA "V"	GLUCOSE GM. URINE	URINE C.C.	NaCl PER CENT	NaCl GM.
17		0	1240	0.4	5.0
18		0	2200	0.2	4.4
19		0	1850	0.26	4.8
20		0	1700	0.26	4.4
21		0	2900	0.19	5.5
22	225	61	3400	0.27	9.2
23		0	2525	0.07	1.8
24	225	74	3300	0.15	5.0
25	270	64	3250	0.14	4.6
26		0	2300	0.056	1.3
27	270	75	3400	0.16	5.4

TABLE XI. CASES OF ECLAMPSIA, 1923-1929

CASES	NUMBER	POST-PARTUM	SPONTANEOUS ONSET OF LABOR	INDUCTION OF LABOR	CESAREAN SECTION	MATERNAL MORTALITY	BABY LIVING	BABY DEAD
Mild	15	6	8	1	1		15	
Severe	18		7	4	4	3 (2 undelivered)	10	6 misc.
Total	33	6	15	5	5	3	25	6

W. H., 3283, gravida ii, thirty-five years old, twenty-eight weeks gestation. Admitted as a private patient on Oct. 8, 1928 at 8 A.M. because of vomiting and loss of consciousness. Onset was about eight hours before admission. Shortly after entry she had a convulsion. Blood pressure was 195/105 and a slight edema existed. Urine coagulated on heating. Therapy consisted of 500 c.c. of 20 per cent glucose solution intravenously, 1000 c.c. of Ringer's solution subpectorally and 10 c.c. of a 25 per cent solution of $MgSO_4$ at 11 A.M. At 4 P.M. 800 c.c. of urine were obtained by catheter. At 7 P.M. patient was given 250 c.c. of a 20 per cent glucose solution intravenously and 1000 c.c. Ringer's subpectorally. At 9 P.M. 300 c.c. of blood were removed. Stimulants were started at this time. At 10 P.M. 250 c.c. of urine were obtained by catheter, 11 P.M. venesection of 500 c.c. of blood, 11:45 P.M. patient given glucose and 450 c.c. citrated blood. Stimulants continued. Respiration gradually increased until the rate was 40 to 50 per minute. Death occurred at 8 A.M., twenty-four hours after admission. Autopsy Diagnosis: Eclampsia. Focal necrosis of liver. Degeneration of the kidneys. The case with which a diuresis was established with the small amounts of fluid, the early admission to the hospital after the onset together with the duration during which the patient was treated warrant the belief that more intensive use of hypertonic glucose solution and delivery in the afternoon would probably have resulted in recovery.

SUMMARY

Eclampsia is best treated by certain definite procedures which by their success or failure permit one to note the progress of the case. After delivery or death of the fetus a marked blood dilution takes place, during which period a diuresis occurs. Clinical improvement is closely associated with these phenomena. The eclamptic patient has an increased tolerance for glucose, probably due to the retention of chlorides found in pregnancy. The injection of large amounts of intra-

venous glucose solution will simulate temporarily at least the effect produced by delivery. The prognosis for the patient with a severe type of eclampsia is chiefly favored by delivery or early fetal death.

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