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## Diabetic coma

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DIABETIC COMA

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OMAHA, NEBRASKA

1932

## DIABETIC COMA

Diabetic coma is a true medical emergency. It is just as much an emergency as an acute appendicitis or an incarcerated hernia, and as in the latter condition, with every hour that passes without treatment the chances for life decrease. It is early intervention that counts. Therefore, a physician should never let an engagement or any personal desires keep him from the bedside of a patient whom he thinks may be in coma.

### HISTORY

Diabetic coma has been known as a clinical entity since 1850 when a German, Von Dusch and a Scotchman, March first described it. Twenty years later, Kussmaul published his classical description of diabetic coma. It is the "Kussmaul breathing," as described by the author which is the outstanding and characteristic feature of diabetic coma.

### ETIOLOGY

A diabetic patient may be thrown into coma by any accident which to a normal individual would cause little reaction. The following case illustrates the effect of an accident upon a diabetic patient.

Case 1: C.W., a white male, age 44 years, was brought in September 15 in coma. Urinary analysis showed sugar, diacetic acid, and acetone to be 4 plus. The cause of coma was undetermined. He was given insulin 20 units every two

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hours, buffered with glucose. By morning his blood sugar was 325 mgm %. The patient was then put on a diet and insulin. He soon became sugar free and was dismissed with an insulin dosage of 15 units. He returned in December with a fractured right elbow. His insulin dosage was increased to 20 units. The patient was dismissed in two weeks in good condition. He returned again in a week with gangrene of the right leg. His blood sugar was resistant to insulin. He soon became comatose and died.

Infractions of dietary regulations is by far the most common cause of coma. Joslin(1) states that 70% of his coma cases are due to dietary infractions. This is especially true in children, as they are unable to appreciate their condition and often succumb to the delights of a "carbohydrate spree." The following case illustrates the difficulty in keeping children from obtaining forbidden delicacies.

Case 2: J.G., an obese white girl, 13 years old was brought to the hospital the night of July 2 in coma. She had been in the hospital five times in the last six years. The etiology in this case was definitely a candy spree. She was given insulin in large quantities, glucose, caffeine sodium benzoate, and adrenalin, but she did not respond. Died July 4 without having become conscious. Necropsy showed very little pathology. The islands of Langerhans were diminished in number, but not sufficiently to account for her severe diabetes. The pathologists diagnosis was that death was due to overeating of carbohydrates.

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Bowen and Hekimian(2) in an analysis of their cases report the following figures; infraction of dietary rules 52%; omission of insulin 17%, infections 15%; undetermined 16%. Insulin was omitted for various reasons-vomiting, the extraction of teeth so that the patient was unable to eat, breaking of an insulin syringe and failure to have another in reserve, self experimentation, carelessness and indifference. In 1927 in a period of 10 days, Bowen and Hekimian(2) saw 4 cases in which coma was abruptly precipitated by withdrawal of insulin, two having omitted only two doses. This is as convincing as the experiment of Hedon who produced typical diabetic coma in partially depancreatized dogs by getting them in good physical condition through the use of insulin and then suddenly withdrawing it.

The incidence of coma due to infections seems to be increasing according to Joslin(1). Infections should not precipitate coma if the patient and the physician realize their importance to the diabetic. Under such circumstances the insulin should be increased, the diet reduced, and the infection treated radically, if possible. In certain conditions this is not possible and if the patient contracts pneumonia or has a focus of infection which cannot be reached, the prognosis is grave and the case terminates very quickly, as in the following case.

Case 3: I.A., a white Jewish boy, 13 years old, was brought into the hospital in coma on May 20. He had been well until a month before that date, when his parents noticed that he had an excessive appetite and thirst. He had polyurea and nocturia two and four times. He was listless and somewhat drowsy for the

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last month. The patient was semicomatose for 24 hours. He became comatose and was brought to the hospital. He had an acetone odor and sunken eyes. The patient was emaciated. There were no lung findings upon admission. He was given 15 units of insulin on admission and 10 to 20 units every half hour. He was given 1000 c.c.'s of 2% soda at 2A.M. and 10 P.M. Caffeine-sodium-benzoate grains  $7\frac{1}{2}$  was given as a cardiac stimulant. Orange juice was given by mouth whenever the patient seemed to respond. He responded at 4 P.M. after administration of 1500 c.c. of normal saline. The temperature increased rapidly from 99.6 to 106 rectally in 16 hours. Signs of pneumonia were discovered. He died 5 P.M.

The chemical cause of coma is still the center of many heated discussions. According to Ross(3) the coma is due to a disturbance in the acid-base equilibrium. He reasons as follows: diabetes manifests itself as an inability of the individual to utilize fats. This in turn is due to the fundamental characteristic of the disease, inability of the individual's system to utilize sugar. For some, as yet obscure reason, "fats burn only in the flame of carbohydrates," in the body. It has been found that, when in the process of metabolism more than two molecules of fatty acid to one of carbohydrate are being utilized the ketone acids, which are products of incomplete combustion appear in the blood, urine, and other tissues. The acid-base equilibrium of the body is thus disturbed and the complicated efforts of the system to bring about restoration of function,

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bring about the symptoms under discussion. Some idea of the delicacy of the acid-base regulation may be gained by the consideration of the fact that a reaction of the blood as acid as distilled water or as alkaline as ordinary tap water on the other hand is incompatible with life.

In direct contrast with this theory is the research by Dodds and Robertson(4) on the relation of aceto-acetic acid and the alkali reserve in the plasma of patients in diabetic coma. Thus, in many cases, non-comatose diabetic patients were found to have a higher aceto-acetic acid content than comatose patients. It was also found that there was no consistent relationship between the acidosis and coma, and it was impossible to correlate the blood chemistry with the clinical condition of the patient.

Dungan(5) made a comparative study of the toxicity of the ketone bodies. He found that though they were all toxic, they required doses which are not present in the body under any circumstances. Beta-oxybuteric acid was the most toxic. He was unable to obtain the typical diabetic coma through the use of any of the ketone bodies although he did obtain a condition somewhat similar to coma by use of sodium butrate.

John(6) sums up the present position in his statement, that coma is not a result of the rise of blood sugar or of formation of ketone bodies. These are only superficial chemical signs of some metabolic derangement which causes the condition. As to the nature of this metabolic derangement, we are at present uncertain and much research work will be required to clarify the

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situation. John has shown that there is no definite blood sugar level which is pathognomonic of coma. In his report of eighty-two cases the blood sugar ranged between 200 and 1,700.

### INCIDENCE AND MORTALITY

Before insulin was used, the mortality rate accompanying coma was practically 100%. Through the use of insulin this high rate has been reduced considerably, the average rate is 25%. This is a tremendous gain and the figure will no doubt improve in time; but it is doubtful if we can ever attain Joslin's ideal of a mortality rate of 0.

Coma attacks the young diabetic and the diabetic who is in the first few years of his disease. Joslin(1) reports that 53% of his cases were between 10 and 20 years of age. Coma, however, is no respecter of age. There is a very high percentage of death's from coma in the 6th decade. This is explained by the fact that the years of 50 and 51 are the peak years for the onset of diabetes.

More than one half of the deaths from coma take place in the first four years of the disease. It is exceptional after ten years of diabetes. This is the period, therefore, that all must concentrate their efforts to overcome coma.

John reports the incidence of coma in his series as 4% and a mortality rate of 5.6%. I doubt if these figures can be reached elsewhere. In some hospitals the mortality rate has run as high as 88%.



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TABLE SHOWING MORTALITY RATE OF JOHN'S CASES

Total number of patients	2,572
Total number of cases of diabetic coma	103
Incidence of coma	4%
Cases treated by John	71
Cases treated elsewhere	32
Deaths from coma treated by John	4 or 5.6%
Deaths from other causes	
Pneumonia	2
Cardiac failures	4
Uremia	1
Septicemia	2
Deaths from coma treated elsewhere	28 or 87.5%

### SYMPTOMS

The symptoms of acute acidosis leading to coma are often protean in their original manifestations. There are, however, certain signs that should put one on guard and suggest careful urinary examination for diacetic acid and acetone and a blood examination for blood sugar and carbon-dioxide.

The most striking symptom is the "Kussmaul breathing." This is characterized by deep, slow, and prolonged inspiration and short expiration with a short pause between. It is as if the patient had some big load on his chest which he is trying to throw off. As the coma continues the respirations increase in frequency. Before the discovery of insulin, such breathing would continue for several days finally disappearing as death approached. This symptom is occasionally absent. Joslin(1) cites two cases in which "Kussmaul respiration" was not present.

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Pain in the epigastrium is often one of the first complaints of the patient. This is sometimes accompanied by nausea and vomiting. Joslin(1) stresses this fact greatly as he says that very many patients have been operated for appendicitis, when they were on the verge of diabetic coma. Since a leucocytosis is often present, the differential diagnosis is often difficult. He has been so enthusiastic in bringing this fact before the medical profession, that many protests have arisen from many great physicians and surgeons who believe that many cases of acute appendicitis in diabetics may be missed, because practitioners will discard a possible diagnosis of appendicitis until too late.

Leucocytosis is very often present, without any concomitant infection. Anderson(7) cites three cases in which the white counts were 79,000, 97,000 and 24,000. He was unable to find any signs of infection. He concluded from a study of the literature it is usually higher in children. Joslin(1) reports 5 cases ranging from 14 to 44,100 without infection or gastric hemorrhage. Allan(8) cites 5 cases ranging from 16 to 66,400 uncomplicated by infection. Usually the degree of leucocytosis is slight, the white count running from 10 to 12,000.

There is a soft eyeball, due to decrease of intraocular tension. This in turn is due to dehydration. The eyeballs grow progressively more soft as the coma deepens. Lawrence(9) says that he has seen cases in which the intraocular tension is so low and the eyeball so flat, that he had to lift the eyelid to make sure that the eyeball had not previously been removed.

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The temperature is usually subnormal, but arises in the preagonal stage or if an infection occurs. A rise in temperature should therefore cause the physician great concern and necessitate a thorough examination to find any infection which may be present.

Dehydration is often extreme. This is evidenced by a dry skin, red glistening throat, mouth, tongue, and lips which are very dry, crack easily. The superficial tissues become flabby. And the blood pressure becomes low. The leucocytosis is in part due to the concentration of the blood.

The Löwy reaction is positive, that is, the pupils dilate when epinephrine is injected into the conjunctival sac. This reaction is supposed to be pathognomonic of pancreatic deficiency.

There are several other clinical symptoms which may be present. Headache is often present. Patellar reflexes disappear (in severe cases of diabetes they may disappear even before coma develops). The eyes present constricted pupils or constriction and dilatation may alternate. The acetone or fruity odor to the breath becomes marked.

## LABORATORY DIAGNOSIS

The laboratory findings are very important, but any of them may be absent. Hyperglycemia is usually present. This is very important as a sign of diabetes, but is not indicative of coma. John(6) reports a variation of the blood sugar, in his coma patients, between 200 and 1,7000. Acetonemia is usually present. Rudy and Levin(10) report a case in which it was absent. The carbon-dioxide content is very low sometimes

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reaching as low as 10 volumes percent. Lipoids and fats are often present in the blood.

A great deal of dependence is placed on the urinary findings as these are easily obtained. There are, however, several possible sources of which should be kept in mind. A urine that gives a Burgundy red color with ferric chloride does not always mean diabetes as it may be produced by salicylic acid or its derivatives. The urine should be boiled and if the color disappears on boiling it was due to diacetic acid. Lemann(11), Starr and Fitz(12), (seven cases), Oliver(13), Argy(14), Paddock(15), McCaskey(16), Evans(17), Rudy and Levin(10), and Appel and Cooper(18), have all described cases in which acetonuria was absent. It is interesting to note that most of these cases showed signs of acute or subacute nephritis. This may possibly afford an explanation, in as much as the kidneys were unable to excrete the ketone bodies due to impaired function.

## PITFALLS IN DIAGNOSIS

Lemann(11) states that there are two factors which may serve to confuse in the case of a person known to be diabetic. Both of these, as Root(19) points out have arisen since the advent of insulin. First the diabetic coma may be due to insulin overdosage. Second, diabetic patients live to an older age and more of them reach the "uremic zone of old age." The differential diagnosis, will rest upon the examination of the urine for sugar and ketone bodies or better still, if possible, a determination of the blood sugar level. One should remember that the first specimen of urine may contain sugar,

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since it was in the bladder before the insulin was taken.

Root(19) points out that a known diabetic may become comatose, not because of diabetes, but because of some inter-current condition, as, for example, septicemia, meningitis, cerebral hemorrhage, or an uremia. He also points out that we may have glycosuria and hyperglycemia without diabetes and illustrates this statement with reports of two cases of intracranial hemorrhage in the neighborhood of the fourth ventricle. These cases offer little confusion, however, because the symptoms are usually acute. A spinal puncture will suffice to make the diagnosis.

## PREVENTION

The most successful treatment of diabetic lies in its prevention. In most cases it is preventable and if it occurs can be handled without considerable difficulty. When treated as an emergency, nearly every patient, unless his case be complicated by a serious disease, would recover unharmed and often be benefited in that he has learned a valuable lesson. It does not appear however that coma can be entirely eradicated as diabetes is often not diagnosed until coma appears. There are also a certain percentage of patients, who, even though carefully taught, are normally careless and indifferent, and there are some severe diabetics who, even though they take good care of themselves may be easily swung into coma by a trivial circumstance. The physician must carefully instruct his diabetic patients to come to him whenever an infection occurs. They should also be very thoroughly cautioned against breaking

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their diet or omitting insulin. Joslin(20) believes that periodic visits to a physician or clinic is the most efficient safeguard that a diabetic may have.

According to Joslin(1) a diabetic patient will not contract coma, as long as he does not overeat. He will not die of coma, although he may die of inanition. He can overeat of food carelessly or overeat of his own body innocently, because in fever or in hyperthyroidism he does not realize that his metabolism is raised and he is overeating. However, overeating for a diabetic is a very different proposition than overeating for a healthy man. The calories which constitute even a normal diet for a man represents overeating or excessive diet for the patient with diabetes. The only way the severest diabetics can exist is by undereating, by living on a lower plane of metabolism. Insulin, it is true, allows the intake of a normal quantity of food, but every insulin taking diabetic is overeating as far as his own resources are concerned, and if he omits his insulin and continues his food, he will pay the penalty of a reckless and disobedient diabetic, and if his case is severe he will develop coma. Furthermore, even if he stops eating and gives up insulin he may develop coma, because he is no longer the thin diabetic with a low metabolism, who can oxidize the little carbohydrates, fats, and proteins which his emaciated body, requires; for today he is usually well nourished and his tissues are capable of furnishing him many hearty meals which he cannot utilize without insulin. There-

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fore, every insulin taking patient is a potential coma case and should be treated as such.

Every surgical diabetic is a candidate for coma. Urinary tests should be made every four hours before and after operation. Preoperative preparation; (1) Size of insulin dose depends on the preceding treatment and urinalysis. (2) 25 grams of carbohydrate in the form of orange juice or oatmeal gruel by mouth or 10% glucose intravenously. Postoperatively the urine should be tested every two, four, or six hours depending on the amount of glycosuria. The diet should contain 75 to 100 grams of carbohydrate during the first two or three days. One must forestall dehydration oliguria, dilatation of the stomach and circulatory collapse. Even with all these precautions the patient sometimes goes into coma several days post operatively as the following case will demonstrate.

Case 4: Mrs. W.C. a white woman, 60 years old, came into the hospital with a diabetic gangrene of the foot. She was prepared for operative removal of the great toe by an intravenous administration of 500 c.c's of glucose and 30 units of insulin. This treatment was repeated postoperatively. She appeared to withstand the operation fairly well. While she was being prepared for removal of the leg, she went into coma. This occurred on the fifth day post operatively. She was in coma for about two hours, when she was given 40 units of insulin intravenously and 20 units subcutaneously. She responded slightly to questioning, but could not be completely aroused even with administration of insulin. Death came about six hours

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after she had lapsed into coma.

Surgeons often consider a diabetic case as one which offers very little more complications than any other case. It is best to bear in mind that some diabetic cases may fall into coma inspite of all diabetic preoperative care.

## DIFFERENTIAL DIAGNOSIS

The differential diagnosis of diabetic coma is not difficult if someone has observed the patient for a while before he became comatose or a good history can be obtained. On the other hand, if the patient is found in coma without an obtainable history, the problem becomes more difficult.

If the patient is elderly, we would probably think of uremic coma first. It would not be difficult to differentiate between these two conditions if one keeps the following points in mind. The blood pressure in uremic coma is high, in diabetic coma low; the urine shows albumin and casts in uremic coma, and sugar and acetone in diabetic coma; the retinitis of nephritis is present in uremia, but since all older diabetics, as Smink(21) points out, soon become arterio-sclerotic and develop some kidney condition, the diagnosis by glycosuria is more reliable; the "Kussmaul breathing" is different from the stertorous respiration of uremia; and the acetone odor of the breath, soft eyeballs, and normal blood urea of the diabetic coma is opposed to the high blood urea and normal blood sugar of uremia. The carbon dioxide content here is of great value here as it is usually normal in uremia.

One thinks also of a brain tumor in a comatose patient, but the choked disk, change in visual fields, normal breathing,



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aglycosuria, normal blood sugar and non-protein nitrogen, normal carbondioxide content, and projectile vomiting will rule it out.

In the case of cerebral hemorrhage we have dilated or unequal pupils as opposed to the soft eyeballs of coma. There is often glycosuria in cerebral hemorrhage, especially if the hemorrhage is due to a skull fracture and is in the neighborhood of the fourth ventricle. The breathing is not "Kussmaul" in type, but is slow and stertorous, and is often interrupted by periods of apnea. The carbondioxide content of the blood is normal, the blood pressure is high, and often there is evidence of paralysis as indicated by unequal resistance of the limbs on lifting, conjugate deviation of the eyes, or facial paralysis that is noticeable. Acetone is absent from the urine, blood, and breath.

Meningitis, may bother sometimes, because a glycosuria is present in this condition also. Since a glycosuria may be present in several other conditions which cause coma, one must be careful in accepting urinary sugar as the diagnostic feature of diabetic coma. The presence of fever, Kernig's sign, the cervical rigidity, the changed or exaggerated reflexes, the normal blood sugar and carbondioxide content of the blood makes the diagnosis.

Sometimes a case treated with insulin may go into coma and the question of diabetic coma or insulin shock may become very embarrassing. One must remember that in insulin shock the first specimen of urine obtained by catheterization may

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show sugar, as it may have been in the bladder previous to the last dose of insulin, which caused the shock, was administered. It is well to get a second specimen by catheterization, if possible. This will show no glycosuria in insulin shock. The blood sugar is high in diabetic coma, and is low in insulin shock. Acetone is not very likely to be present in the urine in insulin shock. The breathing is normal, the eyes are not soft and there is no fruity odor to the breath. The Krause sign, or softness of the eyeball, is a very important sign which is indicative only of diabetic coma, and would serve to differentiate it from any other condition.

## PROGNOSIS

According to Ross(3) no child has ever died of diabetic coma, but many have died of the condition which precipitated the coma. This also holds true in adults, in spite of the fact that often nothing is found to account for the coma. It is, therefore, very evident that one of the first things for the physician to do is to hunt for the cause of the coma and if it is an infection to treat it radically. According to Joslin(1) a patient who became comatose through breaking the diet or omission of insulin, should emerge from the coma unharmed provided the coma is treated promptly and energetically.

## TREATMENT

The object of treatment is to bring about the combustion of sugar in the body and in this way bring about the simultaneous combustion of the ketone acids, with the liberation of the bases bound by these acids. As soon as the diagnosis of diabetic

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coma is established, insulin should be administered subcutaneously, intravenously, or both. Ross(3) states that the amount of insulin to be administered can be approximately estimated if the percentage of the blood sugar is known. It can be assumed that a similar concentration of sugar is present in other fluids of the body. As the body is approximately 60% fluid, this percentage of the weight of the body in kilos will give the amount of fluids present. This number times the percentage of sugar equals the amount of sugar present in the body. Deduct the normal amount of sugar present in health and the excess will be shown. On the basis of it requiring 1 unit of insulin to bring about combustion of  $1\frac{1}{2}$  to 2 gms of glucose, the amount necessary can be estimated. In practice it is best to administer only a part of this amount and to follow with more if the symptoms persist. Joslin(1) has found that insulin given in moderate doses of 20 units every 15, 30, or 60 minutes appears to act much more powerfully than greater doses at longer intervals and there is less danger of overdosage. If one does not have the blood sugar report, insulin is given in doses depending upon the depth of coma, the general appearance of the patient and the response to the previous dose. Joslin's(1) average dose is 150 units the first day and about 63 units the second day. The fear of bringing on an insulin shock is of minor importance. John(6) believes that enough insulin should be given to a patient in diabetic coma to control the condition, whether it is 100, or 1000 units in 24 hours. There is much more danger, in his opinion, in not

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using enough insulin, than in using too much. A patient in coma, especially if he has an infection, soaks up insulin like a sponge, that is, insulin does not seem to produce the same effect per unit as in an ordinary case. The patient must be very carefully watched by the physician until he is out of coma and the danger of insulin shock is passed. Even then instructions should be left with assistants in case the patient went back into coma or into shock. In the following case the patient was left alone without attention after she came out of coma and she died of insulin shock.

Case 5: A white woman, 28 years old, was brought to a hospital in January. She was delirious and stuporous. Symptoms; Hyperpnea, rapid pulse, and a temperature of 101. She was believed to have pneumonia. Urinalysis showed 4 plus sugar which was 3.8% quantitatively. She was then put on emergency diabetic treatment consisting of 40 units of insulin buffered by orange juice and sugar, every hour. She became lucid in 2 days, but she still showed a blood sugar of 190 mgm %. As soon as she became conscious, she was given 60 units of insulin. The physician, considering all danger past, left the hospital with the instructions to call him and not the intern should something occur. No one was given orders to watch the patient. The patient very soon went into hypoglycemic shock. She was not seen for half an hour at which time both the physician and the intern were called. The patient died several seconds after the intern arrived.

In the above case the physician did not realize the danger

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of hypoglycemia. A patient should be watched for a while after insulin administered and every ambulatory patient and his family must be instructed to watch for restlessness, nervousness, sweating, tremor, anxiety, or excitement after taking insulin. The treatment of hypoglycemic shock is simple. A little orange juice, a lump of sugar, or some candy clears up the condition miraculously. Conlin(22) suggests that every insulin taking diabetic carry a card with his name and address, his physician's name, address, and telephone number and a statement that he is a diabetic and that if he suddenly becomes unconscious it is due to insulin. The finder is also instructed to give him some orange juice or two lumps of sugar in water. The patient should always carry some glucose candies or oranges in his pockets.

Opinions as to whether or not glucose should be given intravenously are about equally divided among different workers. An individual with a blood sugar of 600 has a total of 30 grams of glucose in his circulation. If 250 c.c's of a 10% solution of glucose is given intravenously 25 grams more are added, so that 55 grams is in circulation. To take care of this amount 28 units would be needed. If 50 or 100 units are added to the solution, it is more than enough to take care of this added amount of carbohydrate(John). That the dextrose does not embarrass the patient is evident by the subsequent fall of the blood sugar, the disappearance of acetone, and the rise of the carbondioxide. The purpose of the glucose solution is to add fluid to the dehydrated body, to increase the excretion

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of urine, which is markedly lowered and through this to wash out a certain amount of the acid bodies. By means of glucose, oxidation in the blood stream and tissues is increased and this increased oxidation helps to burn up acid bodies. The glycogen--poor liver is restocked with glycogen, which in itself is an important factor in lessening the acidosis. The heart muscle is also restocked with glycogen. In view of these considerations, the use of intravenous glucose is desirable. Any patient in diabetic coma should be given intravenous glucose and insulin before sending him to the hospital. This should be repeated every half hour until he reaches the hospital.

To counteract the anhydremia which is nearly always present, fluids in the form of normal saline solution should be administered. The sub-pectoral administration of saline is preferable to the intravenous, because of the danger of thrombosis, because absorption is more gradual, and because it is far simpler in patients whose blood pressure is low.

Joslin(1) orders gastric lavage on all patients whose carbondioxide is below 20 volumes  $\%$ , even without a history of nausea and vomiting. He also gives caffein-soduim-benzoate in doses of  $7\frac{1}{2}$  grains every few hours, not exceeding 35 grains in 24 hours.

Lawrence(9) differentiates those cases requiring immediate intravenous administration from those which require insulin alone. They show marked evidences of dehydration, collapse, and circulatory failure. The pulse is rapid, the

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blood pressure is low, the veins are empty, the tissues are drained of water, and the most striking sign is the low eye tension. This dehydration is produced by the intense poly-urea of the preceding period and a failure to absorb fluids because of vomiting and unconsciousness. Another feature of these desperate cases is a very scanty secretion of urine or even an anurea. He divides the cases of diabetic coma into two groups according to the chemical findings in the blood as follows:

- |                         |   |
|-------------------------|---|
| (1) Diabetic complex    | (2) Dehydration complex   |
| Hyperglycemia           | Tissue depletion of water                                       |
| Excess of ketone bodies | e.g. low eye tension  |
| Low alkali reserve      | Low blood volume and pressure                                   |
|                         | Concentrated blood, high hemo-<br>globin, and specific gravity. |
|                         | Nitrogen retention-high blood<br>urea                           |
|                         | Low blood chlorides   |

Lawrence suggests the continuous intravenous administration of 1.2% saline at a rate of one liter in 1/2 to 3/4 hour, never faster. This may require 3 to 5 or more pints of fluid. He follows this with a pint of gum acacia, a fluid which remains in the circulation while much of the saline rapidly leaves the blood and enters the tissues. After the intravenous administration, subcutaneous or rectal saline should be continued, unless this remains localized and is not absorbed

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into the tissues. This is an indication that the body is sufficiently supplied with fluid. He sums up his article with the statement that dehydration is the immediate cause of death through circulatory failure in desperate cases of diabetic coma.

Sometimes, in spite of all measures previously mentioned for the relief of coma, the symptoms fail to improve. The blood pressure steadily falls, vomiting and drowsiness return, the pulse fails and the kidneys stop secreting. This is the condition called "Medical shock" by Atchley. The urine at this time, if any can be obtained, is free from acetone, but contains albumen and many casts. In such a case, transfusion of blood has been a life saving measure. It, therefore, should be emphasized that in all cases of diabetic coma especial attention should be given to the blood pressure as a reliable index to the condition of the patient. The blood of every diabetic patient should be typed and a suitable donor kept in readiness in case the emergency for a transfusion arises.

## USE OF ALKALI

The most disputed element in the treatment of diabetic coma is the question whether the use of alkalies are indicated. Most of the best clinicians have discarded their use. There are still very many men who claim that the use of alkalies is justified on an experimental and clinical basis. Bowen and Beck(23) reported cases accompanied by nephritis, urea retention and oliguria or suppression of urine, in which improvement did not take place until alkalies had been given.



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The opposite viewpoint is taken by Lemann(23) who states as follows: It is striking that insulin acts not only as a specific antidote to the ketone bodies but also by raising the sodium bicarbonate ratio of the blood and the tissues, sometimes to the point of causing a definite and menacing alkalosis; so that whether we adopt the acid intoxication theory or the specific intoxication theory, insulin might by itself theoretically be regarded as sufficient. He claims that he has been successful in treating cases of coma without ketonuria successfully with insulin unaided by alkali.

Hartmann and Darrow(25) have recently reported a comparative study of the composition of the plasma in severe diabetic acidosis and the changes taking place during recovery in cases treated with insulin and carbohydrates, but without salt or alkalies, cases with insulin, carbohydrates, and Ringer's solutions, but without alkalies and cases treated with insulin, carbohydrates, Ringer's solution and alkalies. They find that the base-carbonate and hydrogen ion concentration ratios were restored slowly by insulin and it is their belief that the salt helped very little. They state, however, that sodium bicarbonate when given with carbohydrates, insulin and salt solution may provide a rapid and safe and complete relief from acidosis. They comment on the rapid restoration of normal breathing following the administration of alkali.

Ross(3) states that in as much as the air hunger present in acidosis is due to the lowered carbondioxide tension in the lungs, which in turn is caused by the lowered amount of

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sodium bicarbonate in the blood, it might be assumed, and it has been so assumed in the past, that the administration of sodium bicarbonate to a patient in diabetic coma is indicated. In recent years, however this practice has been abandoned by the best clinicians. When sufficient insulin has been administered to cause oxidation of the ketone bodies, the bases freed thereby will form sufficient bicarbonate. The administration of more bicarbonate in such cases tends to disturb the osmotic equilibrium of the tissues and may lead to an alkalosis, which is as serious a problem to deal with as an acidosis. Fruit juices administered by mouth supply alkali in small amounts by their oxidation and as the alkali is slowly liberated it may be used to replenish the bicarbonate.

A simple experiment by Goldblatt is very instructive. He starved himself for 48 hours. At the end of that period the urine contained fairly large amounts of acetone and diacetic acid. After he had taken 50 grams of glucose the ketosis disappeared in an hour. He repeated this experiment taking sodium bicarbonate in addition to the glucose. In this case the ketosis was present for more than eight hours.

Joslin and John both believe that the use of alkali is dangerous. At any rate, the fact that before insulin was used, alkalies were used universally, and found to be of no value, ought to be sufficient evidence for their disuse.

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### PRECAUTIONS

The following series of precautions by John(6) are so well stated and complete that every physician should have them well in mind:

1. Coma is an acute emergency, therefore one should not delay and should answer immediately a call to a patient in coma.

2. If one is reasonably certain that the condition is diabetic coma, the administration of insulin should not be delayed. One should not wait to examine the blood, but should give insulin first and examine the blood afterward.

3. If the patient must come to the physician from a distance in an ambulance, one should direct that 40 units of insulin be given before starting and 20 units every half hour until he arrives or at least until there is some decided clinical improvement.

4. One should not ignore or miss the presence of an acute infection or otitis media in a child.

5. If the patient is dehydrated and vomiting, the stomach should be washed out and water given freely, followed by hypodermoclysis of 2000 c.c's.

6. Care must be taken not to miss a distended bladder. If in doubt catheterize.

7. If the patient seems to be moribund, do not wash out the stomach unless it is distended. The patients life may be lost in so doing.

8. One should not feel that all is well just because the patient responds quickly to treatment, but should continue

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the routine treatment, for a relapse may follow a few hours of inactivity.

9. The patient should not be left until it is certain that the acute emergency is passed. Not till then should one place the responsibility on trained assistants and no matter how experienced the trained assistant may be, definite instructions as to treatment should be given.

10. One should not fail to take a blood culture if coma is due to some infection, such as a carbuncle or pneumonia.

### SUMMARY

Diabetic coma, -a medical state - easy to prevent, expensive, time consuming and difficult to treat, recovery a miracle to all beholders, but death a lasting blot upon the reputation of the patient or his physician (Joslin(1)).

Coma can be prevented in a majority of cases, but it will never be abolished because of infections, self experimentation, and carelessness. There are also many cases in which diabetes is discovered while the patient is in coma and there are patients whose coma threshold is so low that any incident may precipitate them into coma.

Many of the best clinicians believe that alkalies are dangerous and have discontinued their use.

Enough insulin should be given to control the condition and more effect is noticed if moderate amounts are given frequently.

The most desperate cases die of circulatory failure caused by dehydration. Fluids should therefore be given in large

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amounts.

Heart stimulants should be given.

In general the same routine measures which are employed for shock should be utilized in coma, giving the insulin a better opportunity to act and thereby increasing the chances for a complete recovery.

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