A COMPLICATION OF DIABETES DURING SPINAL ANESTHESIA *

MILTON C. PETERSON, M.D., RAPHAEL ROBERTAZZI, M.D., AND JOAN GOEBEL, M.D.†

New York, N. Y.

INSULIN hypoglycemia is accompanied by widespread changes in body functions. The effects in the nervous tissue come about as the result of the decreased sugar available to the brain cells (1). The first type of response is a sympathetic action. Electrocardiographic findings show a transient tachycardia of S.A. origin. As soon as blood sugar levels fall to a range of 13 to 35 mg. per cent, there is noted a marked parasympathetic action. This is manifested in the electrocardiogram findings with the slowing of the heart rate, a prolongation of the P.R. interval and partial heart block. A lowering and broadening of the T wave has also been noted. These changes are identical with those seen in anoxemia. If the vagi are cut above, these findings are not present.

The direct local effects of anoxemia and hypoglycemia on the coronary circulation is indicated by the following electrocardiographic findings: (1) inversion of the T wave; (2) changes in height and width of the T waves, and (3) changes in the R.S.T. segment.

Cutting of the vagi or stellate ganglionectomy does not alter these findings. If the hypoglycemia and anoxemia are not too prolonged, the findings disappear with the administration of glucose intravenously. Ephedrine acts on the myoneural junctions of the sympathetic nervous system, and mobilizes what sugar is already present and available in the tissues. The increase in the work of the heart consequent to the hypoglycemia and the accompanying reduced rate of oxidation seen in this condition furnish a rational explanation for the occasional precipitation of myocardial failure in cardiac patients under rapid insulin treatment (3, 7).

Studies on intracranial pressure during hypoglycemia show a consistent rise in intracranial pressure accompanied by an increase of blood pressure with a widening of pulse pressure. The results of these studies on intracranial pressure in hypoglycemia obtained on kymographic records are so similar to those obtained with anoxia that they point to a striking parallelism between these two conditions (4).

* From the Division of Anesthesia, Department of Surgery, New York Post-Graduate Medical School and Hospital, New York, N. Y.

† Now located at Deaconess Hospital, Evansville, Ind.

CASE REPORTS

Case 1.—A man, aged 44 years, was admitted to the hospital on October 30, 1941 with the chief complaint of pain in the right lower quadrant of the abdomen, the onset of which had occurred eight days previously. Past medical history disclosed diabetes for two years before admission. He had received insulin therapy for seven months but had not had any during the past year.

Temperature on admission was 103.4 F.; pulse was 96 per minute; respirations 24 per minute; blood pressure, 140 mm. systolic and 80 diastolic.

Laboratory studies on admission revealed blood glucose 200 mg. per cent, carbon dioxide combining power 42 volumes per cent; urinalysis: sugar 2.85 per cent, acetone 3 plus and diacetic acid negative. Hemoglobin was 82 per cent. Erythrocytes numbered 4,200,000; leukocytes 10,150, of which 63 per cent were polymorphonuclears, 23 per cent lymphocytes and 13 per cent monocytes. Diagnosis was appendiceal abscess and diabetes mellitus.

The following treatment was instituted:

10–30–41: 11 p.m. 1000 cc. N.S.—regular insulin units 25.
10–31–41: 1 a.m. 1000 cc. 5 per cent glucose in normal saline.
7:30 a.m. 1000 cc. of 5 per cent glucose in normal saline plus regular insulin, units 25.
8:00 a.m. Nembutal, grains 1½.
8:30 a.m. Morphine sulfate, grain ⅙ (0.01 Gm.), scopolamine, grain 1/150 (0.0005 Gm.).
9:00 a.m. Scheduled for operation at which time urinary sugar was 2½ per cent and acetone 3 plus. Because of urinary findings, operation was postponed.
9:30 a.m. 1000 cc. of 5 per cent glucose in normal saline plus regular insulin, units 40.
12:30 p.m. 1000 cc. of normal saline plus regular insulin, units 40. Urinalysis: sugar 2.85 per cent, acetone 3 plus.
2:00 p.m. Regular insulin, units 15.
2:45 p.m. Urine was negative for sugar and acetone.
3:30 p.m. Morphine sulfate, grain ⅙ (0.01 Gm.), scopolamine, grain 1/150 (0.0005 Gm.).
3:40 p.m. Regular insulin, units 10.
4:00 p.m. Temperature, pulse, and respiratory determinations were made. Urine was negative for sugar; there was a trace of acetone. Total glucose was 150 Gm. Total regular insulin, 155 units.
4:30 p.m. Operation.

It was noted that the patient was drowsy when he was brought to the operating room. Respirations were 20 per minute; depth of respiration was normal. Pulse rate was 80 per minute. Marked increase in spinal fluid pressure was noted on spinal puncture. He was given spinal anesthesia, 18 mg. of 1 per cent pontocaine solution plus 1 cc. of 10 per cent glucose. Ephedrine, 0.050 Gm. was given intramuscularly. Immediately following the injection the blood pressure was 200 mm. systolic and 90 mm. diastolic. Pulse rate was 76.

On the operating table the patient became gradually more stuporous. He could not be aroused and the eyeballs were moving slowly. The blood pressure continued to be elevated, gradually reaching the maximum of 240 mm. systolic

and 110 mm. diastolic, and the pulse became slower, 70 per minute. Respirations were regular, deep, at the rate of 20 per minute.

It was not thought that the patient's condition was the result of the spinal anesthesia. The general picture presented was not typical of morphine or scopolamine or barbiturate depression, and was not that of diabetic coma. A diagnosis of hypoglycemia was made. A blood sample for blood glucose and carbon dioxide combining power was taken, following which 50 per cent glucose was injected. Before 30 cc. had been given the patient was awake and responsive. In five minutes the systolic blood pressure fell to 180 mm. and then to 150 mm. Laboratory report on the blood taken before the injection of glucose showed a blood sugar of 26 mg. per cent and a carbon dioxide combining power of 52 volumes per cent. Postoperatively, the patient's recovery was uneventful and rapid. The temperature dropped immediately and he required very little insulin.

Case 2.—A man, aged 53 years, was a known diabetic, under control. He was admitted on May 7, 1942 for amputation of toe because of arteriosclerotic changes in his foot. Blood pressure at the time of admission was 155 mm. systolic and 90 mm. diastolic. Urine was negative for sugar. Two hours before operation he received 1000 cc. of 5 per cent glucose in normal saline solution intravenously and 25 units of regular insulin. Nembutal, grains 1½, was given also at this time. One hour later a hypodermic of morphine sulfate, grain ⅙ (0.01 Gm.) and scopolamine, grain $\frac{1}{150}$ (0.0005 Gm.) were given.

Fifteen minutes before operation the patient was seen in the anesthesia room where he responded to questions very slowly and was difficult to arouse. He was given spinal anesthesia (14 mg. of pontocaine hydrochloride in a 1 per cent solution and 0.7 cc. of 10 per cent glucose). The puncture was made at the fourth lumbar interspace. Spinal anesthesia was chosen for its therapeutic value as well as its anesthetic purpose. No ephedrine was given. Immediately after the spinal anesthetic was given, the patient lapsed into complete unconsciousness. Scattered twitchings of the muscles of the forearm and face occurred. Respirations assumed a type seen in air hunger. Blood pressure taken at this time was 220 mm. systolic and 100 mm. diastolic and the pulse was 90. Glucose determination at this time was later reported as 25 mg. per cent. At this point the patient was given 50 cc. of 50 per cent glucose intravenously. His eyes opened and he began to perspire freely. Another 50 cc. of 50 per cent glucose was given and the patient became entirely rational. Blood sugar taken at this time was later reported to be 220 mg. per cent and the carbon dioxide combining power 46.6 volumes per cent. The blood pressure fell to 160 mm. systolic and 80 mm. diastolic. The patient's subsequent course was uneventful. This patient's preoperative preparation had been deemed satisfactory by the medical department.

These cases present several interesting features. The typical picture of hypoglycemia—trembling, sweating, tachycardia—was not present. Joslin states that the symptoms of insulin shock depend upon the higher centers being intact, and therefore these symptoms are not seen under general anesthesia. He does not state what occurs under spinal anesthesia.

The dramatic response to administration of 50 per cent glucose was convincing and confirmed by laboratory reports. The signs of in-

creased intracranial pressure (increased spinal fluid pressure, increased blood pressure, with increased pulse pressure and relatively slow pulse) which made the picture different from that which is ordinarily seen in insulin shock, may be explained on the basis of temporary cerebral edema. The increase in blood pressure can also be explained on the output of epinephrine which occurs during one phase of insulin shock as a compensatory mechanism on the part of the body to mobilize what glucose is available in the liver and the tissues.

Cerebral edema may occur as the result of rapid insulinization (5), especially when the supply of glucose has not been sufficient for the body's need. The first patient had received a total of 5000 cc. of fluid, of which 3000 cc. was 5 per cent glucose in saline, and 2000 cc. normal saline solution. The insulin administered, 155 U., was considerably more than was needed to metabolize the glucose in the infusions. The blood sugar, although high, was not excessively so, and the history of this case shows that the patient did not have severe diabetes.

The possibility of cerebral damage on the basis of diabetic arteriosclerotic disease has been definitely ruled out by the subsequent course of the patients whose general condition has been excellent.

A few observations on insulin edema may be of interest. Irregular dilatation of cerebral vessels and interstitial edema has been demonstrated. It has also been demonstrated that there is a loss of intracellular and extracellular body water and electrolytes following the withdrawal of insulin therapy and the reverse of the process when insulin is resumed (6). Joslin (5), in his chapter on the treatment of hypoglycemic shock, recommended the use of hypertonic sodium chloride or sucrose to combat cerebral edema when lumbar puncture indicates its presence. He said also, "although many older surgical diabetics are relatively insensitive to insulin the danger of reaction must not be forgotten, especially if no food is taken and glucose solution is given by veins. Under these circumstances the total calories may be low and the effect of a moderate dose of insulin excessive. Often with energetic treatment insulin edema occurs, especially after acidosis, and in diabetes made severe by infection."

SUMMARY

Two case records of insulin shock during spinal anesthesia are presented. The main presenting symptoms were those of cerebral edema. Immediate and complete recovery ensued with administration of hypertonic glucose intravenously.

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CONSERVATION OF SCHOLARLY JOURNALS

The American Library Association created in 1941 the Committee on Aid to Libraries in War Areas, headed by John R. Russell. the Librarian of the University of Rochester. The Committee is faced with numerous serious problems and hopes that American scholars and scientists will be of considerable aid in the solution of one of these problems.

One of the most difficult tasks in library reconstruction after the first World War was that of completing foreign institutional sets of American scholarly, scientific, and technical periodicals. The attempt to avoid a duplication of that situation is now the concern of the Committee.

Many sets of journals will be broken by the financial inability of the institutions to renew subscriptions. As far as possible they will be completed from a stock of periodicals being purchased by the Committee. Many more will have been broken through mail difficulties and loss of shipments, while still other sets will have disappeared in the destruction of libraries. The size of the eventual demand is impossible to estimate, but requests received by the Committee already give evidence that it will be enormous.

With an imminent paper shortage attempts are being made to collect old periodicals for pulp. Fearing this possible reduction in the already limited supply of scholarly and scientific journals, the Committee hopes to enlist the cooperation of subscribers to this journal in preventing the sacrifice of this type of material to the pulp demand. It is scarcely necessary to mention the appreciation of foreign institutions and scholars for this activity.

Questions concerning the project or concerning the Committee's interest in particular periodicals should be directed to Dorothy J. Comins, Executive Assistant to the Committee on Aid to Libraries in War Areas, Library of Congress Annex, Study 251, Washington, 25, D. C.