murmur was noted at the point judged to be the apex of the heart, to the right of the sternum. This murmur was not transmitted to the back. For the first time, a distinct friction sound was heard during systole. The aortic and second pulmonic sounds were accentuated. The systolic blood pressure was 115; diastolic, 70; temperature, 98.6; pulse, 110; respiration, 25. There was an occasional dry cough. A roentgen-ray examination revealed a well defined shadow above and to the left of the cardiac shadow, which on succussion showed a well marked fluid wave, and which on changing position showed a changing fluid level. There was a shadow indicating pulmonary collapse of nearly all of the left lung. A diagnosis of pericarditis with effusion, complicated with pneumothorax and collapse of the major portion of the left lung was made. A blood Wassermann test was negative. A urine examination disclosed no abnormalities.

January 6, the patient was admitted to the Riverview Hospital; operation was done, January 7. Under local anesthesia, an L-shaped incision was made along the left sternal border and extending down the seventh rib on the left side for a distance of 2 inches. The skin was retracted, and after blocking the fifth, sixth and seventh intercostal nerves, a section 1 inch long of the sixth costal cartilage was removed, leaving a portion of its periosteum intact. The pericardium was opened, and 38 c.c. of clear fluid was aspirated. A small rubber tissue drain was left in the pericardial opening and the wound closed about it. An aspirating needle was then inserted in the seventh left intercostal space in the midaxillary line and approximately 500 c.c. of air aspirated from the pleural cavity. No fluid was encountered. On removal of the aspirating needle, the puncture wound was immediately sealed. Heavy gauze absorbent dressings were placed over the pericardial drain, and the patient was returned to bed. His general condition was good and he complained of but little discomfort attending the operative measures. The dressing pads were changed twice within the next six hours, in each instance being saturated with pericardial drainage fluid. At the time of the changing of the second dressing the patient complained of severe pain in the cardiac area. The position of the heart had changed so that the point of maximum intensity of the cardiac impulse was again evident at the left border of the sternum, and a rough friction sound could be heard without the aid of the stethoscope. Because of the pain, the drain was removed.

January 8, the patient felt better. There was a slight amount of drainage from the wound and he continued to improve. January 11, he was placed on forced breathing exercises by means of two Woulfe bottles containing fluid and connected by rubber tubing. By blowing the fluid from one bottle to the other the intrapulmonary pressure was raised and the lung forcibly expanded. These exercises were given for five minutes twice daily at first, but as he continued to improve the periods were lengthened and the intervals decreased. Except for a rather obstinate skin infection which developed at the site where the drain had been removed, his recovery was gradual and uneventful. The collapsed lung expanded gradually. The pericardial friction sound decreased in intensity. There was no pain in the cardiac area. Pleural friction sounds were well marked. March 12, the heart was in its normal position. The lung had fully expanded to the limit of the pleural cavity, and no râles could be detected. The patient felt fine. He had gained about 20 pounds (9 kg.) and was back at work. There was no cough, and the resected rib had apparently regenerated.

COMMENT

The pneumothorax in this case possibly resulted from the penetration of the pulmonary tissue and pleura by a tuberculous ulcer, and the consequent escape of air from an air vesicle or bronchiole into the pleural cavity. The collapse and resultant rest of the lung, incident to the equalization of the intrapulmonary and intrathoracic air pressure, permitted the opening in the visceral pleura to close and heal. Later, with the absorption of air from the pleural cavity and with the increased intrapulmonary pressure induced by forced breathing exercises, the lung once more expanded to its normal capacity. An unrecognized pericarditis with effusion occurring during the nineteenth year probably marked the beginning of the hydropericardium. The fluid accumulated so slowly that it was not until the complication of an accidental pneumothorax that the patient developed any marked cardiac distress.

The very serious nature of such cases and the great need of preventing any further embarrassment of either the respiratory or the circulatory system make the choice of an anesthetic for operative measures one of paramount importance.

I have found that local anesthesia, when properly given, is very satisfactory, whether the case is one requiring a simple rib resection or one of extensive lung decortication. The ease with which such cases may be handled and the freedom from distressing after-effects so often associated with ether or chloroform anesthesia make it the method of choice in the majority of surgical operations on the adult thorax.

HYPOGLYCEMIC SYMPTOMS PROVOKED BY REPEATED GLUCOSE INGESTION IN A CASE OF RENAL DIABETES *

R. B. GIBSON, PH.D., AND R. N. LARIMER, M.D., IOWA CITY

One of us (R. B. G.), in November last, reviewed the chemical findings in a case from our diabetic clinic before the Iowa Clinical Medical Society. Interest in the case centered in the fact that a final diagnosis of what was otherwise a case of pronounced renal diabetes could not be made because the sugar curve indicated a deficient glycogenesis of the mildly diabetic type. A study of the effects of a repeated ingestion of glucose on the sugar curve by Hamman and Hirschman[1] was recalled, and we predicted in our report that a decisive differentiation might be obtained if we employed the double sugar curve test in this case. The patient was requested to return for further observation, and promised to come to the clinic in January of this year.

We have in the clinic, at the present time, a second patient with a fasting hypoglycemia and a glycosuria of long standing which does not respond to diabetic management. The data presented in this communication were obtained in this case.

Glycogenesis is stimulated by glucose ingestion, as is indicated by the rapid fall of the blood sugar from the peak of the curve (usually forty-five minutes) to a figure at the end of two hours almost always less than the fasting control observation. A second administration of glucose brings about a yet more rapid removal of sugar from the blood stream and a consequent lowering of the sugar curve. The desugarized diabetic patient may show an effect similar to the normal person, but of much less degree (one case, Hamman and Hirschman). When tried in our case, the effect of the double sugar curve test was so great that hypoglycemic symptoms were observed in two out of three trials.

REPORT OF CASE

Mrs. B., aged 30, white, weight 110 pounds (50 kg.) (best weight 115 pounds [52 kg.] ten years ago) was admitted to the hospital with a history of glycosuria of ten years' standing. This had been discovered by a urine examination during the first of her two pregnancies. She had never had other symptoms of diabetes except for some pruritus seven years ago; she had dieted off and on since that time. The condition seemed to be familial, the patient stating that she had one sister surely and one probably glycosuric patients without other symptoms; however, she had no knowledge of glycosuria in either of her parents.

The patient was placed on a diet of 50 gm. of protein, 50 gm. of carbohydrate, and 125 gm. of fat; on this, she excreted from 4.5 to 8.5 gm. of glucose daily. Her blood uric acid was 3 mg., and blood urea nitrogen, 18 mg. Fasting blood sugar determinations or figures obtained two hours after meals were always hypoglycemic. The results of our tests with the double sugar curve are given in the accompanying table.

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* From the Chemical Research Laboratory of the Department of Theory and Practice of Medicine and Clinical Medicine, the State University of Iowa.

1. Hamman, L., and Hirschman, I. I.: Studies on Blood Sugar, IV, Effects upon the Blood Sugar of Repeated Ingestion of Glucose, Bull. Johns Hopkins Hosp. **30**: 306 (Oct.) 1919.

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Definite hypoglycemic symptoms were obtained in the first and third trials; they were identical with the several mild insulin reactions which we have observed in our diabetic patients, consisting of burning and flushing of the face, weakness, tremor and sweating. The second shock was the more severe of the two; the patient was completely relieved in fifteen minutes when given 100 c.c. of orange juice. The lowering of the level of the entire curve in the third trial is in accord with the experience that glycogenic effects may become more pronounced if the ordinary procedure is repeated without a sufficient number of days elapsing between tests.

When questioned as to the occurrence of similar attacks at home, the patient stated that she had experienced such of milder degree, but could not associate these with any definite circumstance. One sister had like attacks. It seems likely that hypoglycemic symptoms not artificially produced are a definite clinical entity.

Double Sugar Curve Tests

Time	Blood Sugar	Urine Volume	Urine Sugar, Gm.
December 15			
8:00	0.070	200	0.00
8:30	50 gm. glucose		
9:30	0.190	400	3.12
10:10	0.174		
10:45	0.074	Lost	
11:30	0.069	50 gm. glucose	
11:45	.....	400	3.05
12:15	0.140		
12:45	.....	265	3.58
1:15	0.113		
1:45	.....	230	5.33
2:15	0.062		
2:45	.....	145	1.03
3:30	Begining mild hypoglycemic reaction		
4:00	.....	100	0.20
7:00	.....	610	1.52
December 16			
9:00	0.091	185	Trace
9:05	50 gm. glucose		
9:30	0.143	145	2.13
10:30	0.212	255	4.71
11:30	0.143	290	4.58
11:45	50 gm. glucose		
12:15	0.138	114	4.22
1:15	0.129	240	8.28
2:15	0.097	140	3.59
3:15	0.075	10	0.11
4:15	0.079	360	2.02
December 20			
8:00	.....	235*	0.23
8:20	0.058		
8:30	50 gm. glucose		
9:00	0.097	585	0.53
9:30	0.118		
10:00	0.132	590	3.30
11:00	0.076	555	2.89
11:30	0.040	485	1.50
11:30	50 gm. glucose		
12:00	0.108		
12:30	.....	580	2.09
1:00	0.122		
1:30	.....	495	3.66
2:00	0.079		
2:30	.....	180	1.44
3:00	0.058		
3:15	Beginning hypoglycemic reaction of moderate severity		

* Fluids forced during test in another connection.

In explanation of a diminished glycogenesis in pronounced renal diabetes, it was stated, in the paper referred to above, that "It is quite possible that glycogenesis in our case may be functionally diminished because of the rapid removal of glucose through the kidneys; if so, repeated administration of glucose might so stimulate the glycogenic power that a normal or subnormal sugar curve will result."

Since this report was submitted for publication, threshold hypoglycemic symptoms with a blood sugar of 0.045 per cent. have been induced in our first patient; the maximum hypoglycemic effect is quite transient.

Team Work.—In the medical profession the value of team work has been recognized from the earliest times. When an engineer finds out a new process he patents it, so that none but him may use it. When a doctor makes a discovery it is the tradition of his profession that he should publish it to the world, so that all his colleagues and their patients may benefit thereby.—S. Hastings: *Lancet* **2**:776 (Oct. 6) 1923.

New and Nonofficial Remedies

THE FOLLOWING ADDITIONAL ARTICLES HAVE BEEN ACCEPTED AS CONFORMING TO THE RULES OF THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION FOR ADMISSION TO NEW AND NONOFFICIAL REMEDIES. A COPY OF THE RULES ON WHICH THE COUNCIL BASES ITS ACTION WILL BE SENT ON APPLICATION. W. A. PUCKNER, SECRETARY.

FLUMERIN-H. W. & D.—Disodium-2-hydroxymercuri-fluorescein, $NaOOC.C_6H_4.C.C_6H_2.HgOH.ONa.C_6H_3O$ (O bridge). The disodium salt of 2-hydroxymercurifluorescein.

Actions and Uses.—Flumerin-H. W. D., when injected intravenously, is tolerated by rabbits in doses containing from eight to twenty times the amount of mercury present in the therapeutic dose of other mercurial drugs commonly used. When injected into rabbits with syphilitic lesions, the drug brings about resolution of the lesions without apparent injury to the kidney.

According to the report of White, Hill, Moore and Young (Jour. A. M. A., Sept. 9, 1922, p. 879), flumerin injected intravenously in man in doses of 3 mg. per Kg. causes the disappearance of spirochetes from primary and secondary syphilitic lesions. According to these authors, flumerin will bring about resolution of the lesions and in about one-half the cases it will change a positive blood Wassermann reaction to negative. In tertiary syphilitic lesions, resolution of the lesions and disappearance of a positive Wassermann reaction was brought about in a majority of the cases treated by White, Hill, Moore and Young. Judging from urinary examination no permanent renal damage was done by the injection of the drug.

Dosage.—From 2 to 5 mg. per Kg. of body weight injected intravenously in 2 per cent. aqueous solution. Flumerin-H. W. & D. is incompatible with acids, halides and the salts of many alkaloids. Therefore water and not physiologic solution of sodium chloride is used to prepare solutions. If solutions be made with sterile water they may be used during three days if kept in well stoppered bottles in the dark.

Manufactured by Hynson, Westcott & Dunning, Baltimore. No U. S. patent or trademark.

Flumerin-H. W. & D. Tubes, 0.15 Gm.
Flumerin-H. W. & D. Tubes, 0.20 Gm.
Flumerin-H. W. & D. Tubes, 0.25 Gm.
Flumerin-H. W. & D. Tubes, 0.30 Gm.

Flumerin-H. W. & D. is a red, odorless powder; somewhat hygroscopic. It is soluble in hot water (about 1: 10); insoluble in alcohol, chloroform or ether. On incineration flumerin-H. W. & D. yields an ash containing sodium carbonate. Aqueous solutions of flumerin-H. W. & D. are deep orange-red in strong concentrations, but show a greenish-red fluorescence in very dilute solutions, the fluorescence being less intense than that of fluorescein in the same concentration. Aqueous solutions of flumerin-H. W. & D. are decomposed by acids, chlorides, bromides, iodides and the salts of many alkaloids, and local anesthetics.

Dissolve 1 Gm. of flumerin-H. W. & D. in 50 Cc. of water; no precipitate is produced when separate portions of this solution are treated with sodium hydroxide solution, potassium iodide solution and solution of blood serum. No precipitate is produced when a portion of the 2 per cent. aqueous solution is treated with freshly prepared ammonium sulphide solution (*absence of ionic mercury*), but on standing or on boiling, a suspension or precipitate of mercuric sulphide is produced. To a 2 per cent. solution of flumerin-H. W. & D. add one-half its volume of hydrochloric acid; a precipitate forms. Boil the mixture for one minute, cool, dilute, filter and add ammonium sulphide solution to the filtrate; mercuric sulphide is formed. Add 1 Cc. of a 1: 1,000 solution of flumerin-H. W. & D. to 1,000 Cc. of water; the solution shows a greenish-yellow fluorescence and the color is yellow by transmitted light. Add 1 Cc. of iodine solution and stir. An eosin-like color develops immediately and the solution is pink by transmitted light (*identification of the fluorescein nucleus*).

Determine the chloride content of flumerin-H. W. & D. by ignition with lime and precipitation of the chloride as silver chloride in the usual way. Not more than 0.2 per cent. of chlorine is found. (*Absence of excessive amounts of salts of chloromercurifluorescein.*)

Dissolve about 1 Gm. of flumerin-H. W. & D., accurately weighed, in about 50 Cc. of water at 50-60 C., filter through a weighed Gooch crucible, wash the residue thoroughly until the washings have only a slight color, dry at 110 C. and weigh. No weighable residue is found. Place about 0.2 Gm. of finely powdered flumerin-H. W. & D., accurately weighed, in an 800 Cc. Kjeldahl flask and slowly add 10 Cc. of sulphuric acid, in such a way as to wash down any adherent particles. Mix the materials carefully, heat to a temperature of from 60 to 75 C., remove the flame and add, little by little, finely powdered potassium permanganate, mixing thoroughly after each addition, until the presence of a considerable excess of brown manganese compounds is noted. The appearance of a slight flame after the addition of each portion of the oxidizing agent is immaterial. Cool the mixture to room temperature, add 100 Cc. of water, then gradually add powdered oxalic acid with shaking until the solution becomes clear. Filter if necessary, make the volume to about 200 Cc. with water, pass in hydrogen sulphide, collect the precipitate in a Gooch crucible, wash it successively with water containing hydrogen sulphide, alcohol, ether, carbon disulphide and ether, dry at 100 C. and weigh. The weight of mercuric sulphide corresponds to not less than 30 per cent. of mercury.

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