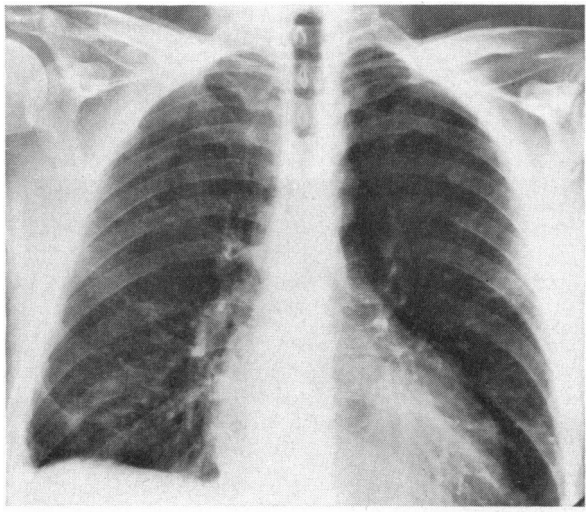

Figure 3.—Postoperative x-ray film showing normal appearance of the heart.

seen four months later the patient felt well. He volunteered that he was no longer short of breath, that he felt as well as he had before the attack in February, 1959, and was back at work.

DISCUSSION

A case of a rapidly developing and progressing left ventricular aneurysm during a four-month period after myocardial infarction is presented. The rapidity of growth of the aneurysm and the fact that the aneurysmal sac removed at operation was quite thin-walled, suggested that this patient would probably have died of rupture of the sac within a very short time. It is felt that all patients with left ventricular aneurysm following myocardial infarction should be operated upon if there is any evidence of heart failure or embolization. If neither of these findings is present, the patient should be observed closely with serial x-ray examination at least every three months, and if there is enlargement of the aneurysm, operation should be performed.

Excision of ventricular aneurysms with use of the heart-lung machine is believed to be better and safer than the closed technique. With the heart open, a more definitive procedure can be done and there is not the danger of embolization from the clot within the sac that there is with closed heart operations.

At last report the patient had returned to work.* He felt essentially the same as he did before the myocardial infarct. On physical examination a Grade II systolic murmur was heard to the left of the sternum at the third and fourth intercostal spaces.

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*May 23. The patient continues to work and do well.

Spontaneous Remission of Diabetes

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PERSONS who recover from diabetic acidosis usually need insulin or dietary restriction or both for the rest of their lives. Sustained remission or recovery is extremely rare either in children or adults. "Once a diabetic always a diabetic" is almost axiomatic.

The woman whose history follows has remained aglycosuric and normoglycemic, without taking insulin and with no restriction in diet, for six years.

REPORT OF A CASE

A 62-year-old widow was admitted to the Glendale Sanitarium and Hospital on the evening of February 11, 1954. Her husband had died suddenly late in 1953. On January 10, 1954, the patient became ill with what she thought was influenza. Her principal complaints were low-grade fever, malaise, insomnia and poor appetite. On February 1, 1954, she was given Aureomycin capsules. On February 7 she noticed that her mouth felt dry "like cotton." Her tongue was coated, and she was mildly nauseated and vomited once. She had several brief episodes of epigastric-retrosternal distress which she described as "angina." She stated that in the month preceding admission she had lost 16 pounds. Her weight on February 11, 1954, was 110 pounds. There was no significant family history.

The temperature was 99° F., the pulse rate 80 and blood pressure 170/90 mm. of mercury. The lips were dry and cracked, the tongue coated and the superficial vessels in the pharynx moderately engorged. There was no physical evidence of disease of the heart or lungs, although an electrocardiogram was interpreted as showing "lateral myocardial ischemia and left ventricular hypertrophy." The optic fundi appeared normal for a person of the stated age. There were no abnormal abdominal or neurological findings.

On routine urinalysis there was a 4 plus reaction for sugar and 2 plus for acetone. The hemoglobin was 11.4 grams per 100 cc. of blood, and leukocytes numbered 11,300 per cu. mm., 83 per cent neutrophils. Fasting blood sugar on February 12, 1954, was 610 mg. per 100 cc. The carbon dioxide combining power was 41 volumes per 100 cc. Nonprotein nitrogen was 30 mg. per 100 cc. of blood. The serum acetone reaction was 2 plus.

During the next four hours 225 units of crystalline insulin and two liters of 0.85 per cent sodium chloride were administered intravenously. After four hours the blood sugar was 62 mg. per 100 cc.

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and the carbon dioxide combining power 54 volumes per 100 cc.

The diet prescribed was 145 grams carbohydrate, 55 grams protein and 80 grams fat (1,500 calories). Ten units of crystalline insulin were given one-half hour before meals, with an additional 5 units for every plus over 2 plus (Clinitest).

On February 19 the fasting blood sugar was 309 mg. per 100 cc., the carbon dioxide combining power 57 volumes per 100 cc. Serum cholesterol was 345 mg. per 100 cc., and cholesterol esterified was 245 mg. per 100 cc. Cephalin flocculation was 1 plus. Thymol turbidity was 1 Maclagen unit. Serum protein was 5.5 grams per 100 cc. with normal albumin-globulin ratio. Beginning February 19, 30 units of NPH insulin was given before breakfast, with crystalline insulin before meals in amounts governed, as previously, by the Clinitest reaction.

An x-ray film of the chest February 19 was reported as showing "radiographically normal heart, lungs and pleura." Radiological studies of the gallbladder, colon and upper gastrointestinal tract showed no abnormalities except a single ovoid gallstone, nonopaque, measuring 15 mm. in its greatest diameter.

From March 3 to March 9 there was no glycosuria. The fasting blood sugar March 9 was 73 mg. per 100 cc. The NPH insulin dosage was gradually reduced—to 25 units on March 4, 20 units on March 6 and 10 units on March 9. No crystalline insulin was given after March 1.

On March 12 insulin was discontinued. The fasting blood sugar March 15 was 110 mg. per 100 cc. The patient left the hospital March 18, with no prescription of drugs and with moderate dietary restrictions (1,500 calories—80 grams protein, 2:1 carbohydrate). She weighed 120 pounds.

Fasting blood sugar values as determined in the office March 23, April 6 and May 5, 1954, were 80, 105 and 66 mg. per 100 cc. respectively.

The patient was put in hospital again from October 9 to October 11, 1954, because of fever, cough and "chest distress." No abnormalities were noted on physical examination or in results of laboratory tests. On October 9 urinalysis showed no sugar or albumin, and the fasting blood sugar was 89 mg. per 100 cc. An x-ray film of the chest showed no change since February 18, 1954. The diagnosis was acute upper respiratory infection. The patient said that she "had not paid much attention to the diet."

The patient diligently tested her urine by Clinitest and at no time found evidence of glycosuria. Nevertheless, she continued to worry about a possible recurrence of diabetes. On December 7, 1954, the blood sugar two hours after a "loaded" breakfast was 76 mg. per 100 cc. On February 15, 1955, the fasting blood sugar was 72 mg. per 100 cc., the nonprotein nitrogen 30 mg. per 100 cc., and the results of urinalysis within normal limits.

On January 23, 1956, the patient was admitted to the hospital with complaints of exhausting cough, chills, fever (102° F.) and pain in the right chest. Examination disclosed inspiratory rales and wheezing breath sounds in the right lower lung posteriorly. An x-ray film of the chest showed a small pneumonic infiltration in the right middle lobe. Urinalysis January 24 was within normal limits and fasting blood sugar was 92 mg. per 100 cc. The patient chose her own diet. She was dismissed February 7 with no medication and no dietary recommendations.

On May 17, 1957, the fasting blood sugar was 76 mg. per 100 cc. Results of urinalysis and blood cell count were within normal limits.

On July 27, 1958, the patient was admitted to hospital because of complaints of persistent pain in the left upper abdominal quadrant and in the left costovertebral area. An excretory urogram was reported as showing good bilateral kidney function, no abnormality of the kidney or ureteral outline, but a moderate cystocele. A barium enema study was carried out and no lesion was noted in the colon.

During this hospital stay the patient was encouraged to eat as she pleased. Since she had been doing this for two and a half years, it may be more correct to say that she continued to eat "what came naturally." A strict vegetarian, the patient presumably had a diet, in and out of the hospital, with a high carbohydrate content. On July 28 the results of urinalysis were within normal limits and fasting blood sugar was 97 mg. per 100 cc. The patient left the hospital July 30, 1958, asserting that she felt "better than I have in years." Her weight was 118 pounds.

From March, 1958, to the time of this report, the only drug she received was chlorothiazide (Diuril®) 250 mg., one tablet twice daily. Blood pressure as determined in the office from time to time, ranged from 120/80 to 160/90 mm. of mercury. There was no recurrence of the "anginal" symptoms. The serum cholesterol on September 26, 1958, was 256 mg. per 100 cc. Electrocardiograms continued to show left ventricular strain pattern but it was less pronounced than in 1954.

In December, 1959, the patient was admitted to hospital for influenza with low-grade fever. A glucose tolerance test at that time showed 93 mg. of glucose per 100 cc. of serum while fasting, 116 mg. per 100 cc. 30 minutes after ingesting 100 gm. of glucose and 170.6 mg. per 100 cc. after 60 minutes. There was no glycosuria. The blood urea nitrogen and albumin-globulin ratio were normal. Results of a liver "profile" (including serum bilirubin, serum alkaline phosphatase, thymol turbidity and

cephalin flocculation) were within normal limits. The protein-bound iodine was 4.4 micrograms per 100 cc.

The patient was again in hospital on January 4, 1960, with fever (102° F.), cough, chills, and girdle-type pain in the lower left thorax. Inspiratory rales were heard over the lower left lung posteriorly, and an x-ray film of the chest showed a small pneumonic infiltrate above the left diaphragm. Within a few days the typical eruption of herpes zoster appeared. No abnormalities were noted on examination of the blood and urine. The results of an Exton-Rose glucose tolerance test were essentially the same as those of December, 1959.

On February 7, 1960, the blood sugar two hours after breakfast was 107 mg. per 100 cc. An x-ray film showed almost complete resolution of the infiltrate in the left lung. No dietary restrictions had been ordered and the patient gained 10 pounds in weight during the month in the hospital. She requested a low-calorie diet because she "feels best" when her weight is 115 to 120 pounds.

DISCUSSION

That remissions can occur in insulin-deficient diabetes, usually by diet restriction and administration of insulin, is indisputable. Harwood,[3] in his recent review, expressed belief that the evidence also provides a convincing explanation of how they occur. He noted: Hyperglycemia, however produced, may stimulate the beta cells of the pancreatic islets to the point of exhaustion. The pancreas responds by hypertrophy of the islets and by formation of new islets from the acinar ducts. If the stimulus is too overwhelming or too prolonged, the islets will be irreversibly destroyed. However, if the stimulus is removed, and/or the hyperglycemia relieved, the production and normal tolerance for glucose are restored. In rare instances the stimulus is removed surgically by excision of diabetogenic tumors of the adrenal or thyroid glands. More commonly it is removed by administration of insulin and reduction of food intake.

In 1953 Cheng, Jahraus and Traut[1] reported the case of a woman, 68 years of age, who, after an episode of coma, extreme hyperglycemia and profound ketosis, apparently made a spontaneous and complete recovery. Five weeks after the episode of coma and ketosis, insulin was discontinued. However, two years and eight months after her first hospital stay she relapsed into a mild, chronic diabetic state. In a follow-up history of this patient, Traut[5] made brief mention of Harwood's "less dramatic report." He proposed no hypothesis of his own as to the modus operandi of spontaneous remissions from diabetes. He did not question the validity of the data on which Harwood bases his "explanation." He expressed belief that an "adequate explanation" of this phenomenon is not at present available, and apparently dismissed Harwood's theory on the rather vague grounds that "assumption of a primary disturbance in the islet physiology would be oversimplification."

Disturbance in the islet cell physiology must surely be involved. Traut[5] may be correct in stating that to assume a "primary" disturbance would be to oversimplify. But did Harwood[3] postulate a "primary" disturbance in islet cell physiology? To us it seems he did not. In Harwood's view the "primary" factor is in ". . . certain circumstances that produce hyperglycemia . . ."

Sometimes the "circumstances" seem obvious. Diabetogenic tumors disturb islet cell physiology, but the "primary" factor is the tumor, not what the tumor does to the islet cells. In Newburgh's[4] cases the primary factor was probably overeating and obesity. In the case reported by del Greco and Scapellato[2] the primary factors were probably infection plus overconsumption of carbohydrates: A 62-year-old man, recovering from pneumonia and a penicillin reaction, consumed 2 kg. of cane sugar and 3 kg. of honey within two days. Severe diabetes with acidosis quickly developed, although after treatment with insulin a remission occurred and continued for at least two years.

In the case reported by Harwood[3] no "primary factors" are discernible.

In the case herein reported the only "primary factors" admissible seem to be infection and emotional stress. Subsequent to the diabetic-acidotic episode the patient had several episodes of infection, five deserving hospitalization, with no detectable diabetogenic effect. The results of glucose tolerance tests in 1959 and 1960 indicate that she is a potential or "latent" diabetic. Nevertheless, in a period of six years, in spite of several serious infections, she had no clinical signs or symptoms of diabetes and required no treatment.

Might emotional stress serve as a "primary factor"? No definitive answer can be given. There are scattered references in the literature on the role of emotional upsets in producing hyperglycemia, but the evidence is fragmentary and inconclusive. In the case reported here, nothing more can be asserted than that emotional strain (following the sudden death of the husband) may have been one of the precipitating causes.

If Harwood's[3] "explanation" is correct or partially correct, why is not the remission phenomenon more commonly encountered? Harwood replies that (1) the beta cell apparatus in such persons is inherently weak and a certain amount of irreversible damage has occurred before hyperglycemia, glyco-

suria, etc., are detected. (2) Patients are notoriously unwilling or unable to submit to the rigors of a restricted diet. (3) Physicians and patients both fear provocation of attacks of hypoglycemia. Thus the majority of patients with severe diabetes are systematically undertreated. They are kept in a state of chronic mild diabetes, and this makes remission or amelioration impossible.

There may be another reason more difficult of proof. The "once a diabetic always a diabetic" attitude may have so greatly reduced awareness of the possibility of remission or recovery that physicians do not expect it, rarely attempt to induce it, and may not record it when it occurs.

Whether Harwood's explanation of the remission phenomenon is correct or not, his review makes the prognosis of diabetes less fatalistic. Diabetic patients, particularly "new diabetics," should be treated vigorously and hopefully. The possibility of remission should always be borne in mind.

SUMMARY

A 62-year-old woman was put in hospital in February, 1954, with hyperglycemia and mild acidosis. She was given insulin and intravenous fluids and a restricted diet was prescribed. Within four hours the laboratory signs of diabetic acidosis were reversed. Insulin dosage was gradually reduced and in four weeks was discontinued. She adhered to a mildly restricted diet till October, 1954. Since then she has not modified her food intake in any way except to reduce consumption of cholesterol-rich foods. Glucose tolerance tests indicate that she is still a latent diabetic. Nevertheless, in the past six years a number of severe infections, some requiring admittance to hospital, have not provoked signs or symptoms of diabetes.

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