glimpses of the road science has traversed to assist them in steering it wisely into the future.

There are many items of interest in the new Summarized Proceedings that are not mentioned in the Preface. For example, the registration and the numbers of papers presented at each meeting of the association since its founding are given, as well as the corresponding data for the Pacific and Southwestern divisions, except in a few cases in which the information is not available. For the first time the Summarized Proceedings contain in the report of each meeting, whether of the association or of one of the divisions, a complete list of the participating societies. Not only are they named, but also the number of papers on the program of each of them is given and all the symposia they organized or in which they participated. The number of papers in each symposium is given and the place of its publication, if published.

The advance sale of the new Proceedings has been so large, now about 4,200 copies, that it is not planned to print a number greatly in excess of those that have been sold. Since the printing will be from type that will be held for only a short time, instead of from plates, copies can be supplied only up to the number originally printed. The prepublication price to members of the association is $2.75 per copy, cloth-bound; after publication it will be $3.00. To those who are not members of the association it will be $4.00 per copy. The pages are 6 by 9 inches.

Table 2 gives comparative data for the preceding four volumes of the Summarized Proceedings and for the one now in press.

TABLE 2

PERIOD	COPIES PRINTED	UNIT PRICE	TOTAL RECEIPTS	TOTAL COST
1915–20	2,262	$1.00	$ 2,794	$ 5,430
1921–24	4,500	2.00	6,390	9,297
1925–28	4,250	3.50	11,060	11,060
1929–33	4,000	2.50	8,001	11,404
1934–39	4,500(?)	2.75	11,550	9,800(?)

The estimated cost for the volume in press includes printing, binding, cartons, mailing, postage, clerical help and circularization. The total deficit of the four Summarized Proceedings published from 1915 to 1933 inclusive was $8,886, or an average per issue of over $2,200. As a consequence of these deficits the office of the Permanent Secretary has been setting up reserves of about $1,000 per year to cover the prospective deficit on the present volume. Apparently the reserve may be held intact for the next volume.

F. R. MOULTON,
Permanent Secretary

SPECIAL ARTICLES

MORPHOLOGICAL AND FUNCTIONAL RECOVERY OF THE PANCREATIC ISLANDS IN DIABETIC CATS TREATED WITH INSULIN

PERMANENT diabetes has been produced in the normal dog by the injection for a few weeks of a crude saline extract of anterior pituitary glands.[1,2] Young[1] was not able to make normal cats permanently diabetic; nor were we in similar experiments. However, if one half to three fourths of the pancreas is removed, leaving enough to prevent spontaneous hyperglycemia and glycosuria, it is possible to make such cats permanently diabetic by a course of injections of anterior pituitary extract. Fifteen such animals have been prepared. The diabetes has persisted throughout the period of observation, which in some instances has been as long as five months after the last injection of anterior pituitary extract. Some of the permanently diabetic cats have been treated with insulin in an attempt to control the hyperglycemia. In five of these animals insulin was stopped after varying periods of freedom from glycosuria and hyperglycemia. In these five animals (see Table I) the withdrawal of insulin was not followed by a return of glycosuria, and the blood glucose was within normal

TABLE I

DIABETIC CATS RECOVERING AFTER INSULIN TREATMENT

Cat No.	Glycosuria: During injection of ant. pit. extract	Glycosuria: Interval between ant. pit. extract and insulin	Severity of diabetes*	Duration of insulin therapy	Duration of recovery†
	days	days	per cent.	days	days
R– 3	43	29	60	24	105
R– 5	18	15	64	27	56
R–10	25	56	43	20	100
R–12	10	12	63	32	113
R–21	13	6	66	9	36

* Expressed as the percentage of the calculated available glucose of the diet (21.6 gm/day) excreted in the urine for 6 days prior to insulin therapy. For R–21 the previous 2 days were used.
† Non-diabetic period from cessation of insulin therapy to end of experiment.

[1] F. G. Young, *Biochem. Jour.*, 32: 513, 1938.
[2] F. C. Dohan and F. D. W. Lukens, *Am. Jour. Physiol.*, 125: 188, 1939.

limits before and after feeding. The diet, with one minor exception, was constant. The daily rations consisted of 200 grams of beef heart supplemented with cod-liver oil, yeast and bone ash. This recovery from the previous diabetic state has been maintained to the termination of the experiment, a period of more than three months in several of the animals. The cats continued to gain weight throughout this period.

Biopsy specimens of the pancreas of permanently diabetic cats taken *before* treatment with insulin show marked hydropic degeneration of the islands of Langerhans. In cats exhibiting functional recovery *following* insulin therapy the islands are histologically normal. Some animals did not show "permanent" recovery with insulin treatment. These instances were associated with infections, poor control of the diabetes by insulin or institution of insulin treatment after more than five months of diabetes.

In dogs made diabetic by removal of about nine tenths of the pancreas hydropic degeneration of the islands is present for the first few months.[3] Using such dogs, Copp and Barclay[4] have observed morphological restoration of the islands *during* periods of insulin therapy. Despite the morphological improvement it was necessary to continue the administration of insulin. Since the diabetes had been produced by partial pancreatectomy alone, it was not to be expected that morphological restoration of the remaining islands would maintain functional recovery. We have failed to obtain morphological (or functional) recovery in dogs made permanently diabetic with anterior pituitary extract. This we attribute to the early development of atrophy of the islands of Langerhans in our dogs, in contrast to the hydropic degeneration found in the experiments of Copp and Barclay and in our cats. However, it has recently been demonstrated in dogs that the concurrent administration of insulin may hinder the fall in the insulin content of the pancreas and the hydropic degeneration of the islands which occurs during the period of injection of certain anterior pituitary extracts.[5]

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THE NEURO-MOTOR MECHANISM OF THE SMALL BLOOD VESSELS OF THE FROG

THE mechanism which regulates capillary blood flow has not been definitely established. In Krogh's laboratory, Vimtrup[1] reported the contraction of Rouget cells in Amphibia, which caused folding of the endothelium. Field,[2] using the frog and rat, and also Beecher,[3] using the rabbit's ear, confirmed Vimtrup and in addition observed the swelling of endothelial nuclei which blocked the lumen of the capillary. The Clarks[4] reported endothelium to be contractile in the tadpole's tail but not in the rabbit's ear. They deny the contractility of extra-endothelial cells (Rouget cells) in mammals and amphibians, with the possible exception of the nictitating and hyaloid membranes of the frog. Zweifach[5, 6] reported endothelial contractility in the frog and mouse. In response to mechanical stimulation, contraction of the endothelium in the frog "completely closed the lumen of the vessel only at its ends." At the "capillary exit in those regions where the capillary offshoot leaves the arteriole," he observed valve-like folds of endothelium which opened and closed passively with dilatation and constriction of the arteriole. In the mammal Zweifach[7] comes to the conclusion that the contractility of capillary endothelium has little physiological significance.

We have examined, by means of stimulation with a micro-electrode, the distribution of the contractile elements of the small blood vessels in the retrolingual membrane of the frog. In contrast to the uniform layer of the typical smooth muscle of the arteriole, and the somewhat scattered arrangement on the precapillary, the modified smooth muscle cells of the capillary are confined to the region of its origin. If the capillary branches before emptying into a venule, the branches are devoid of smooth muscle cells and do not contract. The region of the capillary origin may act as a unit with its adjacent blood vessels, but frequently it acts independently of them as a sphincter-like mechanism. This concept of the control of capillary blood flow is supported by the evidence presented below.

In the frog with brain and medulla pithed, the retrolingual membrane was prepared for illumination by transmitted light, after the method of Pratt and Reid.[8] A micro-electrode, 1–5 micra, was placed in the field by an Emerson micromanipulator. Cinephotomicrographs were obtained, using a light-splitting prism.

Brief faradic stimulation of the small vasomotor nerves produced dilatation of the small blood vessels, followed by constriction. Weak stimulation usually

[1] Bj. Vimtrup, *Zeitsch. f. d. ges. Anat.*, 65: 150–182, 1922.
[2] M. E. Field, *Skand. Arch. f. Physiol.*, 72: 175–191, 1935.
[3] H. K. Beecher, *Skand. Arch. f. Physiol.*, 73: 1–6, 1936.
[4] E. R. Clark and E. L. Clark, *Am. Jour. Anat.*, 66: 1–49, 1940.
[5] B. W. Zweifach, *Anat. Rec.*, 59: 83–108, 1934.
[6] B. W. Zweifach, *Anat. Rec.*, 73: 475–495, 1939.
[7] B. W. Zweifach, *Am. Jour. Physiol.*, 120: 23–35, 1937.
[8] F. H. Pratt and M. A. Reid, SCIENCE, 72: 431–433, 1930.

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[4] E. F. F. Copp and A. J. Barclay, *Jour. Metab. Res.*, 4: 445, 1923.
[5] J. Campbell, R. E. Haist, A. W. Ham and C. H. Best, *Am. Jour. Physiol.*, 129: P328, 1940 .