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GLYCOGEN METABOLISM IN THE RAT AFTER PARTIAL HEPATECTOMY.

by

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In the present work, an attempt has been made to study the metabolic phenomena that occur in partial hepatectomy, the rate and extent of carbohydrate changes in the liver and muscles due to various agents being investigated after recovery from the operation. The present observations are chiefly devoted to the glycogen content, with incidental notes on the blood sugar level and of the hexose phosphate of muscle.

The method of partial hepatectomy has been chiefly elaborated by Rous and Drury (1925), Drury, Eleman, & McMaster (1927) and McMaster and Drury (1929), who found that in rabbits, as much as 90 per cent. of the liver could be removed without fatal consequences. In rats, the tolerance is considerably lower, and the mortality rate rose considerably if more than 50 per cent. was removed. Since satisfactory results can be obtained with removal of 33 per cent. and upwards, no attempt has been made to obtain the maximum possible reduction of the liver. The curtailment is always greater than the figures actually given for the weight removed, since the complications of fat infiltration, adhesions and necrosis always occur to some extent.

METHODS.

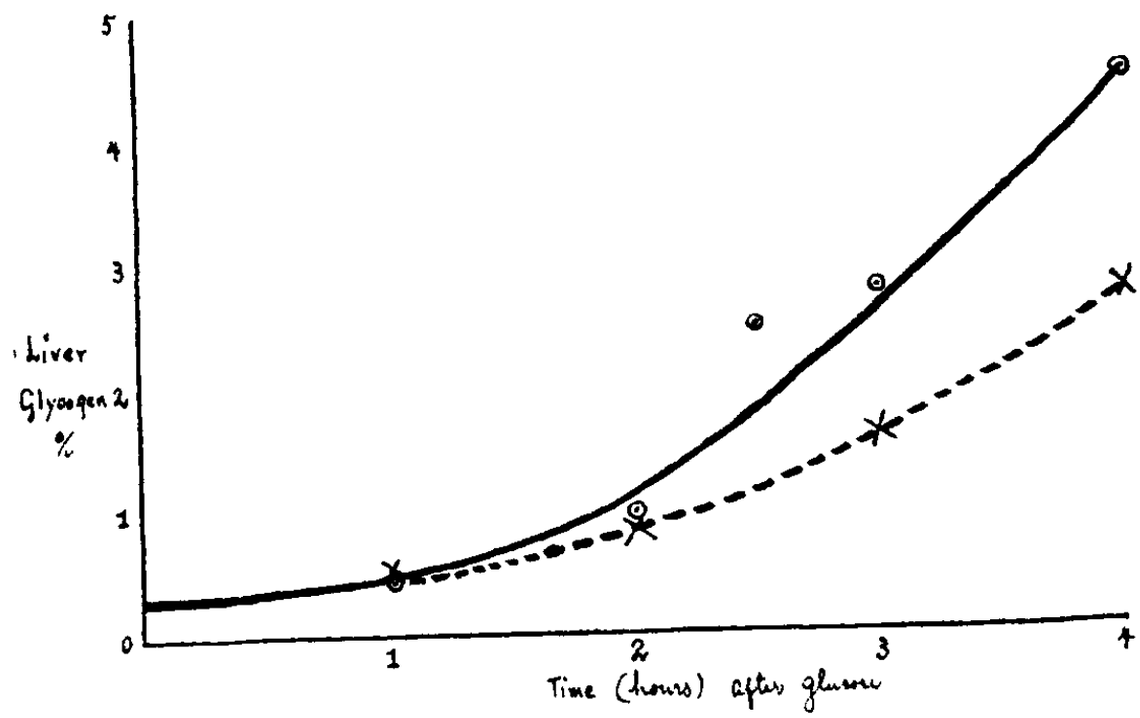
The technique of the operation finally adopted was as follows: the rat, preferably weighing between 100-150 grams, is anaesthetised with veronal and ether, or ether alone, opened ventrally along the mid line, the liver presented by gentle pressure on the abdomen, and two or more of the main lobes firmly ligatured with fine catgut near their bases (taking care not to involve the vena cava or the main hepatic vessels). These lobes are removed and weighed, and amount on an average to 40 to 50 per cent. of the whole liver. The survival rate is about 80 per cent. After 10-14 days the animal appears normal and can be used for glycogen determination.

Glycogen was determined by the method outlined by Evans Tsai and Young (1931), hexose phosphate in muscle by the method described by Cori & Cori (1933), blood sugar by the Hagedorn-Jensen method, and phosphates by the method of Robison.

1. Changes in liver and muscle glycogen after a glucose meal.

Hepatectomised rats, starved for 24 hours previously, were given 2 c.c. of 50 per cent. glucose by stomach tube. After a period varying from one to three hours they were killed by decapitation and the liver and muscles sampled. A number of control animals were treated in the same way. A small number of determinations of the glycogen level in fasting rats, control and hepatectomised, were also performed. In some instances, determinations of the hexose phosphate ratios in

Fig 1



muscle before and after stimulation were made, according to Cori's technique: the gastrocnemius from the leg was quickly dissected out and placed in trichloracetic acid solution at 0° C while the opposite gastrocnemius was given 15 seconds maximum stimulation from an induction coil and then treated similarly. The variations in hexose and phosphate were large, but fell within the limits assigned by Cori; no satisfactory differentiation between the hepatectomised and control groups was established.

Table I gives the details and Fig. I a graph of these experiments.

In order to express the results in terms of percentage residual

TABLE 1.

Effect of Glucose Meal on Hepatectomised

DOSE:—2 c.c. 50% Glucose per 100 gm. I

No. of rat.	*Wt. (gm)*	*hepat-ectomy %*	*Wt. of liver when killed (gm)*	*Time after sugar (hrs)*	*Muscle Glycogen%*			
					1	*2*	*Av*	*1*
11	100	45	2.5	1				.75
14	120	43	3.8	1				.39
23	130	48	4.4	1				.58
38	110	40	3.3	1				.62
1	112	33	5.3	2	.41	.38	.40	.39
3	78	40	4.0	2	.42	.40	.41	.81
4	82	38	2.5	2	.53	.57	.55	1.28
5	92	35	3.3	2	.50	.42	.46	1.08
16	130	48	5.2	2	.31	.30	.31	1.30
21	139	42	5.4	2	.33	.32	.33	1.10
170	160	35	5.9	2½				2.02
171	130	38	4.4	2½				2.00
172	140	48	6.3	2½				3.23
173	140	44	5.0	2½				3.11
174	140	40	5.5	2½				2.13
180	100	34	3.9	3	.56	.50	.53	2.11
181	90	39	3.7	3	.63	.60	.62	3.80
183	85	44	4.2	3	.38	.38	.38	1.90
184	130	40	6.0	3	.45	.40	.43	2.30
185	120	41	6.1	3	.41	.48	.45	2.90
61	130	44	5.4	3	.40	.39	.40	2.10
62	100	38	5.0	3	.38	.30	.34	2.80
63	120	42	4.9	3	.61	.68	.65	3.40
64	120	45	5.8	3	.62	.63	.63	3.12
65	110	45	5.0	3	.69	.60	.65	3.80
20	160	38	5.6	4	.56	.72	.64	2.76
59	105	45	4.1	4	.28	.24	.26	4.30
14	125	33	4.3	4	.89	.80	.85	2.50
44	120	36	4.4	4	.76	.70	.73	3.90
46	140	40	5.3	4	.68	.64	.66	4.30
47	135	42	4.9	4	.63	.60	.62	4.10

Average Glycogen in 10 hepatectomised rats starv
Muscle — do. — : 0.23 gm/100 gm Liv
Liver Glycogen: 0.12 gm/100 gm Mu

Rats.

ɪat.

Liver Glycogen% 2	Av	*Total Glycogen in liver gm.*	*Blood Sugar mg per 100 gm*
.83	.79	0.2	
.40	.40	0.15	
.63	.61	0.26	
.63	.63	0.21	
.39	.39	0.21	
.83	.83	0.33	
1.28	1.28	0.59	
1.00	1.04	0.34	
1.20	1.25	0.65	
1.12	1.11	0.60	
2.13	2.08	1.20	
1.93	1.97	0.87	
3.00	3.12	1.96	
3.16	3.14	1.57	
2.18	2.15	1.18	
2.21	2.16	0.84	
3.55	3.72	1.38	
1.51	1.70	.72	
2.62	2.46	1.48	
2.60	2.75	1.68	
2.20	2.15	1.16	
2.35	2.65	1.32	
3.22	3.31	1.62	
3.30	3.21	1.86	
3.65	3.72	1.86	
2.70	2.73	1.53	
4.80	4.60	1.88	172
2.70	2.60	1.12	126
4.10	4.00	1.76	138
4.00	4.15	2.16	150
4.20	4.20	2.96	144

ɾed 24 hrs.
ver.
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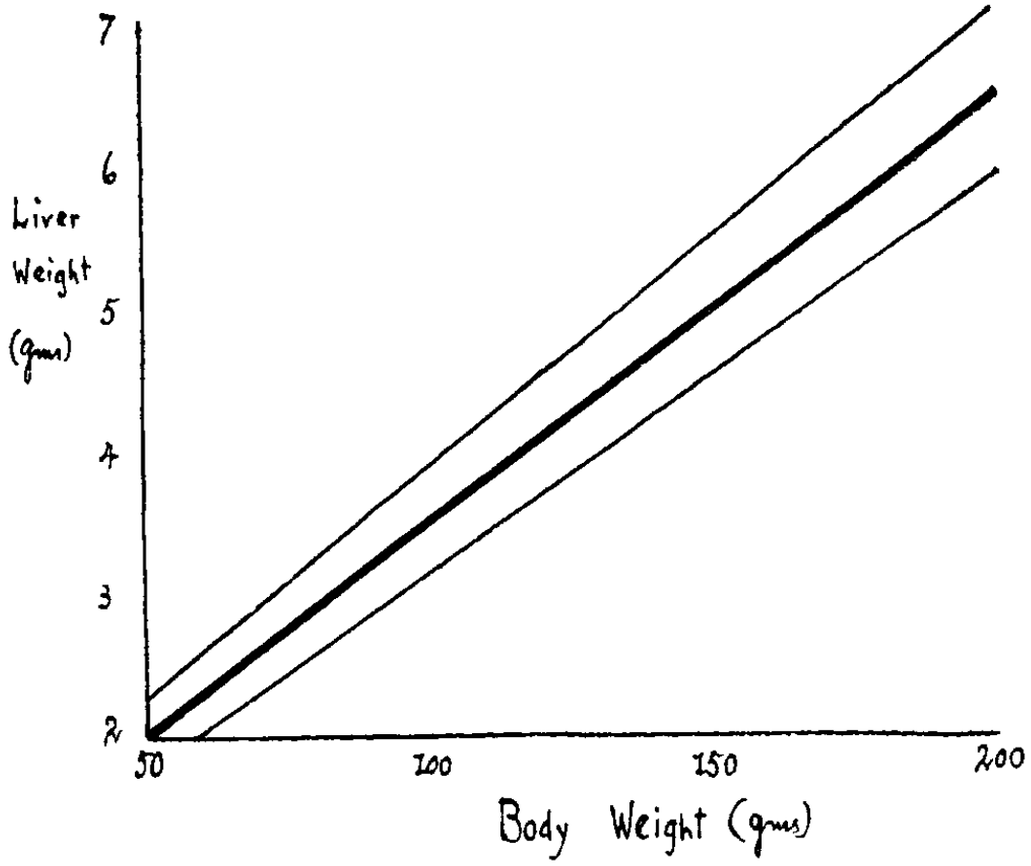
liver in the case of the hepatectomised group, it was found necessary to investigate the correlation between weight of liver and body weight at various stages of growth. Fig. 2 shows this relation graphically, and is based on a sample of about seventy rats of various weights taken from the stock colony. Though all these animals were apparently normal, the individual variations are considerable. The limits of reasonable variation are enclosed by the upper and lower lines and the median line has been accepted as the average liver weight for any particular body weight. It is approximately a straight line over the whole range, and though only a rough estimate, may be accepted for the particular end in view.

Fig 2. Relation between Body Weight and Liver Weight in Stock Black and White Rats



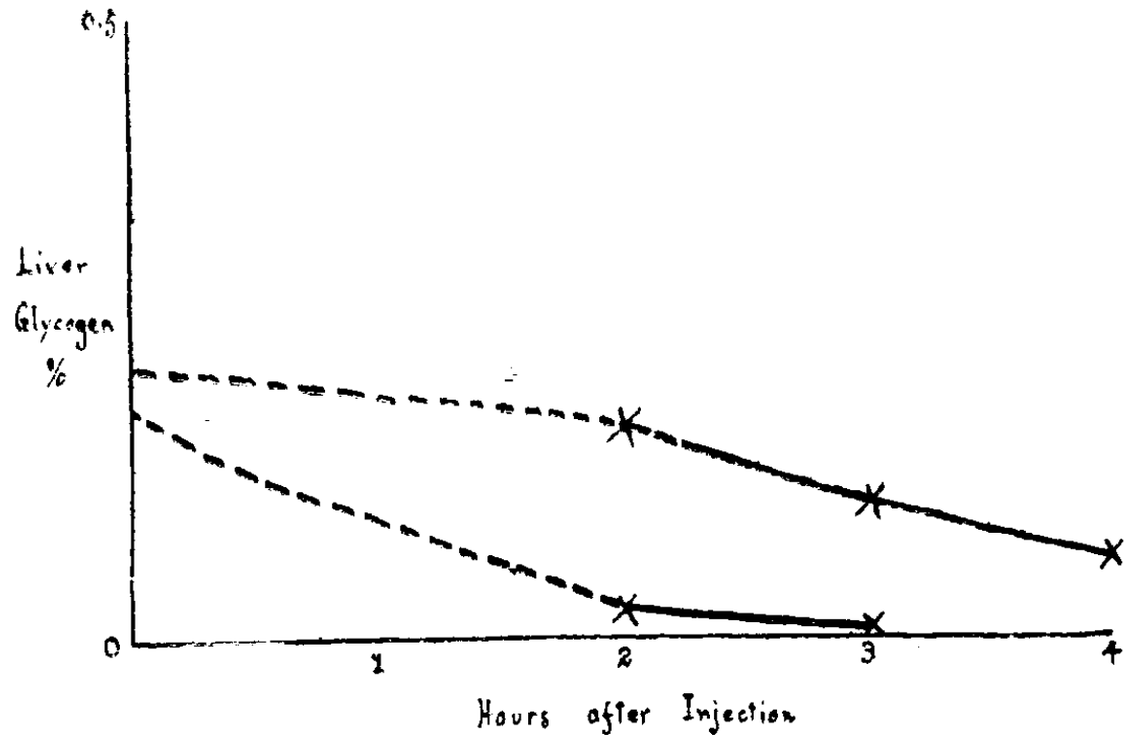
This correlation for the hepatectomised group of rats was only to be obtained in this indirect manner, since the weight of liver removed at operation was not complementary to the weight of the liver found at death because (a) the animal increased its body weight (b) an abnormal increase of the residual liver occurred, during the intervening period. The latter phenomenon, sometimes described as the "regeneration" of the liver following hepatectomy, appears rather to be, in the experiments here described, in the nature of a hypertrophic change. The liver underwent a marked change in macroscopic appearance: it became pale, friable and engorged with fat, judging from

the large amounts of fatty acids set free by the action of the potash in the glycogen determination, which was notably greater than in the controls.

2. *Changes in the liver and muscle glycogen of hepatectomised rats due to Insulin.*

Thirty hepatectomised rats and a smaller number of control rats were given insulin injections after fasting for 24 hours. A dose of 0.5 units per 100 gm body weight was found to cause convulsions even in the control group. With 0.3 units per 100 gm. a marked differential result was obtained between the two groups. The majority of the hepatectomised animals underwent typical hypoglycaemic convulsions, and several died, while only a few of the control animals had convulsions, and no deaths occurred. The details of these experiments are set out in Table 2, and graphically in Fig. 3.

Insulin Effect on Liver Glycogen Fig 3.



3. *Effect of adrenalin upon liver and muscle glycogen of hepatectomised rats.*

Thirty hepatectomised animals, and fifteen control animals, fasted for 24 hours, were used. They were given injections of adrenalin at the rate of 0.04 mg. per 100 gm. body weight, synthetic adrenalin (Ciba brand) being used. This dosage is comparable with that of Cori and Cori (1928) where natural adrenalin (Parke, Davis) appears to have been used, at the rate of 0.02 mg. per 100 gm., and their

TABLE 2.

Effect of Insulin on Hepatectomised Rats (fed, or starved 12 or 24 hours).

No. of rat.	*body weight (gm)*	*estimated hepatectomy %*	*total liver wt. (gm)*	*killed after Insulin (hours)*	*Muscle Glycogen %*			*Liver Glycogen %*			*Blood Sugar %*
					1	*2*	*Av.*	*1*	*2*	*Av.*	
1	2	3	4	5	6	7	8	9	10	11	12
A. *Insulin dosage: 0.2 unit per 100 gm.—starved 24 hours.*											
19	115	43	3.5	3	.26	.26	.26	.16	.14	.15	60
64	75	52	2.3	3	.24	.31	.28	.07	.04	.05	42
69	95	40	3.7	3	.12	.15	.14	.16	.13	.15	73
66	85	41	3.6	3	.24	.29	.27	.12	.11	.12	62
B. *Same dose and starvation period as A*											
961	120	45	5.1	4	.13	.07	.10	.04	.02	.03	40
21	155	38	5.5	4	.06	.06	.06	.02	.02	.02	45
306	85	41	3.3	4	.04	—	.04	.09	.10	.10	62
169	95	40	3.4	4	.13	.17	.15	.10	.19	.15	80
106	80	48	3.0	4	.18	—	.18	.02	.02	.02	38
C. Insulin dosage: 0.5 unit per 100 gm— — do. — (2 others in this Group died in 3½ hrs.)											
308	120	41	3.8	2	.15	.15	.15	0.0	0.0	0.0	33
301	75	45	2.9	2	.21	.14	.17	0.0	0.0	0.0	40
304	90	48	2.8	2	.31	.30	.31	0.0	0.0	0.0	44
305	80	32	4.8	2	.29	.25	.07	.07	.07	.07	39
307	105	30	4.9	2	.11	.08	.10	0.0	0.0	0.0	38
D. Insulin dosage: same as C—not starved. (3 others in this group died)											
100	120	40	3.9	3	.51	.56	.54	.12	.11	.12	55
101	100	35	3.3	3	.55	.58	.57	.23	.18	.21	60
102	130	35	5.1	3	.22	.19	.21	.08	.04	.06	45
103	130	48	4.4	3	.43	.43	.43	.66	.60	.63	80
104	160	45	4.6	3	.30	.39	.35	.35	.30	.33	63
105	110	40	4.0	3	.46	.40	.43	.28	.22	.25	72
120	130	40	5.0	3	.68	.62	.65	.19	.25	.22	—
121	130	45	5.2	3	.45	.42	.44	.31	.38	.34	—

122	130	48	5.0	3	.52	.48	.50	.20	.20	.20	—
123	110	40	3.8	3	.33	.37	.35	.31	.22	.27	—
124	100	35	4.1	3	.70	.73	.72	.86	.70	.78	—
125	100	39	4.0	3	.38	.38	.38	.09	.14	.12	—

TABLE 2—*Continued*

1	2	3	4	5	6	7	7	9	10	11	12
E. *Insulin dosage: same as C—Starved 12 hours.*											
290	175	43	5.8	3	.26	.26	.26	.10	.16	.13	48
909	175	38	5.1	3	.34	.40	.37	.44	.50	.47	62
333	135	36	4.6	3	.26	.28	.27	.51	.58	.55	70

A. Insulin dosage 0.2 unit per 100 gm. Starved 24 hours.
B. — do. — — do. —
C. 0.5 unit per 100 gm. — do. —
D. — do. — Not starved.
E. — do. — Starved 12 hours.

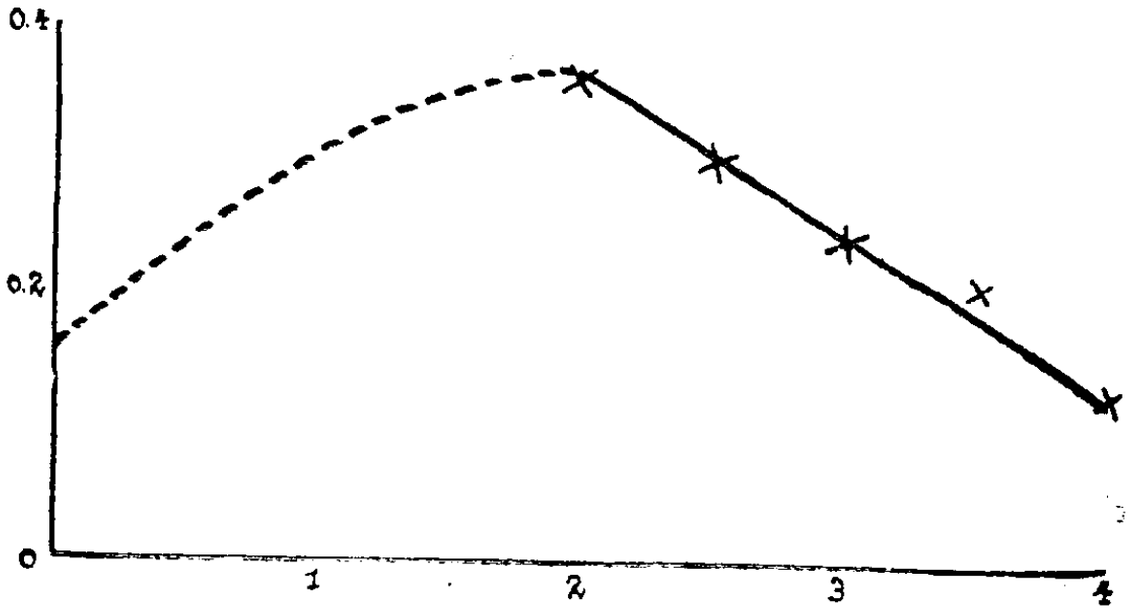
TABLE 2A.

1	2	3	4	5	6	7	8	9	10	11
A.										
C.1	100	4.3	3	.40	.47	.44	.62	.74	.68	68
C.2	130	5.2	3	.18	.22	.20	.58	.67	.62	82
C.23	70	3.2	3	.39	.37	.38	.68	.87	.78	71
C.3	120	3.9	3	.19	.24	.22	.59	.45	.52	69
B.										
C.417	100	4.1	3	.52	.59	.56	.14	.18	.16	44
C.422	120	5.2	3	.49	.48	.49	.20	.14	.17	52
C.415	95	4.4	3	.59	.55	.57	.12	.10	.11	—
C.19	90	3.9	3	.48	.46	.47	.08	.12	.10	40
C.										
C.440	170	6.2	3	.62	.55	.59	.10	.15	.13	40
C.442	130	4.3	3	.44	.54	.49	.20	.12	.16	53
C.18	100	4.1	3	.50	.44	.47	.14	.12	.13	48

A. Insulin dosage: 0.2 per 100 gm. Not starved
B. 0.5 — „ — Starved 12 hours.
C. 0.5 — „ — Not starved.

results were introduced for comparison. The animals were killed at 1, 2 or 3 hours after injection, the majority after the longest intervals. The results are set out in table 3 and graphically in Figure 4.

Effect of Adrenalin on Liver Glycogen Fig 4.
Hepatectomised (and control) rats



DISCUSSION.

The effect of partial hepatectomy is apparently to embarrass the liver function to an extent roughly proportional to the severity of the operation. The effect is well marked in the case of rats given a glucose meal; as compared with control animals, they show a considerable increase in the percentage of glycogen stored in the liver when the absorptive state is well advanced, and the divergency increases steadily up to four hours, the longest period tested. (At this time a large amount of the glucose administered still remains to be absorbed). If the relative sizes of the livers are taken into consideration, however, it is found that the total glycogen formation is not markedly different in the two groups. The liver glycogen curves also suggest that the formation of glycogen is a process which increases cumulatively with time, and that the hepatectomised animal is at no advantage over the control in this velocity of formation—indeed that its metabolic function is in no way expedited by reducing the effective mass of the liver.

In the case of rats receiving insulin or adrenalin, a less marked differential effect is shown. This is possibly due to the low level

of glycogen in the fasting rat, which causes small absolute differences to appear relatively great. Assuming that the level of liver glycogen in the hepatectomised and control rat are identical, the effects may be attributed solely to the diminished store of reserve glycogen available in the former. The death of a number of the hepatectomised animals from insulin convulsions at a moderate level of dosage, which the control rats could survive without severe symptoms, tends to support this view. It would seem likely that the partially hepatectomised rat may be a suitable method for studying the problem of glycogen metabolism in the liver, and further experiments have been planned for this purpose.

SUMMARY.

1. Rats partially hepatectomised show a greater percentage glycogen level in the liver after a glucose meal, but the total glycogen stored is comparable with that stored in the normal animal.

2. Injection of insulin causes more rapid and extensive glycogenolysis than in the normal animal.

3. With adrenalin the change in blood sugar level and glycogenolysis is greatly increased by partial hepatectomy.

I desire to express my thanks to Prof. C. Lovatt Evans for facilities of his laboratory and his continued interest in this work, and to Dr. H. P. Gilding for a demonstration of the Rous hepatectomy technique.

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TABLE 3.

Effect of Adrenalin on Glycogen Storage of Hepatec

DOSAGE: 0.4 mg. adrenalin (synthetic: Ciba brand) per

No. of rat	*Body wt. (gm).*	*Hepatectomy (estimated) %*	*Total liver wt. (gm)*	*Killed after adrenalin (hrs)*	*Muscle Glycogen %*		
					1	*2*	*Av.*
1	2	3	4	5	6	7	8
97	85	32	3.4	2	.15	.15	.15
96	105	40	4.1	2	.18	.08	.13
34	100	48	3.8	2	.12	.14	.13
8	65	35	2.5	2	.23	.27	.25
6	115	42	4.4	2	.16	.15	.16
9	105	33	4.0	2	.23	.26	.25
29	80	35	3.2	2	.22	.23	.23
19	75	38	3.4	2	.20	.19	.20
3	100	30	4.0	2	.19	.19	.19
18	165	32	6.2	2	.26	.27	.27
700	92	38	3.5	$2\frac{1}{2}$	.28	.21	.25
701	98	30	3.4	$2\frac{1}{2}$	.04	.04	.04
702	95	36	5.3	$2\frac{1}{2}$	0.0	.06	.03
610	90	33	3.5	3	.22	.18	.20
611	140	48	6.0	3	.09	.07	.08
612	120	40	5.0	3	.18	.20	.19
615	120	42	4.4	3	.33	.30	.32
616	130	50	4.8	3	0.0	0.0	0.0
617	100	53	4.0	3	.02	.08	.05
618	100	39	3.8	3	.20	—	.20
619	150	30	5.9	3	.16	.10	.13
620	160	43	5.3	3	.22		.22
623	110	40	3.8	3	.25	.28	.27
625	100	48	4.2	3	.18	.10	.14

TABLE 3—*Continued*

1	2	3	4	5	6	7	8
129	150	30	6.3	$3\frac{1}{2}$	.12	.10	.11
144	95	32	4.4	$3\frac{1}{2}$	.04	.02	.03
900	90	38	3.9	$3\frac{1}{2}$	.05	.06	.06
115	95	34	4.2	4	0	0	0
9	95	32	3.9	4	0	0	0
117	95	35	4.0	4	0	0	0
3	120	35	5.0	4	0	0	0

tomised Rats.

r 100 gm. rat.

Liver Glycogen %			*Blood Sugar mg/100 c.c.*
1	*2*	*Av.*	
9	10	11	12
.47	.45	.46	212
.24	.26	.25	183
.25	.27	.26	164
.40	.33	.37	168
.45	.44	.45	193
.39	.41	.40	180
.37	.39	.38	163
.21	.19	.20	133
.42	.45	.44	190
.39	.41	.40	150
.45	.36	.41	160
.34	.38	.36	144
.14	.11	.13	128
.33	.35	.34	
.22	.28	.25	
.16	.11	.14	
.51	.42	.47	
.16	.11	.14	
.19	.23	.21	
.28	.33	.31	
.22	—	.22	
.28	.20	.24	
.20	.16	.18	
.14	.19	.17	

9	10	11	12
.28	.26	.27	133
.48	.42	.45	160
.13	.15	.14	145
.21	.17	.19	123
.17	.12	.15	118
.17	.16	.17	141
0	0	0	170

TABLE 1A.

Effect of Glucose Meal on Control Rats, starved 24 hours.

DOSE: 2 c.c. 50% glucose per 100 gm. rat.

No. of rat	Wt. (gm)	Wt. of liver when killed (g)	Time after sugar	Muscle Glycogen% 1	Muscle Glycogen% 2	Muscle Glycogen% Av.	Liver Glycogen% 1	Liver Glycogen% 2	Liver Glycogen% Av.	ʃ	Blood Sugar. mg/100 gm.
99	120	5.0	1	.28	.29	.29	.56	.56	.56		
17	90	4.0	1	.32	.30	.31	.49	.53	.51		
19	135	5.2	1	.33	.30	.32	.40	.42	.41		
									Av:	0.47	
82	108	3.9	2	.31	.30	.31	.81	.85	.83		
83	100	3.7	2	.42	.45	.44	.90	.86	.88		
84	120	4.0	2	.40	.40	.40	.88	.92	.90		
									Av:	0.87	
151	120	5.5	3				2.10	1.86	1.98		
152	110	5.6	3				1.83	1.80	1.82		
153	120	5.0	3				1.06	1.30	1.18		
154	120	5.8	3				1.40	1.62	1.51		
									Av:	1.63	
100	165	7.0	4	.31	.31	.31	2.22	2.65	2.44		105
190	135	5.2	4	.56	.59	.58	3.26	3.33	3.30		140
18	170	5.9	4	.72	.56	.64	2.64	2.24	2.44		130
									Av:	2.73	

Fasting Average in control rats (see Barbour et al. (1927))
Liver Glycogen 0.16 gm/100 gm.
Muscle — ,, — 0.30 gm/100 gm.

TABLE 1B.

Hexose-phosphate in Resting and stimulated muscles of Hepatectomised and Control Rats.

Starved 24 hours: given 1 gm. Glucose 2 hours before death.

R — resting muscle. L — Stimulated muscle.

Rat No. (*hepatectomised*)		*Hexose* (*mg.* %)	*Organic P.* (*mg.* %)	*Ratio* $\frac{L}{R}$
1	R	6.8	16.8	2.3
	L	15.4	18.4	
3	R	4.5	16.1	1.8
	L	8.2	22.5	
4	R	16.0	6.2	1.3
	L	20.6	9.4	
5	R	6.7	15.1	3.1
	L	21.0	50.5	
16	R	3.7	15.3	2.6
	L	9.7	21.5	
21	R	8.2	16.0	2.3
	L	18.4	40.8	
		(Control)		
82		12.2	17.0	3.7
		45.5	38.6	
83		16.1	23.8	2.2
		35.2	60.0	
84		8.9	20.4	3.2
		28.2	58.2	

*THE LABORATORY DIAGNOSIS OF MALARIA.

by

Aubrey V. Greaves, M.B., (Tor.), D.T.M., (Liver.).

Government Bacteriologist, Hong Kong.

It hardly seems necessary to say that the diagnosis of malaria rests upon the examination of a stained blood film. Nevertheless, it is astonishing how many diagnoses of malaria are made on clinical grounds alone without such an examination. It is not putting it too strongly to say that such a procedure is inexcusable in modern practice. The question naturally occurs as to why this procedure is omitted. It is possible that the physicians referred to are either too busy or too lazy to make a proper diagnosis; or it is possible that their training has led them to the believe that malaria may be diagnosed on clinical grounds alone, especially by reference to the temperature chart. It is unfortunately true that a certain proportion of the profession falls into the first category and nothing can be done to alter their ways. With the second category, however, the teaching schools should deal, and they must assume their responsibility for their existence. There remains a third group which, it is possible, contains the largest proportion of men. This group, owing possibly to inefficient training, consider the procedure one which is beyond their technical ability. This group can be and should be eliminated.

Quite apart from the diagnosis of malaria, the examination of blood films fulfils other diagnostic criteria as well, such as examination of leukocytes, red cells and finding of parasites other than malaria.

It is a fact that good blood films may be made and stained successfully by any one who has two hands and a modicum of intelligence. It is true that the examination of the stained film requires a certain amount of experience, but by no means as much as many other procedures carried out by the young physician.

Technique.—No examination of the blood in the tropics is complete without the preparation of two slides, *(a)* a slide containing "thick drops" and *(b)* a slide referred to as a "thin film." The necessity for the former will be referred to later.

Slides to be used for blood work must be scrupulously clean. If not kept in absolute alcohol, they should be cleaned with 95% alcohol at the bed-side immediately before use. In addition to the two plane surfaces, the narrow edge of the slide at each end should be cleaned. The importance of this may be realised when it is remembered that it is with this edge that the blood drop is spread. At least three slides should be so prepared and placed in a convenient place near the patient.

* From The Bacteriological Institute, Medical Department; Hon. Dr. A. R. Wellington, Director of Medical & Sanitary Services.

The point of preference from which blood is obtained is the thumb and not the ear. The particular point on the thumb is laterally between the base of the nail and the pulp of the finger. This point should be cleansed with 95% alcohol and allowed to dry. A sharp stab is made with a needle and the first drop appearing is wiped away quickly with a dry swab. The next drop appearing is touched with the plane surface of one of the prepared slides at the point of junction of the upper one-third and the lower two-thirds, the slide being picked up by the thumb and fore finger of the right hand, each finger being on one plane surface, upper and lower. The slide is then turned over so as to bring the drop uppermost and is grasped at the two corners nearest the drop by the thumb and fore finger of the left hand, allowing the opposite end of the slide to rest on the knuckle of the fore finger. Without any pause, one of the other slides is picked up by the thumb and fore finger of the right hand at the two corners of one end and the opposite end applied to the drop at an acute angle without pressure, so as to allow the drop to spread along the width of the slide by capillary attraction. This only requires a second or two and then the slides is gently, smoothly, but with definite intention pushed along the lower one, *dragging the blood after it,* until the manœuvres ends off the edge of the slide in the air, i.e. the pushing of the one slide on the other does not stop at the end of the latter, so that a smooth action results. This should result in a smear which begins about 20 m.m. from one end and extends short of the full length of the slide and should show a series of fine lines at the end resembling a horse's tail. A slide of this description is almost invariably a good one.

This completed slide should be put face upwards beside the patient, the remaining blood on the finger being wiped away and a fresh drop obtained by squeezing. This drop should be rather larger than the previous one. A second slide should then be touched three times to the drop so as to leave three drops on the surface of the slide near the centre with a fair space between them. These drops should then be touched in turn with the corner of the other slide and each one slightly enlarged by a "puddling" motion. This procedure completes the taking of the specimen.

The first (thin) film should be waved in the air or in front of a fan so as to quickly dry it, and the thick drops should be placed under cover at room temperature for several hours until completely dry. Unless they are placed under cover, dust will accumulate on the drops and confuse the stained picture. Staining should be proceeded with as quickly as possible, the best results being obtained with fresh films.

Staining.—There are many directions for staining scattered throughout the literature. The result of one method probably differs comparatively little from the other, provided due care is taken in every step of the process. It is essential that a good reliable brand of stain be

used. A good method which has given excellent results in our hands for staining the thin film is a modification of Shute's technique. One of the most important points of his method is the maintenance of a neutral reaction of the diluted stain. It is necessary to have a supply of distilled water brought to a pH of 7.2. This can be obtained quite easily as a rule through someone who has access to a pH comparator, such as a hospital technician. If kept in a well-stoppered bottle it should last for months.

The thin smear is laid face upwards on the staining rack and sufficient Leishman stain is dropped on it to thinly cover the blood; the slide is then quickly rocked from side to side for ten seconds. Three times the amount of the prepared water is then dropped quickly on the slides and the diluted stain mixed by rocking and tilting the slide, care being taken that none of the stain is spilled. The slide is then left for forty-five minutes. At the expiration of this period it is then placed under a gentle stream of tap water so that the stain and any deposit is washed off the slide and development continued by moving the slide from side to side under the water for fifteen seconds. It is then shaken dry and placed on edge to drain.

No blood examination for the plasmodia of malaria is complete without the preparation and examination of thick drops. In fact from the clinical stand point the thick drop preparation is by far the more important of the two. The reason for this is that the increased concentration of parasites in a small area enables their presence, even in small numbers, to be recognised with speed and certainty. Unless one has had experience with the phenomenon it is astonishing in many instances to find parasites in every field of the thick drop, while an examination of the thin film reveals none, or at the most only an occasional one after prolonged search. The thick drop enables a negative report to be made after five minutes examination, and this with a high degree of certainty. It does not, however, enable the examiner to diagnose the species present, unless of course crescents are seen. It is quite enough, however, for the clinical conduct of the case, in the great majority of instances at least, to be assured merely that parasites are present.

Before staining, the thick drop film should be dehæmoglobinised. The slides is placed on a staining rack and the prepared distilled water added so as to cover the drops. This is allowed to act until the drops are reasonably free of hæmoglobin; the water being occasionally changed. No attempt, however, must be made to completely remove the hæmoglobin, as prolonged immersion in water is liable to cause the "drops" to fall off the slide in process of being stained.

Staining with Giemsa is preferable in the case of large numbers of films, but for an occasional film (up to three or four at a time) the following combined method has given equally as good or better results in our hands.

Five drops of Giemsa stock solution are placed in the bottom of a test tube and eighty drops of prepared distilled water added and mixed. The slide is placed on the staining-rack and approximately 10 drops of Leishman stain applied, this is rocked back and forth for about ten seconds. An equal amount of the prepared Giemsa solution is then placed on the slide and mixed with the Leishman by rocking and tilting. Three minutes are allowed to elapse and at the expiration of this period the slide is washed in a running stream of water and developed for about fifteen seconds. It is then placed on edge to dry.

If a large number of film is to be stained the longer method is perhaps preferable. Sufficient Giemsa stain, diluted one in twenty, is mixed and placed in a staining trough. After de-hæmoglobinisation, the films are placed in the dish and allowed to remain for half an hour, after which they are treated in exactly the same manner as in the short method.

General Hints.

(1) Before attempting to make a film, it should be realised that everything to be used must be placed in proper position *before-hand*, so that the whole procedure goes on without a hitch. For example, the three slides must be placed upon some object so that the ends to be handled must be free; thus they may be picked up as the directions previously given suggest.

(2) In taking the blood drop from the patient it must be remembered that if it is necessary to wipe away a drop, this should never be done with the alcohol swab, always with a dry swab, which should be ready at hand.

(3) The necessity for speed in touching the slide to the drop and making the smear cannot be emphasized strongly enough. Even the slightest beginning of clotting absolutely prevents a proper smear being made, and this is one of the commonest failures which is met with.

(4) Another cardinal fault in making smears is taking too much blood. A good film may be made with a very tiny drop, but beyond a certain point nothing can be made of a drop which is too big except a mess. Always wipe away the drop and allow a smaller one to well up.

(5) A useful point in carrying out the actual staining process is to use capillary pipettes with teats. One is reserved for the water and the other for the stain. They deliver smaller drops than drop bottles, and the drops can be perfectly controlled. Ordinarily speaking ten drops of stain from one of these to thirty drops of water is about correct and gives sufficient stain to flood the slide, while the total of forty drops is just as much as the slide can contain without spilling.

(6) One of the disadvantages of the method of staining described is the tendency to precipitation of the stain on the slide. This can be almost completely overcome by observing the following points. Staining should be carried out in a place free from air draughts which would tend to evaporation of the stain. From the moment the Leishman stain is put on the film until the dilution with water is complete *the process should be hurried,* and no time should be lost between steps. Finally, when the period of staining is complete the first stream of water directed on the slide should be a vigorous one, which with a slight tilting back and forth removes the deposit before it has time to fix itself on the slide. This is important. Fortunately, however, most of the deposit is confined to the edges of the slide and good clean parts are always available for examination.

CHEMICAL CHANGES IN THE BLOOD AND CARDIAC TISSUE IN THE HEART LUNG PREPARATIONS.*

K. Mackenzie, M.S., PH.D.

The object of these experiments I am about to describe is one of considerable importance since it relates to the normal functioning of the heart—the source from which it draws the energy expended in the beat, the chemical changes which occur in the metabolites, and the more general question as to whether cardiac muscle functions in the same manner as voluntary muscle in respect to the chemical changes which bring about recovery from the effects of contraction. Taking the last point first, as the one on which general interest centres, cardiac muscle is generally placed in a class by itself, intermediate between the quick acting voluntary muscles on one hand, and the sluggish autonomic-visceral type on the other. Cardiac muscle shows properties derived from both—it is autonomic, yet largely under nervous control; it is rapid in action, and yet shows no symptoms of fatigue under continuous operation at moderate loads. The chemical system of energy production in the heart remains largely unexplored. Both "phosphagen" (adenyl pyro-phosphate) and "asparagen" (asparagine pyro-phosphate) the labile esters involved in glycogen breakdown and resynthesis in voluntary and smooth muscle respectively—have been reported in the heart, but in amounts much less than its steady activity would seem to necessitate, while the presence of various di and tri-saccharides and of inositol, a hexa-hydroxy-benzene, have been reported. The glycogen is high, and no satisfactory proofs of its reduction in excessive cardiac exercise have been forthcoming, which has rendered many observers sceptical as to the role it plays, though the considerable stores which are present form an equally strong argument in favour of such function as we find in skeletal muscle, viz., to provide a reservoir of available energy. Such light as we are able to throw on this question is largely inferential, and will be considered after a survey of the experiments to which I now refer.

It is ten years since the chemical changes in the blood and tissues of the heart lung preparation began to be studied. The classical experiments of Starling, at first elucidating the dynamics of the heart, were extended by his pupils to the more intricate and less spectacular researches on its energy requirements and their source. It soon appeared probable that these were met by the oxidation of the glucose in the circulating blood, since the fall of the blood sugar in the isolated heart lung preparation proceeded regularly with time. But Anrep showed that lactic acid also played a significant part, since in high concentration of carbon dioxide in the air supplied to the lungs, a

* Read before the Hong Kong University Medical Society.

very considerable amount of lactic acid was metabolised—not, apparently to the end products of carbonic acid and water, since the respiratory quotient rose little, if at all, but probably, by synthesis to carbohydrate. It was generally conceded, however, that the blood sugar was the staple source of energy. Our first experiments, therefore, aimed at depriving the blood of its sugar. There are two possible methods of doing this (1) by prolonging the experiment, so that the heart will exhaust the supply of blood sugar (ii) by removing sugar from the blood by selective absorption. The first method has the disadvantage that the impairment of function in the heart lung system is progressive and continuous—loss of CO_2 from the blood increases its alkalinity—drying of the lungs occurs owing to excessive ventilation, bringing on pulmonary oedema, diminished oxygenation, consequent distension of the coronary vessels, and further diminution in the oxygen saturation of the blood—a vicious circle which rapidly develops and brings the heart to a standstill. To keep the heart lung functioning for eight hours is possible, if attention is paid to the aqueous vapour pressure and carbon dioxide concentration, but it cannot be said that after this lapse of time the system is functioning well, and it is at this point that the disappearance of the blood sugar begins to make the experiment interesting. The second method was devised to overcome the difficulty, and consists in centrifuging the blood, separating the plasma, stirring this with a suspension of fresh washed yeast (as suggested by Somogyi) and re-centrifuging. The plasma is then found to contain only 10-12% of its normal content of sugar, and if it is mixed with the corpuscles gives a hypoglycaemic blood which is apparently normal in other respects. Its use in the heart lung preparation enable the blood sugar to be completely exhausttd in one or two hours. It is equivalent to starting the experiment at the fifth hour of the ordinary system, and I have so represented it on the same diagram (Fig. 1).

The result is interesting. The lactic acid content of the blood, always considerable in the admittedly abnormal conditions of the heart lung (where it replaces the CO_2 normally present, but here lost by over ventilation), and constant in the presence of blood sugar, commences to fall as soon as the latter is used up, and soon descends to zero. Lactic acid, therefore, is an effective energy provider for cardiac muscles. But subsequently the system continues to function in the apparent absence of either. One says apparent, because anoxaemia shows that the possibility of their formation is present.

Performance of the Heart during Anoxaemia.

Our second series of experiments were complementary to the first. If the normal heart lung preparation, ventilated with air, or oxygen and carbon-dioxide mixture, is ventilated with nitrogen, the blood becomes venous within half a minute, and oxidative processes probably

cease almost simultaneously. Yet the heart continues to function for some minutes before severe distress supervenes. Then the coronary flow suddenly increases to an enormous extent, and the best becomes slow and feeble and finally ceases, without the occurrence of fibrillation. Restoring the normal ventilation brings about an almost complete recovery, though the coronary vessels never recover their original tone and oxygenation is somewhat impaired as a result. The chemical changes are likewise interesting: so far as analysis can say with certainty oxidation processes cease during anoxaemia and the blood sugar remains constant, while the blood lactic acid rises rapidly and the tissue glycogen falls towards zero. (Fig. II). During recovery on oxygen, the blood sugar and the lactic acid fall, the former at its normal rate, the latter rapidly, and glycogen reappears in the tissue (Fig. III). The process appears to be an emergency one, resembling the way in which skeletal muscle "goes into debt" for oxygen during vigorous exercise. This system is apparently still in normal working order in the absence of blood sugar or blood lactic acid, (Fig. IV) since if the heart supplied with blood free from these metabolites is put into anoxaemia it continues to beat (for a shorter time than in the former case, however,) and lactic acid appears in the blood, while glycogen disappears from the tissues, and on re-oxygenation, these processes are reversed. There is at least a suspicion that some material detectable as "blood sugar" appears in the blood during the anoxaemic period, though its estimation is beyond exact analysis. The quantity is in any case small. It tends to confirm the suspicion that the glycogenic reserves of the heart are breaking down into lactic acid and lower carbohydrates, which are capable of undergoing resynthesis. Is it possible to deduce from these conclusions (admittedly vague and tentative) any general hypothesis for our guidance in future experiments on the question of cardiac metabolism? I will venture to give an outline of my personal view. This postulates a dual mechanism in the heart of—(i) a normal system of metabolism, and (ii) an accessory, emergency system.

(I) The normal system of metabolism consists of the conversion of the sugar of the blood into carbon dioxide and water, possibly according to the scheme [B.S. $\rightsquigarrow$ glycogen $\rightleftharpoons$ lactic acid $\rightsquigarrow$ CO_2† H_2O] as in normal muscle But I do not like to rule out the possibility that the intermediate stages may be quite different from this—that the glycogen plays an accessory and not an essential part, and that other intermediate carbohydrates may have a more important place in the general scheme.

(II) The emergency mechanism functions only during maximum output or severe anoxaemia. This involves the breakdown of glycogen to lactic acid, possibly via the labile esters such as phosphogen, possibly not. Of the processes of resynthesis and the part played by lower carbohydrates we are totally in ignorance, and must accept provisionally the behaviour of skeletal muscle in nitrogen as an analogy.

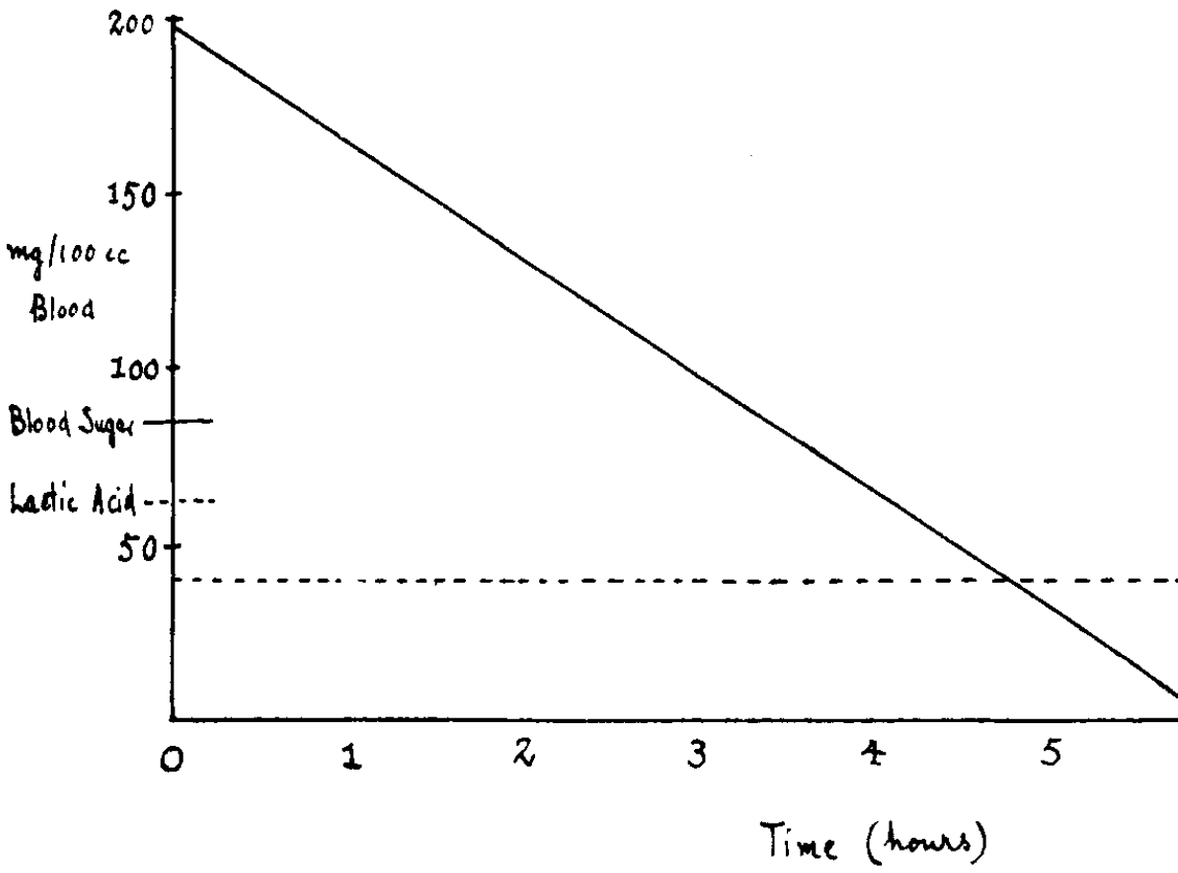

200
150
mg/100 cc
Blood
100
Blood Sugar
Lactic Acid
50
0
1
2
3
4
5
Time (hours)

Fig. I

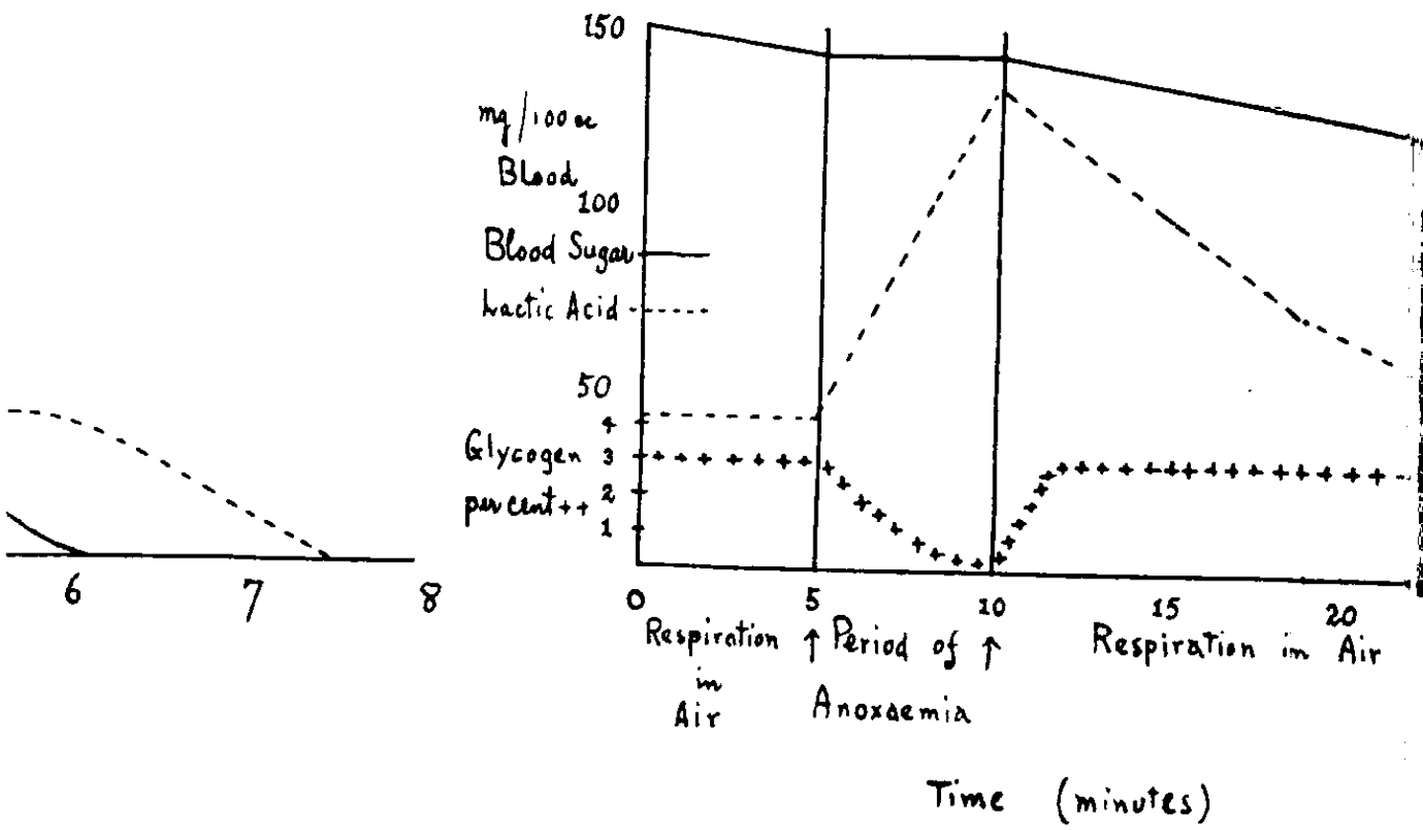
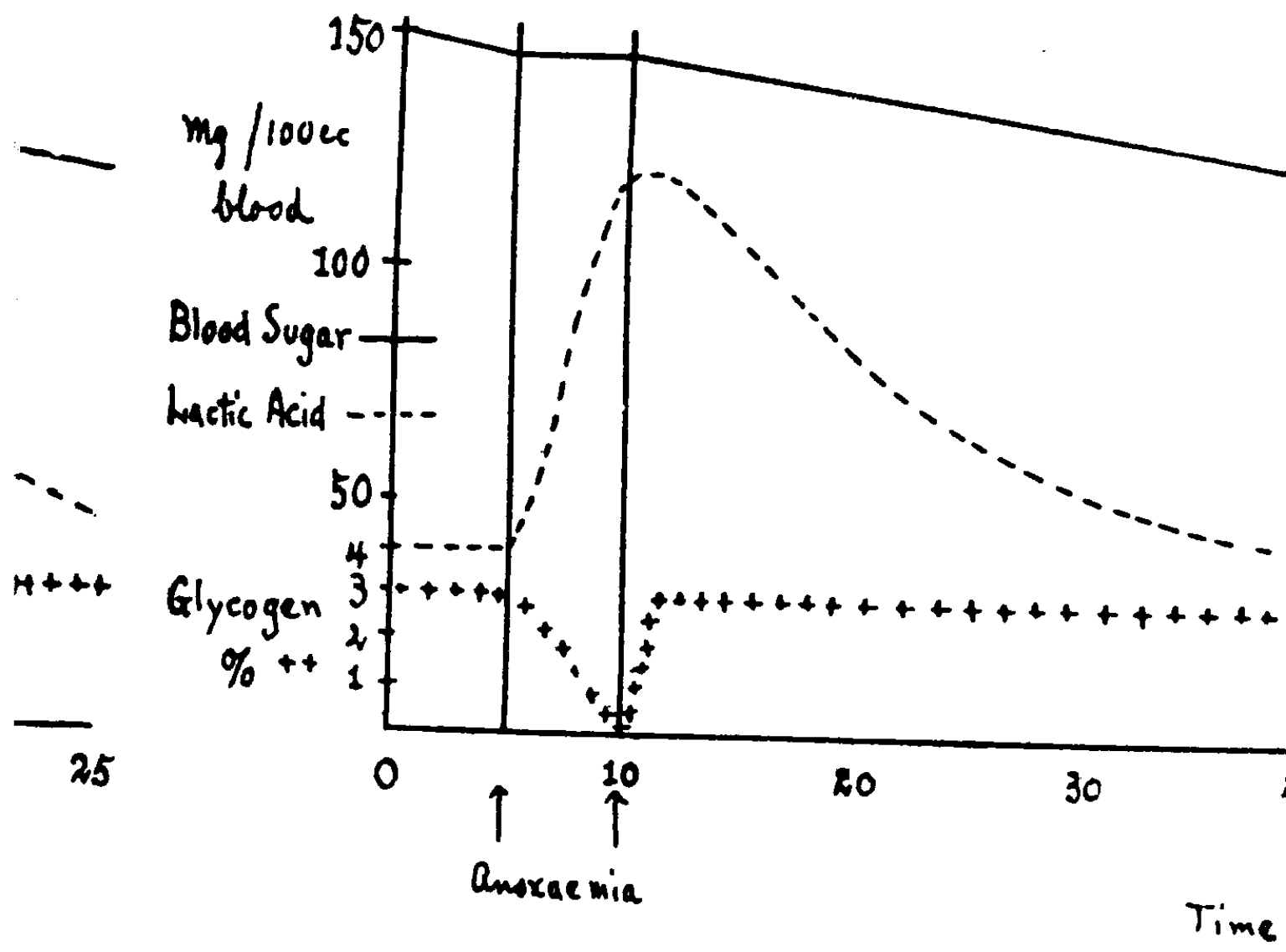
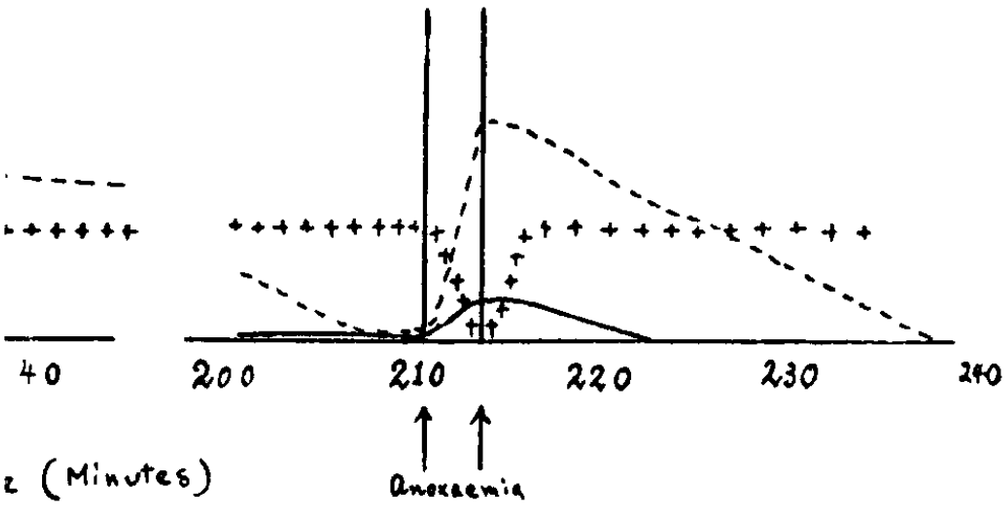

150
mg/100 cc
Blood
100
Blood Sugar
Lactic Acid
50
Glycogen
per cent
4
3
2
1
0
5
10
15
20
Respiration in Air
Period of Anoxaemia
Respiration in Air
Time (minutes)
6
7
8

Fig II


150
mg /100cc blood
100
Blood Sugar
Lactic Acid
50
4
3
2
1
Glycogen %
++++
++
25
0
10
20
30
Anoxaemia
Time

g III

Fig IV continuation of fig. III



I have placed before you the results of a considerable research, which merely provides a finger post, on which the indications are undertain and the diverging paths from which are obscure. The field is wide, the problems are formulated—it remains for the workers of genius to map out yet another settled territory in the fascinating and wide domain of experimental physiology. I hope that some of you may set your feet upon the path and reach the goal.

CORRESPONDENCE.

To the Editor
of the Caduceus, Hong Kong University.

Dear Sir,

I have read with much interest the article in your May number on the Pathology of Hematogenous Osteomyelitis by Dr. Chiu Put Po.

I can only speak of the condition from the clinical standpoint, and do so on the basis of cases seen in Central China between the years 1898 and 1918, and one case seen subsequently in Tsinan.

One sequela mentioned by Dr. Chiu—that of *fracture* of the bone I saw twice. In each case the sequestrum had escaped and the involucrum had spontaneously fractured. In one case the lower half of the humerus was concerned, the fracture caused some angulation of the shaft and incidentally involved the muscular-spiral nerve, which was paralysed. The other case was one where the shaft of the femur was affected, with the result that the bone had telescoped causing a shortening of 3 to 4 inches. Neither of these cases could be attributed to the surgeon's fault, as they had occurred prior to the patients coming under treatment. In each case the fracture had been caused by the patient (in his ignorance) having subjected the diseased bone to a greater strain than it could bear.

There is another sequela which Dr. Chiu has not mentioned, namely *overgrowth* of the affected bone. Of this I saw two cases, in each of which there had been long-continued congestion of the bone due to the chronic inflammatory process. Both cases concerned the tibia and caused a lengthening of the bone (when measured against the opposite side) of 1 to 1½ inches. In each case it appeared that the increased vascularity in the neighbourhood of the epiphyseal cartilage had resulted in more vigorous growth of the bone than had had occurred on the normal side. In one case the lower half of the tibia was affected, patient's age being about 10; in the other case the upper half of the bone was involved and patient's age was about 15. In neither case was the fibula involved in the disease. I regret that I did not get X-ray pictures of the leg in each case, so as to determine how far the normal level of the upper and lower tibio-fibular articulations were affected, and whether there had been any elongation of the fibula or not. It seems to me that this overgrowth is of special interest, in view of the fact that if the active disease involves the epiphyseal cartilage, the result, as Dr. Chiu points out, is exactly the opposite and leads to an arrest of the growth of the bone.

Wishing the Caduceus all success, I remain,

Yours sincerely,

(P. L. McALL).

School of Medicine,
Shantung Christian University,
Tainanfu.

Acknowledgements

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On the Types of Cholera Vibrio of the Shanghai Epidemic of 1932, by Masahiko Kuroya, M.D. and Kempu Oshio.
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Notes and Comments

It is very pleasing to note that Dr. Yang Lin, M.B., B.S. (H.K.), F.R.C.P.I. will be back with us sometime next year. He has been away doing post graduate work in Great Britain and Ireland and has distinguished himself. We hear he will be attached to the Gynæcological Department.

An addition to the Medical Buildings is in the course of erection. This time it is the Surgical School. This long felt want by the Surgical Department has at last been filled by the University. We envy the new operative surgery students who at last have no need to be crowded out by the Seniors.

We have to thank Dr. Wu Lien-teh for inviting six of our members to the Hong Kong Chinese Medical Association Meeting when an address on "The Future of the Chinese Medical Profession" was delivered by him.

The bust of Professor C. Y. Wang will be unveiled at the end of the year. There is still an amount to be paid for it. We would like to appeal to the members and friends of the late Professor who have not yet subscribed to it to do so.

The annual function of the Society in the form of a Supper Dance will be held sometime in January.

The Dean of the Medical Faculty has received a letter from The National Economic Conference, Division of Women and Children's Welfare-Nanking saying that they are contemplating steps towards the improvement of Women and Children's Health Welfare. The Director of this division wishes to know if any Medical Graduates of Hong Kong University are particularly interested in this matter. Those interested should apply to the Dean's Office.

Review of Books

"Aids to Pathological Technique": By D. H. Haler, M.B. p. 183. Figures 18, price 3/6d. Bailliere, Tindall & Cox, London.

This addition to the Student's Aids series is intended to give the student and other laboratory workers some ideas as to laboratory methods of proved value. It consists of a brief description of the commoner procedures employed in "clinical bacteriology," hæmatology, parasitology and bio-chemistry. The little book should prove helpful to the student commencing such work. It is, however, somewhat disappointing from the standpoint of tropical medicine. The method described on page 35 for the cultural examination of the faeces is not in the opinion of the reviewer the best technique for the isolation of dysentery bacilli, and the "Widal Reactions" (author's italics) for dysenteries cannot be described as a standard technique for diagnosis of this condition. There appears to be no mention of the significance of the presence of macrophages and Charcot-Leyden crystals in faeces from suspected cases of dysentery. The very important question of laboratory diagnosis of malaria is but briefly dealt with and no description is noticed of the thick film method or of the significance of monocytosis. The Leishmania are dismissed in five lines and filarial infections in four.

It is regrettable that authors of British books on clinical pathology and allied subjects frequently lay so little stress on diseases of first importance in a considerable portion of the Empire, and of surely more than academic interest to many practitioners in London.

L. J. D.

Studies from The Institute for Medical Research, Federated Malay States.

"No. 21, Melioidosis": By A. T. Stanton, C.M.G., M.D., F.R.C.P., and William Fletcher, M.D., M.R.C.P., London: John Bole, Sons and Danielson, Ltd. 37 plates. Pp. 59.

Melioidosis, a disease resembling glanders was first described in man at Rangoon in 1912. The following year it was discovered in guinea pigs and rabbits at the Research Institute, Kuala Lumpur. In 1917 Dr. Stanton discovered human cases in Kuala Lumpur. He identified the causal organism as Pfeifferella whitmori, a bacillus of the glanders group, which had originally been isolated from the Rangoon cases. Subsequently, cases have been described in Ceylon, Cochin-China and Central Africa.

It has been assumed but not proved that that rats constitute the normal reservoir of infection. In man the disease runs a rapid and

"*Catechism Series: Zoology*" *(Invertebrata)*. Part I. (3rd Edition). By Robert A. Staig, M.A., PH.D., F.R.S.E. Lecturer in Zoology, University of Glasgow.

Within this little booklet (88 pages, demi 12 mo) the author has packed an amazing amount of information on the fundamentals of Zoology, the whole phyla Protozoa and Porifera, and nearly all of the Coelenterata. As indicated by the title, the Catechism Series, it is not to be regarded as a text book on the subject, but rather a method of reviewing the field already covered in lecture and laboratory with the aid of more complete texts. For this purpose the student will find the system of questions and answers on which the whole book is based very helpful, in as much as many of the questions might well be set on an examination paper, and the answers give with admirable terseness the essentials necessary in a successful reply.

The first sixteen pages are devoted to general zoological considerations, i.e. the nature of protoplasm and cells, their methods of division and reproduction, the production and union of germ cells and the subsequent development of the embryo with its germ layers and the organs and structures so produced.

The Protozoa are dealt with by classes and orders, a typical example of each being described and sometimes figured, and a good number of others cited. While the system of classification followed is quite orthodox there is no justification for the name "Gregarines" being applied to the Sporozoa as a whole; it was done perhaps from lack of any popular name for the Class, but no name is better than a misleading one. Moreover, in spite of this being a third edition and presumably revised up to date, a number of old scientific names and terms are used which for several years have been rejected in favour of others with prior claim or more acurately descriptive. True, some of the accepted names are given in brackets as synonyms, e.g. *Piroplasma (Babesia) bovis;* kinetonucleus (parabasal body); but *Schizotrypanum* for the organism causing Chagas Disease has reverted to *Trypanosoma* and *Borrelia* as a generic name for spirochaetes causing Relapsing Fever had a very short life. Similarly it is now questioned whether *Balantidium* is pathogenic to man, and the contractile vacuole of the protozoa is now considered to be a means of extruding excess water taken in by camosis, and the function of excretion formerly ascribed to it relegated to the cytoplasm as a whole. These are minor points which should have been set right before the 3rd edition was printed, but the author may be criticised more severely for discussing the Spirochaetes under the heading "Hæmoflagellates" with which group they have only the remotest connection; he should have placed the discussion at the end of the Protozoa and emphasised the features of a non-protozoal nature which relate them quite as closely to the Bacteria as to the Protozoa.

The sections on the Porifera and Coelenterata are admirable examples of condensed scientific information, but two figures for each phylum seems a lamentable allowance for illustration. It is also regrettable that a few pages were not added in which to describe the Ctenophores and so complete the Coelenterata in the one volume.

L. G. S.

"*Forensic Medicine Catechism Series.*" By Andrew Allison, M.B., ch.B., B.SC., etc. (Edinburgh). Third Edition, Paper, 80 pages, E. & S. Livingstone.

The limitations of this little volume are so great that one cannot help wondering what useful purpose can possibly be served by its publication. That it will have a wide circle of readers need not be doubted—the fact that it has run into three editions is in itself sufficient proof—but that the readers of it will receive adequate benefit from its perusal is to be doubted. The very form of presentation—by question and answer—tremendously limits the information which can be gleaned from its pages. The only possible use which it can serve is as an examination cram-book. Unfortunately, however, it will be used by some students to take the place of attendance at lectures.

The value of some of the answers to the questions is well shown by the following:—

"Q. *How should the internal Examination (of a body) be*" "*carried out?*" A. Thoroughly. "All the organs of all the cavities should be examined even though the apparent cause of death has been found in one of them." A fair examiner might give about 5% for this answer and call it charity. Nevertheless, some of the questions and answers are useful, and as a quick refresher outside the door of the examining room may do their part in enabling an ignorant student to pull through.

It is refreshing to note that the three too commonly used tests for blood viz. the benzidine, the Kastle-Meyer and the Guiacum come in for some apt criticism. It is unfortunate, however, that no mention is made of Takayama's test, a test which has received much favourable attention from the Edinburgh School. No mention is made either of the disappearance and reappearance of spectroscopic bands when solutions are treated with alkali and ammonium sulphide.

The section on offences of a sexual nature is full and well written within the limits of the method adopted. There are, however, answers which verge on the silly. The following is a good example: "*Q. How should you examine a child in a case of Rape?*" "A. Note if the child's account is lesson-like; if its statement heard apart

from the presence of the parents is the same as when they are present. Note any signs of precocity." This answer conveys no information to a student on the technique of an important procedure.

This book can in no sense be recommended to students and it is not likely to appeal to others.

A. V. G.

"*History of Chinese Medicine.*" By Drs. K. C. Wong and Wu Lien Teh in one volume 8 vo. cloth. Price 30s. and G.$7.50 nett. National Quarantine Service, 2 Peking Road, Shanghai.

This large volume of 706 pages should be of great interest to our local graduates and students for many reasons. Firstly its joint authors both have some connections with our own University. Dr. Wong is a licentiate of the old Hong Kong College of Medicine, the fore-runner of our present medical faculty, and Dr. Wu Lien Teh, as every one knows is one of our Hon. LL.D.'s. The second and more important reason why it should be of interest to our readers is that in the attempt to introuce Western Medicine into a country such as China, a knowledge of old customs and habits, beliefs and fears which have to be overcome is invaluable. For that reason one would like to see the first part of the book amplified in later editions. An excellent production such as this could easily stand two volume. It is the older part of Chinese medical history that is of the greatest interest to medical historians, and the greatest value to modern workers and it does seem slightly misproportioned when one finds that this period of 2697 B.C.—1800 A.D. dealt with in 120 pages, while to the rest up to 1930, about 500 pages are devoted, especially when the period 1931—1930 has itself 100 pages.

The volume is liberally supplied with photographs of general interest, and here again our graduates will welcome the inclusion of a photo of the late Professor C. Y. Wang, our first Professor of Pathology. It is impossible in a short review such as this to touch on all the interesting points of the book, but it will be safe to say that the volume as it now stands will form an excellent basis for a larger work which this important and interesting subject demands.

L. T. R.