It will suffice to quote here in tabular form the six experiments that gave positive results :

	Flea put on Rat.	Flea Recovered.	Trypanosomes first Seen in Blood of Rat.	Multiplication Period Ended.	Condition of Flea when Dissected.
1	Aug. 2, '10	Aug. 5, '10	Aug. 11, '10	Aug. 13, '10	Trypanosomes scanty in stomach.
2	Sept. 19, '10	Sept. 22, '10	Sept. 28, '10	Sept. 30, '10	Trypanosomes swarming in stomach.
3	Oct. 10, '10	Oct. 14, '10	Oct. 17, '10	Oct. 22, '10	Trypanosomes not found.
4	Oct. 20, '10	Oct. 25, '10	Oct. 28, '10	Oct. 31, '10	As last.
5	Nov. 1, '10	Nov. 4, '10	Nov. 14, '10	Nov. 16, '10	As last.
6	Nov. 11, '10	Nov. 15, '10	Nov. 23, '10	Nov. 25, '10	Trypanosomes swarming in rectum.

With regard to this table it should be remarked, first, that the multiplication of the trypanosome in the rat occupies the first eleven or twelve days after the actual infection has taken place; secondly, that the fleas which produced these infections showed, when dissected, various conditions, from an infection of the digestive tract so scanty that trypanosomes were not found by microscopic examination in the fresh state, to others in which the stomach or rectum were teeming with them.

Flea A.

No. of Rat.	Flea put on.	Result.	Trypanosomes First Seen in Blood.	Multiplication Ended.
265	Feb. 14 to 18, '11	+	Feb. 24, '11	Feb. 27, '11
249	Feb. 24 to March 1, '11	0	—	—
260	March 1 to 6, '11	+	March 15, '11	Rat died March 16, '11
291	March 6 to 13, '11	0	—	—
293	March 13 to 17, '11	0	—	—
295	March 17 to 22, '11	+	March 27, '11	March 31, '11
249	March 23 to 27, '11	0	—	—
291	March 27 to April 1, '11	+	April 5, '11	April 9, '11
293	April 1 to 5, '11	0	—	—
249	April 5 to 10, '11	0	—	—
262	April 10 to 15, '11	0	—	—
296	April 15 to 20, '11	+	April 22, '11	April 25, '11
263	April 20 to 25, '11	+	May 1, '11	May 5, '11
292	April 25 to 29, '11	+	May 1, '11	May 6, '11
293	April 29 to May 4, '11	0	—	—
*294	May 4 to 9, '11	?	—	—
249	May 9 to 15, '11	0	—	—

* Rat died May 9, 1911.

When the flea was searched for on May 15th it could not be found.

Flea B.

No. of Rat.	Flea put on.	Result.	Trypanosomes First Seen in Blood.	Multiplication Ended.
272	Feb. 14 to 18, '11	+	Feb. 24, '11	Feb. 28, '11
259	Feb. 24 to March 1, '11	0	—	—
253	March 1 to 6, '11	0	—	—
292	March 6 to 13, '11	0	—	—
294	March 13 to 17, '11	0	—	—
296	March 17 to 22, '11	0	—	—
259	March 23 to 27, '11	+	March 30, '11	April 4, '11
253	March 27 to April 1, '11	+	April 5, '11	April 7, '11
292	April 1 to 5, '11	0	—	—
294	April 5 to 10, '11	0	—	—

When the flea was searched for on April 10th it could not be found.

Still more striking are the results obtained by feeding a single flea on a number of rats successively. In this experiment we took, on February 14th, 10 fleas from the infected breeding-cage, and put each flea on a separate rat in a flea-proof cage of the kind already described. On February 18th each of the 10 fleas was recovered and put by itself in a test-tube containing a little damp sand, while the rats on which they had been put were examined daily. On February 24th 2 of the 10 rats were found to be infected; the other 8 never showed infection. The 8 fleas which had produced no infection were returned to the infected breeding-cage, but the 2 that had infected their respective rats, henceforth to be known as Flea A and Flea B, were put on a succession of clean rats, with the results shown in the accompanying table.

The experiments show, in the first place, that Flea A infected seven rats and Flea B three rats, consequently that these infections could not have been brought about by the rats eating the flea. Secondly, they show that an infected flea does not infect at every feed, and consequently they throw light on the small percentage of fleas taken from the infected breeding-cage which produce an infection when placed on a rat *only for a short time*. Thirdly, they show incidentally that a flea lives a considerable time. Flea A, a female of uncertain age on February 18th, was to all appearance healthy and vigorous on May 9th, when last seen. Possibly both A and B were eaten finally by the last rats on which they were put; we cannot say.

In conclusion, we venture to assert that the experimental results cited by us support our belief that the infection of rats with *T. lewisi* is not brought about normally through the rat eating the flea; and from other evidence, which we hope to publish in full detail at a future time, we believe that the normal method of transmission is that the ripe, infective form of the trypanosome—the final form of the developmental cycle which it passes through in the flea—is regurgitated from the stomach of the flea into the wound made by the proboscis of the flea during the act of feeding. Considering the problem of the transmission of trypanosomes as a whole, the conclusion that infection is effected by the bite of the invertebrate host receives abundant support from other known cases of trypanosome transmission. No one can suppose that human beings acquire trypanosomiasis by eating tsetse-flies in Africa or bugs in Brazil; and Miss Robertson's recent researches on fish trypanosomes² show conclusively that fishes are infected by the bites of leeches. We consider, therefore, that the infection produced by the vertebrate host devouring the invertebrate is an exceptional occurrence without any bearing on the general problem of the transmission of trypanosomes.

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¹BRITISH MEDICAL JOURNAL, May 6th, 1911, p. 1049. ²Ibid., March 18th, p. 621. ³Phil. Trans., B., vol. 202, p. 29.

COMPARATIVE NUTRITIVE VALUE OF WHITE, STANDARD, AND WHOLE- MEAL BREAD.

BY

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(Second Note.)

DR. CHALMERS WATSON,¹ criticizing our first note,² said that we ought to have fed our rats on bread and milk, not on bread and water. Just after reading his letter we passed through a slum in Bethnal Green. The children were out from afternoon school, and had been home for tea. Many were out in the street munching slices of bread. Some of the slices were brown and some white, but all had nothing on them; plain bread and a drink of tea or water was their meal.

The bread question is one that affects particularly the very poor. It matters little to those who can afford milk, meat, bacon, cheese, etc. The point we have to settle is how

the children of the very poor may secure the bread best for growth—for they too often get bread without milk.

It is beside the mark to test the matter by feeding rats on bread *plus* milk. Milk is a food formed for the growth of the young mammal, and contains all the chemical groups necessary for growth. To add white flour to it is to give additional bricks to a body builder.

H. Snyder fed men on bread and milk for four days at a time, and gave us the results of a careful and full metabolic study.³ His conclusion was that white flour yields more digestible and absorbable protein for the same money than wholemeal. We do not deny that this is so. White bread is suitable enough for those who eat other food which, like milk, contains all the groups necessary for growth and repair.

The wheat berry, just as milk, is a body builder; it, too, contains all the groups necessary for the growth of the sprouting corn. The question of the value of the different parts of the berry cannot be decided merely by estimation of protein and carbohydrate content. Who would separate the casein and lactose from milk and feed our babies on these *plus* water and *minus* the salts and fats? And yet this is comparable to what the miller does to the wheat berry.

The ovum of the wheat grows on the foodstuffs contained in the whole berry. We may be sure there is nothing in it which has not its use in the growth of the sprout.

Of first importance must be the contents of the germ, for in it reside not only the initial elements of growth, but ferments which prepare the contents of the berry for the nutrition of the sprout.

To grind the wheat and take away not only the germ—the very embodiment of growth—but to impoverish the flour in most of the essential organic phosphorus compounds contained in the outer layers, to do this seems a most extraordinary manipulation of the “staff of life.”

More extraordinary still is the fact that the State allows this to be done, and does not insist on the miller notifying that the flour is thus impoverished. If such a thing were done to any other article of food, the law surely would step in and hold it to be fraudulent. The question which has to be settled in respect to any flour is this: How far are all the groups necessary for growth present—the ringed amino-acids, phosphorized fats, salts, ferments, or their activators, etc.—bodies the utility of many of which can be detected only by their effect on growth, by biological, and not by chemical tests? In other words, How far is the flour in question a body builder in itself? Our experiments show that white flour is wholly deficient, and we have ascertained that our white flours are unbleached and free from “improvers.”

For rats standard flour is very much better, and wholemeal better still. For mice standard is the better so far (one week). We find that wholemeal and water is a diet on which rats can grow, keep in very fair condition, and beget their own kind—the denizens of wheat stacks, mills, and granaries must live almost entirely on wheat. We have a rat bringing up a family quite well, and she herself has grown up and lived for ten weeks, first on standard and then on wholemeal flour and water. The traditional prison diet—bread and water—seems a possible diet so long as wholemeal or standard and not white bread is supplied. The black bread of the old, hard times is better, not worse, for the worker than the white bread of the rich.

The diet of white flour and water hits the younger rats far harder than the older ones. The germ and the bran are needed above all for growth. Two of the youngest rats, as a control, have been fed on white bread and milk, bacon and cheese, and have done much better than those fed on wholemeal and water—a fact which shows that the bread question is of supreme importance only to those who get little else than bread.

In the case of our first set of rats, the 15 fed on white flour, which lost 36 grams in the sixth week, were then put on wholemeal. They gained 31 grams in the seventh and 167 grams in the eighth week.

Drs. Edie and Simpson have obtained results similar to ours on pigeons, and cite others of like nature obtained by Schaumann. Who can doubt, then, that the children of the poor, who are fed mainly on bread, ought to be given what the rat, the pigeon, and the sprouting wheat require for their growth?

The tables we append of our control experiments show the deficiency of white flour conclusively. In this series

cannibalism has been prevented almost entirely, and this has made the ill effect of white flour far more evident. The Bread and Food Reform League urge the use of what is termed “standard” bread because of the prejudice against the brown wholemeal bread. In our opinion the State should use all its powers to make standard and wholemeal bread the food of the children of the poor.

White flour will continue to be eaten by the well to do because of its neutral flavour—it sets off the tastes of other foods, and white flour is necessary for the arts of pastry making. White flour robbed of the germ will keep easily, makes a pretty loaf, and saves trouble to miller and baker. But the whole meal is the real “staff of life,” and no saving of trouble or liking of a nice-looking loaf should be the pretext for taking away substances essential for the growth of the children.

It is recognized now that the protein content of a flour does by itself not determine the nutritive value, for the proteins do not all yield on digestion the same kind or proportion of amino acids—the building stones out of which the bioplasm of the different organs are built. The “ringed” amino-acids have been shown to be some of the most important. Thus, Dr. Gowland Hopkins found that mice can live longer on zein (a protein obtained from maize) plus tryptophane than on zein alone.

It occurred to us that the superior nutritive properties of standard flour might in part be due to the fact that the valuable amino-acids, such as tryptophane, were split off

TABLE I.
Wholemeal Flour.
Weights of rats in grams.

Rat.	First Week.	Second Week.	Increase.	Third Week.	Increase.	Fourth Week.	Increase.
1	107	116	+9	125	+9	147	+22
2	102	114	+12	123	+9	134	+11
3	101	107	+6	117	+10	131	+14
4	98	106	+8	110	+4	124	+14
5	95	106	+11	107	+1	109	+2
6	87	93	+6	100	+7	109	+9
7	72	75	+3	86	+11	97	+11
8	70	73	+3	77	+4	87	+10
9	68	72	+4	77	+5	87	+10
10	57	60	+3	67	+7	82	+15
11	55	59	+4	66	+7	82	+16
12	51	58	+7	63	+5	73	+16
13	49	56	+7	57	+1	75	+18
14	49	55	+6	57	+2	72	+15
15	47	53	+6	55	+2	65	+10
16	46	53	+6	55	+2	63	+8
17	46	51	+5	54	+3	61	+7
18	45	50	+5	52	+2	57	+5
19	44	48	+4	45	-3	57	+12
20	42	46	+4	44	-2	43	+5
21	42	45	+3	44	-1	48	+4
22	38	40	+2	42	+2	42	+0
23	38	40	+2	39	-1	40	+1
24	37	37	+0	35	-2	39	+4
25	35	32	-3	33	+1	Dead	—
26	27	32	+5	31	-1	32	+1
1548			+129		+84		+240

Allowing for one death the percentage increase is in four weeks 30.07.

Two control rats fed on white bread and milk, bacon and cheese:

First Week.				Fourth Week.			
32	...	...	...	...	...	52	...
32	...	...	...	...	...	55	...
64				107			

Increase in four weeks 43. Percentage increase in four weeks 67.

TABLE II.
Standard Flour.
Weights of rats in grams.

Rat.	First Week.	Second Week.	Increase	Third Week.	Increase.	Fourth Week.	Increase.
1	108	107	-1	118	+11	118	+0
2	100	101	+1	110	+9	117	+7
3	91	99	+8	104	+5	114	+12
4	95	97	+2	103	+6	104	+1
5	84	89	+5	died	—	—	—
6	74	76	+2	80	+4	87	+7
7	72	67	-5	67	+0	76	+9
8	72	65	-7	68	+3	71	+3
9	63	62	-1	63	+1	67	+4
10	60	59	-1	62	+3	64	+5
11	57	59	+2	63	+4	60	-3
12	51	56	+5	56	+0	61	+5
13	51	53	+2	55	+2	57	+2
14	49	51	+2	53	+2	56	+3
15	48	49	+1	52	+3	56	+4
16	47	48	+1	49	+1	54	+5
17	46	48	+2	48	+0	53	+5
18	45	47	+2	48	+1	52	+4
19	44	46	+2	48	+2	51	+3
20	44	45	+1	47	+2	50	+3
21	42	45	+3	46	+1	50	+4
22	42	43	+1	43	+0	50	+7
23	41	42	+1	42	+0	45	+3
24	36	40	+4	40	+0	45	+5
25	35	37	+2	39	+2	35	-4
26	33	35	+2	38	+3	dead	—
	1530		+36		+65		+94

Eliminating animals 5 and 26, which died, the percentage increase in four weeks is 13.02.

earlier in the digestion of standard flour than in white flour, and therefore better assimilated and less likely to be lost through bacterial action. To test this the flours were digested with two different pancreatic extracts (P and P¹), and the amount of tryptophane liberated estimated by (1) the glyoxylic test, (2) the bromine water test. Bacterial action was prevented by the addition of toluol.

In regard to the latter test we found that digestion *in vitro* was slow, and the test was not marked for several days. However, in five positive cases the test was in favour of the standard flour. In regard to the glyoxylic test, this was performed after acidifying with acetic acid, boiling, and filtering. In all cases the depth of colour was greater in the case of standard flour. In a few cases

TABLE IV.

Enzyme.	W.	St.	W.	St.	W.	St.	W.	St.
C.cm.								
1.0	A	A+	A	A	A	A+	A	A+
0.8	A	A+	A	A+	A	A+	A	A+
0.6	A	A+	A	A+	A	A+	A	A+
0.4	0	A	A	A+	A	A+	A	A+
0.2	0	0	0	A	A	A+	A	A+

W. = white; St. = standard flour.

A expresses the depth of glyoxylic test given by white flour.

white flour plus germ and the germ itself were digested in addition. It is worthy of note that the digestion of the germ by itself gave least evidence of liberation of tryptophane, yet when added to the white flour in the proportion of 5 per cent. it increased the colour depth of the test as

TABLE III.
White Flour.
Weights of rats in grams.

Rat.	First Week.	Second Week.	Increase.	Third Week.	Increase.	Fourth Week.	Increase.
1	106	108	+2	105	-3	104	-1
2	104	99	-5	99	+0	103	+4
3	103	99	-4	94	-5	92	-2
4	96	95	-1	dead	—	—	—
5	87	83	-4	83	+0	dead	—
6	82	79	-3	73	-6	68	-5
7	75	67	-8	67	+0	67	+0
8	71	66	-5	66	+0	67	+1
9	65	65	+0	64	-1	61	-3
10	59	61	+2	59	-2	59	+0
11	54	55	+1	57	+2	dead	—
12	53	51	-2	53	+2	52	-1
13	49	49	+0	51	+2	dead	—
14	49	49	+0	50	+1	47	-3
15	48	48	+0	49	+1	dead	—
16	48	48	+0	46	-2	42	-4
17	46	47	+1	45	-2	45	+0
18	46	46	+0	43	-3	43	+0
19	44	45	+1	43	-2	38	-5
20	44	45	+1	43	-2	37	-6
21	43	43	+0	40	-3	dead	—
22	42	43	+1	40	-3	33	-7
23	42	41	-1	40	-1	33	-5
24	40	39	-1	38	-1	dead	—
25	38	38	+0	36	-2	32	-4
26	37	38	+1	35	-3	dead	—
	1561		-24		-33		-41

Eliminating the figures of the 8 animals which died, the percentage decrease in four weeks is 8.4.

compared with that for white flour alone. This points to the presence of adjuvant bodies in the germ. Not only is the tryptophane-containing portion of the proteins split off in larger amount, but also more easily in the case of standard flour, as the table shows. Here an equal amount of flour suspension (2 c.cm.) was digested with 1.0 c.cm., 0.8 c.cm., 0.6 c.cm., 0.4 c.cm., 0.2 c.cm. respectively of the pancreatic juice.

It has been asserted that the decay of teeth in modern times is due to the fact that a greater acidity is developed in the mouth on chewing white bread than on chewing the old-fashioned household bread. We do not find this to be the case.

To test the amount of acidity developed while chewing in the mouth, 5 grams of bread (standard or white) were taken, and, after preliminary washing out of the mouth with distilled water, were chewed for two minutes. At the end of this time the bread was ejected into a beaker containing 10 c.cm. of distilled water, and thoroughly shaken for one minute. The titration was then made with $\frac{N}{10}$ NaHO. From the figures (Table V) it will be seen that there is little difference in the results obtained with the two breads. It is interesting to note that with two students working together (for example, A. and L.) the results would sometimes be exactly opposite.

After ejection the acidity continues to increase, and therefore a small margin of error must be allowed for the time of titration. In carefully conducted experiments with $\frac{N}{10}$ NaHO, M. F., working to time as near as possible, found that there was no difference in the two breads in his case.

In the table the letters denote the order in which the chewings were made. Both breads were slightly acid to begin with, to an equal extent. The acid developed is,

in part at any rate, lactic acid. Neither bread gave Uffelmann's or Hopkins's tests previous to the chewing. Both gave these tests markedly afterwards.

TABLE V.

Table showing Amount in C.cm. of N/10 NaHO required to Neutralize 5 Grams of Bread of well-known London Make after Chewing for Two Minutes.

Name.	Standard.	White.	White.	Standard	Order of Chewing.
B.	1.7	1.3	0.8	1.0	S. W. W. S.
H.	1.3	1.5	1.4	1.5	"
F.	0.8	0.9	1.2	1.0	"
M.	1.1	0.9	—	—	"
X.	1.5	1.8	1.9	1.3	"
A.	2.0	1.5	1.5	2.2	W. S. S. W.
J.	1.2	1.4	1.6	1.3	S. W. W. S.
Ba.	2.2	1.8	1.3	1.9	W. S. S. W.
Bo.	1.7	1.3	1.5	1.9	S. W. W. S.
L.	1.6	1.4	—	—	S. W.
C.	0.8	0.5	1.1	1.6	S. W. W. S.
P.	2.1	2.3	3.0	2.8	W. S. S. W.
S.	1.6	2.0	1.2	1.4	S. W. W. S.
Y.	1.5	1.9	1.1	1.0	W. S. S. W.
W.	1.8	1.4	1.3	1.8	"
V.	1.7	1.4	—	—	W. S.
Pr.	1.5	1.0	1.0	1.7	S. W. W. S.
M. F.	1.0	1.0	1.1	1.1	"

We draw attention to the fact that Reid Hunt⁴ has found mice more susceptible to poisoning by acetonitrile when fed on biscuit powder and water than when fed on oatmeal and water.

REFERENCES.

¹ BRITISH MEDICAL JOURNAL, May 13th. ² Ibid., May 6th. ³ United States Department of Agriculture Bulletin 156. ⁴ Treasury Department, U.S.A., Hygiene Bulletin No. 69, 1910.

Memoranda :

MEDICAL, SURGICAL, OBSTETRICAL.

THE USE OF X RAYS AND THE ADMINISTRATION OF METALLIC SILVER IN THE TREATMENT OF CERTAIN DISEASES OF THE ALIMENTARY TRACT.

(Preliminary Note.)

THE undoubted effect which x rays have in stimulating the healing of chronic ulcers on the external surface of the body led me, some years ago, to employ them in the treatment of presumed ulceration of the alimentary tract. I used a hard tube with an aluminium filter, and irradiated the skin area over the supposed site of the lesion. Benefit seemed to accrue in some cases, but I was only too conscious of the weak points of the method. The rays used were of a kind not commonly believed to possess any great therapeutic activity, and it is probable that only a small fraction of the total dose was absorbed by the diseased surface. I had had one or two complete failures, and was beginning to lose interest in the subject, when the publication of Sir J. J. Thomson's address describing Professor Barkla's experiments on secondary radiation¹ caused me to take up the matter afresh. The central fact which struck me was that silver emitted a radiation similar in its properties to the beta rays of radium. I obtained some of the metal in a chemically pure precipitated form, and administered it to myself in doses of 30 grains t.d.s. for a fortnight. As no evil results occurred, I concluded that it might be taken with safety. My idea was this: That the irradiation of a hollow viscus containing metallic silver would result in a "reflection" of soft secondary rays; and that, provided the primary stream were directed successively at various angles to the part affected, a number

of these must of necessity "strike back" at any diseased area in the walls of the organ.

I have made use of this principle now for a period of six months with considerable success, at least temporary. My cases include ulceration of the caecum, gastric ulcer, and doubtful malignant obstruction of the pylorus. It is, of course, much too early to speak of ultimate results, but I may at least claim for my method that it is a reasonable attempt to make practical use of the newly-discovered facts enunciated in the address referred to.

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AN OVERDOSE OF PHENACETIN.

A GENTLEMAN of vigorous physique was ordered a cachet containing 9 grains of phenacetin and 1 of caffeine during influenzal pyrexia. He took in error eight cachets within the space of one hour, containing 72 grains of phenacetin.

One hour after taking the phenacetin he felt nauseated and much depressed; the headache had gone, and he complained of giddiness. On rising from his bed he could scarcely stand, and had to be helped back. There was profuse perspiration. He vomited after a cup of tea, but no trace of the phenacetin was observed in the vomit.

Two hours after the phenacetin had been taken the temperature was subnormal, surface cold, countenance ashy-grey and drawn. There was marked cyanosis, the breathing was slow and laboured, the pulse irregular, slow, and weak. The patient complained of feeling very weak and of nausea. I gave him 2 oz. of whisky, which he was able to swallow without vomiting, and a hypodermic injection of $\frac{3}{16}$ grain of strychnine. Oxygen was administered freely, with marked effect upon the cyanosis and respirations.

The urgent symptoms passed within three hours. The next day the patient felt depressed, and cyanosis was still in evidence; on the following day he had completely recovered.

Rome.

A. G. WELSFORD, M.D., F.R.C.S.

LIVING CHILD AND DEAD FETUS.

THE following case may prove of interest alongside the one recorded in the BRITISH MEDICAL JOURNAL of February 4th, p. 252: On July 14th, 1900, I attended Mrs. C., 7 para; she informed me that she did not expect her confinement for fully a month, that she had not been in good health for about two or three months, that she felt "queer," being easily tired, unable for her work, very restless and sleepless at nights, and that she had not had similar feelings in her previous pregnancies. She had been in town in the afternoon for two hours and returned so exhausted that she went to bed. An hour later labour commenced. She gave no history of abortion or miscarriage. She was seen about five hours after labour began, the pains being fairly strong; the os uteri was found to be almost fully dilated. There was no protrusion of the membranes during the pains, nor did they get much tenser, but they felt thickened, and lying within them a number of small loose bones were made out. As the patient felt tired an anaesthetic was given, when it was clearly made out to be a dead fetus. The thickened membranes were ruptured and fully 6 oz. of a dark, non-offensive fluid, resembling thickened brown soup, came away. The dead fetus was easily removed. Enclosed in a separate sac, lying well up in the uterus, was a living child; the membranes were ruptured, a foot seized, and delivery was easily effected. There was one placenta of large size, which appeared quite healthy, but two sacs of membranes.

The dead fetus was slightly macerated; it was 4 to 5 in. long, the skull bones lying loose inside the scalp. The epidermis was gone, the cutis of a dark red colour, the umbilical cord shrivelled. The fetus had the appearance of having died about the fifth month of pregnancy.

The living child was a female of small size, fully formed, but evidently three to four weeks premature, judging by the finger-nails. Artificial respiration was required for a few minutes after delivery. On the following day there was lividity of both legs, more pronounced in the left, which disappeared in a week. The child remained rather