acids is the fat of the body. In general it must be emphasised that an increase of the fat in food has no material influence on the formation and elimination of the acetone bodies. This depends in the first place upon the internal condition of the cell, and this condition is affected by a deficiency of carbohydrates (and therefore in the liver by deficiency of glycogen). The extent to which the metabolism of fats is affected in diabetes and diabetic coma may be judged from the fact that no less than 342 grammes have been found in the urine in the course of three days (Magnus Levy). The fats therefore yield the chief material necessary for the formation of acetone bodies and their formation in any notable quantity is in the first place caused by lack of carbohydrates. They are not formed in the intestine. They may possibly be formed in all organs but the chief seat of the formation is the liver and muscles.

Disturbances in hepatic fat metabolism are thus largely connected with deficiency of glycogen whether arising from want of carbohydrates (as in starvation) or from excessive loss of sugar (as in diabetes). The conditions in which increased acidosis arises are those especially affecting glycogenesis, such as poisoning with carbonic oxide, extirpation of the pancreas, poisoning with phloridzin, chloroform, exclusive feeding on animal fats, and lastly severe cases of diabetes.

DISTURBANCES OF PROTEOLYTIC AND ANTITOXIC FUNCTIONS.

The symptoms vary according to the degree with which the proteolytic functions are affected simultaneously.

1. If these latter are fully maintained the symptoms produced in severest cases are those of diabetic coma. In these conditions, owing to the absolute deficiency of carbohydrate and the excessive movement of fat from the tissues to the liver the acidosis is absolutely excessive. The proteolytic functions of the liver are, however, fully maintained.

2. The chief effect of such disturbances of fat metabolism arises when an acidosis of lesser degree is combined with disturbance of proteolytic activity of the liver cell. This is the condition presented in the final stages of severe liver disease such as cirrhosis, acute yellow atrophy, phosphorus poisoning, or chloroform poisoning. A disturbance in fat metabolism (with increased acidosis) which would not affect a fairly healthy liver cell becomes then much more important. At a time when the intracellular alkalinity of the liver cell is already at a low level (owing to inanition, deprivation of food, and diminished absorption of ammonia in the portal blood consequent thereon) the additional acidosis arising from excessive fat metabolism may, as already described, suffice to lower the alkalinity below the point necessary to keep its own digestive proteolytic ferments in check. There then results auto-digestion or autolysis of the liver cell by its own ferments, with complete arrest of all its functions. The clinical picture then presented in slighter or severe degree is not that of acidosis, as in diabetic coma, but that of acute yellow atrophy and chloroform poisoning—viz., essentially one due to the abolition of all the antitoxic functions of the liver. The symptoms largely resemble those produced when the portal blood is conveyed directly into the general circulation by means of an Eck's fistula.

CONCLUSION.

This conclusion which one thus reaches as to the relation of disturbances in fat metabolism to the severest forms of liver disturbance represented by acute yellow atrophy and chloroform poisoning is, in my judgment, of great practical importance. The vomiting which occurs after administration of anæsthetics is not of nervous origin; but it is, I consider, essentially toxæmic, due to the profound depression of liver function with consequent diminution in its antitoxic function during the period of the administration. This depression will be the greater if a liver already weakened by disease or by poor nutrition be further unduly weakened by food having been withheld for many hours before the administration. This enforced abstention from food before administration of an anæsthetic may thus in individual cases be carried too far, and it is, in my opinion, largely responsible for the fatal effects of delayed chloroform poisoning in exceptional cases. Such effects could in all probability be completely prevented if, instead of withholding food, particular care was taken that the patient had always a very nutritious and easily digestible meal, well-sweetened, two or three hours before the operation.

Harley-street, W.

OBSERVATIONS ON THE BACTERIOLOGY OF SCARLET FEVER.

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THE following are the results of an investigation of the organisms present in the throats, noses, &c., of a series of 160 cases of scarlet fever admitted to the wards of the Bury and District Joint Hospital during the years 1906-07, together with an examination of the throats of 40 healthy individuals.

Throat.—The first step was the general classification of organisms found in cultures on blood serum from swabs taken from the pharynx and fauces; in the case of the hospital patients these were made immediately on admission. The colonies which appeared included streptococci, diplococci, staphylococci, bacilli, and yeasts in various combinations, and their frequency was as follows:—

Organism.	Scarlet fever.	Healthy.
Streptococci	134, or 84 per cent.	33, or 83 per cent.
Diplococci	125, or 78 "	35, or 88 "
Staphylococci	98, or 61 "	19, or 48 "
Yeasts	16, or 10 "	3, or 8 "
Bacilli	77, or 48 "	20, or 50 "
Bacillus of Klebs-Löffler	14, or 9 "	—

Among the "bacilli" are included all forms of "diphtheroid," only the true diphtheria bacillus being separately classified.

In this list the close correspondence between the two columns is very striking, the only point of marked difference being the more frequent occurrence of staphylococci and diphtheria bacilli in scarlet fever. In none of these patients was there any clinical sign or symptom of diphtheria.

Nasal discharge.—During the same period the purulent nasal discharge was examined in 67 cases of scarlet fever and there were found:—

Streptococci in 28, or 42%	Bacilli in 41, or 61%
Diplococci " 34, or 51%	Bacillus of Klebs-Löffler
Staphylococci " 50, or 75%	in 25, or 37%
Yeasts " 1, or 2%	

Here the diphtheria bacillus occurred more frequently than in the throat, while streptococci were less frequent and staphylococci more frequent.

Ear discharge.—The ear discharge was examined in 17 cases of scarlet fever, with the following result:—

Streptococci appeared in 7, or 41%	Bacilli appeared in 8, or 47%
Diplococci " " 10, or 59%	Bacillus of Klebs-Löffler
Staphylococci " " 11, or 65%	appeared in 5, or 29%
Yeasts " " none.	

Thus streptococci and diplococci occurred in much the same proportion of cases in the ear as in the nose, and in considerably less than in the throat, while staphylococci were more frequent than in the throat and less frequent than in the nose. The diphtheria bacillus was relatively much more common than in the throat, though not quite so common as in the nasal discharge.

Pus.—On opening a cervical gland abscess in six cases the pus gave a pure culture of streptococci in each, as also in two cases in which the pus was obtained from the mastoid antrum.

Blood.—Cultures (both solid and liquid) were made from the blood of six cases on the second day of the disease and a streptococcus with chains of from six to ten individuals was obtained from one, a diplococcus without chain formation from another, while the remaining four gave no growth.

The occurrence of streptococci in pure culture in so true secondary conditions as the adenitis and mastoid infections, taken together with the frequency of their occurrence in the throat, is strong evidence that they play an important etiological part in at least the secondary symptoms of scarlet fever.

Examination of streptococci by Gordon's tests.—The next step undertaken was a full and detailed examination of the streptococci obtained from scarlet fever cases (chiefly from the throat, but also from the nose, ear, and mastoid) and from the throats of healthy individuals and others suffering

Table showing Cultural Reactions of Streptococci from Various Sources, as described in the Text.

No.	Andrews's and Horder's classification.	Reaction to Gordon's tests.										Growth on gelatin.	Morphology.	Scarlet fever.		Healthy.		Other diseases.			
		Litmus milk.		Neutral red.	Saccharose.	Lactose.	Raffinose.	Inulin.	Salicin.	Coniferin.	Mannite.			Nitrate.	No. of specimens.	No. of cases.	No. of specimens.	No. of cases.	No. of specimens.	No. of cases.	
		Acid.	Clot.																		
1	I., 1a	-	-	-	+	+	-	-	+	(-)	-	-	+	M. to L.	1	1	1	1	2	2	
2	1c	+	-	-	+	+	-	-	-	(-)	-	-	+	C. & M. to L.	5	4	3	3	2	2	
3	1f	-	-	-	-	+	-	-	-	(-)	-	-	+	M.	-	-	1	1	-	-	
4		-	-	-	-	-	-	-	-	-	-	-	+	M. to L.; C.	-	-	1	1	2	1	
5		-	-	-	+	-	+	-	-	-	-	-	+	M. to L.	1	1	2	2	-	-	
6		+	-	-	+	-	+	-	-	-	-	-	-	L.	1	1	1	1	-	-	
7	1h	-	-	-	+	-	-	-	-	(-)	-	-	+	M. and Liss.	1	1	1	1	-	-	
8	1i	+	-	-	+	+	-	-	+	(+)	-	-	-	M. to L.	1	1	-	-	1	1	
9	II., 2d	+	+	-	+	+	-	-	+	(+)	-	-	-	B. to M.	1	1	1	1	-	-	
10	2f	+	+	-	+	+	+	-	+	(+)	-	-	+	B.	-	-	1	1	-	-	
11	2i	+	+	+	+	+	+	-	+	(+)	-	-	+ Liq.	B.	-	-	1	1	-	-	
12	2l	+	-	+	+	+	+	-	+	(-)	-	-	+	M. to L.	-	-	1	1	-	-	
13		+	-	+	+	+	+	+	-	-	-	-	+	M. to L.	-	-	-	-	1	1	
14	III., 3a	+	+	+	+	+	+	-	-	(-)	-	-	+	L. to Liss; C.	4	3	1	1	1	1	
15		+	+	+	+	+	-	-	-	-	-	-	+	C.	1	1	-	-	-	-	
16	3b	+	+	+	+	+	-	-	-	(-)	-	-	+	M. to Liss; C.	7	4	1	1	7	3	
17		+	+	+	-	+	-	-	-	-	-	-	-	Liss.	-	-	-	-	1	1	
18	3c	+	+	+	+	-	-	-	-	(-)	-	-	+	M. to Liss; C.	8	6	-	-	6	3	
19		+	+	-	+	-	-	-	-	-	-	-	+	L. to Liss; C.	4	4	2	2	1	1	
20		+	+	+	-	-	-	-	-	-	-	-	+ Liq.	L.	-	-	1	1	1	1	
21		+	+	-	-	-	-	-	-	-	-	-	+	M. to L.; C.	1	1	2	2	-	-	
22		-	+	-	+	-	-	-	-	-	-	-	+	L. to Liss.	3	2	-	-	-	-	
23		-	+	-	-	-	-	-	-	-	-	-	+	L. to Liss.	2	2	-	-	-	-	
24	3d	+	+	-	+	+	-	-	-	(-)	-	-	+	M. to Liss; C.	6	4	5	4	6	4	
25	3e	+	+	-	+	+	+	-	-	(-)	-	-	+	M. to Liss; C.	6	6	4	4	1	1	
26	3f	+	+	-	+	+	-	-	+	(+)	-	-	+	M. to L.; C.	7	7	1	1	4	1	
27		+	+	-	+	+	-	-	+	+	-	-	+ Liq.	L.	-	-	-	-	1	-	
28		+	+	-	+	+	+	+	-	-	-	-	+	L.	1	1	-	-	-	-	
29		+	+	-	-	+	+	-	-	-	-	-	+	M. to L.	2	1	-	-	-	-	
30		+	+	-	-	+	+	-	-	-	-	-	-	L.	-	-	-	-	1	1	
31	3i	+	+	-	+	+	+	-	+	(+)	-	-	+	M. to Liss; C.	5	4	5	4	-	-	
32	3j	+	+	+	+	+	-	-	+	(-)	-	-	+	M. to Liss; C.	5	4	-	-	2	2	
33		+	+	+	+	+	-	-	+	-	-	-	+	Liss.	1	1	-	-	-	-	
34		+	+	+	+	+	-	-	-	-	-	-	+	L.; C.	1	1	-	-	1	1	
35	3k	+	+	+	+	+	+	-	+	(+)	-	-	+	M. to L.	6	4	2	2	3	2	
36		+	+	+	+	+	+	+	+	+	-	-	-	M. to Liss.	2	2	-	-	1	1	
37		+	+	+	+	+	+	+	+	-	-	-	-	M. to L.	1	1	-	-	2	1	
38		+	+	+	+	+	+	+	+	+	-	-	+	L.	1	1	-	-	-	-	
39		+	+	+	+	+	+	+	+	-	-	-	+	L.	1	1	-	-	-	-	
40		+	+	+	+	+	-	+	+	-	-	-	-	Liss.	1	1	-	-	-	-	
41		+	+	+	+	+	-	+	+	-	-	-	-	L.	1	1	-	-	-	-	
42		+	+	+	-	-	-	+	-	-	-	-	+	M.	-	-	1	1	-	-	
43	3m	+	+	-	+	+	-	+	-	(-)	-	-	-	B. to M.	-	-	1	1	-	-	
44		+	+	-	+	+	-	+	+	-	-	-	+ Liq.	C.	-	-	1	1	-	-	
45	3n	+	+	-	+	+	-	+	-	(-)	-	-	+	Liss.	1	1	-	-	1	1	
46		+	+	-	+	+	-	+	+	-	-	-	-	L.	1	1	-	-	-	-	
47		-	+	-	+	+	-	+	+	-	-	-	+	L.	1	1	-	-	-	-	
48		-	+	-	-	-	-	-	+	-	-	-	+	L.	1	1	-	-	-	-	
49	3o	-	-	+	+	-	+	-	-	(-)	-	-	-	M.	1	1	-	-	-	-	
50		-	-	+	+	+	+	-	-	-	-	-	+	Liss.	1	1	-	-	-	-	
51	3s	-	-	+	+	+	+	-	-	(-)	-	-	+	M.	-	-	1	1	-	-	
52		+	-	+	+	+	+	+	-	-	-	-	-	L.	1	1	-	-	-	-	
53		+	-	+	+	+	+	+	-	-	-	-	-	C.	1	1	-	-	-	-	
54	3l	+	-	+	+	+	+	-	+	(+)	-	-	-	M. to L.	2	2	1	1	-	-	
55	IV.	+	+	+	+	+	-	-	+	+	-	-	-	Liss.	-	-	-	-	1	1	
56		+	+	+	+	+	-	-	-	-	+	-	+	Liss.	1	1	-	-	-	-	
57		-	+	+	+	+	-	-	+	-	+	-	+	L.	-	-	1	1	-	-	
58	4b	+	+	-	+	+	+	+	+	(-)	-	-	+	M.	-	-	1	1	-	-	
59	4c	+	+	-	+	+	+	+	+	(-)	-	-	+	M. to L.	-	-	1	1	-	-	
60		+	+	-	+	+	+	+	+	+	-	-	+	C.	-	-	1	1	-	-	
61		+	+	+	+	+	+	+	+	-	-	-	-	L.	1	1	-	-	-	-	
62		+	+	-	+	+	-	-	+	-	-	-	+	L.	-	-	-	-	1	1	
63		-	-	-	-	+	-	+	-	-	-	-	-	L.; C.	-	-	2	2	-	-	
64		-	-	-	-	+	+	-	-	-	+	-	+	L.	-	-	1	1	-	-	
																100	27	50	20	50	12

from catarrhal and diphtheritic affections. In all 200 specimens were taken from the following sources for comparison. I. 100 separate specimens from 27 scarlatinal patients, viz.:—80 specimens from 26 different throats, 6 from 2 different noses, 8 from 2 discharging ears (different patients), and 6 from 2 different cases of mastoid infection. II. 50 specimens from 20 different healthy throats. III. 50 specimens from 12 cases of other diseases, not scarlet fever—viz: 30 specimens from 6 different diphtheria throats, 13 from 5 different catarrhal throats, and 7 from 1 case of follicular tonsillitis with abscess formation. Swabs were taken from each case and nasagar plates inoculated from them and incubated at 37° C. for 24 hours.

The resultant colonies were then examined macroscopically and under the low power; subcultures of different colonies were made in lemco bouillon at 37° C. for 48 hours, and the growth examined macroscopically and microscopically. On finding streptococci in the bouillon, these were re-subcultured after Mervyn H. Gordon's method¹ in the following media: (1) Litmus milk; lemco-litmus broth containing (2) saccharose, (3) lactose, (4) raffinose, (5) inulin, (6) salicin, (7) mannite, and (8) potassium nitrate, all in 1 per cent. solution, and (9) neutral red broth, the last anaerobically in Buchner's tubes, all at 37° C.; and (10) on gelatin at 20° C. Coniferin was unfortunately unobtainable either in the home or foreign market. Incubation of the subcultures was maintained for seven days and record then made of the decomposition of the various sugars from the colour of the media, &c. As far as possible the investigation was made to correspond, for the sake of comparison, with that of Dr. F. W. Andrewes and Dr. T. J. Horder, described in THE LANCET of Sept. 15th, 22nd, and 29th, 1906.

Taking into account only the morphological differences between individual specimens as seen in the growth in bouillon, the following classification may be made according to the length of the chains, percentages alone being stated:—

Chains.	Healthy.	Scarlet fever.	Other conditions.
Longissimus (Liss)	8	20	18
Longus (L.)	34	48	38
Medius to Longus (M. to L.) ...	14	14	10
Medius (M.)	22	8	8
Brevis to Medius (B. to M.) ...	2	—	—
Brevis (B.)	4	1	—
Conglomeratus (C.)	16	9	26

The longer forms of streptococci then appear to be relatively more numerous in the diseased than in the healthy conditions, the short forms appearing relatively more numerous in the healthy throats. The results pointed to what Andrewes and Horder² referred to as a replacement of the common short forms by long varieties quite early in the disease. It will be noticed that the conglomerate forms, described by Gordon as being peculiar to scarlet fever, were found much less frequently in scarlet fever than in health, and very much less frequently than in other catarrhal conditions of the throat.

In classifying the specimens the method adopted by Andrewes and Horder was as closely adhered to as possible. Macroscopically the appearances were generally similar to those noted by the same observers. The accompanying large table summarises the results obtained from the subcultures, arranged on the system suggested by Andrewes and Horder. Where one of the present series corresponds with one of theirs, their classification (Group and Variant) numbers are indicated in italics, and also their result with the coniferin broth medium in brackets. Decomposition of the media is indicated in the table by a positive (+) sign and no visible reaction by a negative (—). Two points of importance which do not appear in the table are: (1) that two or more specimens from a single case often differ from one another in only a single reaction; and (2) that no coincidence was found in any scarlet fever case between organisms obtained from the ear when discharging or from the mastoid antrum and any that were obtained from the throat. In one case an organism was obtained from the nasal discharge which gave the same reactions as one of those found in the throat. It will be seen that out of 200 specimens 64 variants were obtained.

The following table shows the different distribution of the

variants (percentages only) in the groups suggested by Andrewes and Horder:—

Group.	Healthy.	Scarlet fever.	Other diseases.
I.	20	10	14
II.	8	1	2
III.	58	87	80
IV.	14	2	4

It is obvious that very little can be learnt for purposes of diagnosis from these results, taking the reactions in their series. Subjoined is a table of percentages comparing the reactions of organisms from the different sources to the individual tests, and it is also obvious that they are equally valueless for the purpose. Mannite alone is more readily decomposed by organisms from healthy throats than from diseased, but there is no test among them by which scarlet fever can be distinguished from the other diseases.

Test.	Healthy.	Scarlet fever.	Other diseases.
Milk pink	78 per cent.	88 per cent.	92 per cent.
Milk clot	68	83	84
Neutral red	22	48	54
Saccharose	86	92	90
Lactose	72	68	72
Raffinose	50	34	20
Inulin	6	12	12
Salicin	34	33	32
Mannite	14	2	4

With a much smaller number of specimens of streptococci (33) from scarlet fever cases, Andrewes and Horder obtained results with which the above correspond most strikingly. As a means therefore of diagnosis in scarlet fever, Gordon's tests must be regarded as of little value in the present state of our knowledge of the etiology of the disease. If the tests are of real differential worth for the various strains of streptococci—and the remarkable constancy shown by different generations of individuals to them, in my own experience, indicates that they are—we find ourselves faced by the fact that they fail absolutely to point to any particular strain as being even more frequently present in scarlet fever than in other catarrhal and inflammatory conditions of the throat. That the infecting agent is present in the pharyngeal and faucial mucus is absolutely all that has been established for certain regarding the causation and source of the disease. If none of the streptococci in the throat are causative of the primary condition some other agent must have made it possible for them to obtain entrance to the tissues in order to produce the secondary complications. It was shown above that the staphylococci are relatively increased in scarlet fever. Possibly a careful examination of these, on similar lines to that described for the streptococci, might reveal something of importance.

I am indebted to Dr. A. E. Brindley, medical superintendent of the Bury and District Joint Hospital, for permission to publish the above.

Newcastle-upon-Tyne.

UNIVERSITY COLLEGE HOSPITAL.—The annual general meeting of the supporters of this hospital was held on March 26th, under the presidency of Mr. Henry Lucas, treasurer to the charity. The report on the work of the hospital during the past year stated *inter alia* that owing to lack of means the committee has been unable to open a ward containing 24 beds which should be available for the sick poor; in other words, about 350 patients annually, many requiring immediate treatment in the wards, have to be refused admission. An additional annual expenditure of about £1200 would be required to keep the ward open, and the committee has not felt justified in incurring this further liability, when in order to meet the deficiency in income it has had to trench upon its small invested funds by the realisation of £1500 Consols, and this notwithstanding that it has had to devote to purposes of current expenditure legacies which in more prosperous times could have been invested. The committee urgently appeals to the benevolent to do their utmost to relieve it from the financial anxieties and difficulties with which it is constantly faced, as it is obvious that only by strenuous efforts on the part of everyone interested in the welfare of the hospital can its efficiency be maintained.

¹ Report of the Medical Officer to the Local Government Board, 1903-04.

² A Study of the Streptococci Pathogenic for Man, THE LANCET, 1906, vol. ii., pp. 708, 775, and 852.