8480 C

Iodine Number of Serum Fatty Acids in Acute Infections of Infants With and Without Eczema.

A. V. STOESSER. (Introduced by Irvine McQuarrie.)

From the Department of Pediatrics, University of Minnesota.

The changes which take place in the cholesterol content of the serum during the acute infections occurring in the infants observed in the preceding paper[1] are accompanied by a fall in the plasma total fatty acids. The serum unsaturated fatty acids have already been found to be replaced in part by more and more saturated acids during acute respiratory infections in older children.[2] In infants the observation has been made that eczema is associated with an abnormally low serum content of unsaturated fatty acids[3] but no study has been recorded of the influence of acute infection on the unsaturation of the serum fatty acids.

This paper presents the observation made in 14 infants with acute illnesses. They are the same babies which were chosen for the cholesterol study. All blood samples obtained during and after the height of each infection were drawn between 12 and 16 hours after a meal. The first blood sample was collected as soon as possible after the infant was admitted to the hospital at the height of the disease and the second blood sample after the seventh day of convalescence. Bloor's methods[4] were followed to determine the blood lipids. The Rosenmund-Kuhnhenn method[5] as modified by Page, Pasternak and Burt[6] was used to determine the iodine absorption of the serum fatty acids.

The results are summarized in Tables I and II.

The fall in plasma total fatty acids which takes place during the height of acute infections in infants is paralleled by a drop in the iodine absorption values. This signifies that in most instances the iodine number of serum fatty acids is definitely lower in the febrile period of the disease than during the period of recovery. Acute infection produces in the infant the same changes previously found to occur in the older child. The serum fatty acids are less

[1] Stoesser, A. V., PROC. SOC. EXP. BIOL. AND MED., 1936, **34**, 10.

[2] Stoesser, A. V., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 1326.

[3] Hansen, A. E., PROC. SOC. EXP. BIOL. AND MED., 1933, **30**, 1198.

[4] Bloor, W. R., *J. Biol. Chem.*, 1928, **77**, 53.

[5] Rosenmund, K. W., and Kuhnhenn, W., *Z. f. unter. d. Nahr. d. Nahr. u. Genuss.*, 1923, **46**, 154.

[6] Page, H. H., Pasternak, L., and Burt, M. L., *Biochem. Z.*, 1930, **223**, 445.

TABLE I.
Iodine Numbers of Serum Fatty Acids in Infections of Upper Respiratory Tract and in Pneumonia of Infants without Eczema.

Case No.		Total fatty acids		Iodine absorbed		Iodine number	
		Mg. per 100 cc. serum					
A	B	A	B	A	B	A	B
			Height of disease				
1	8	426	304	416	252	97	82
2	9	278	323	303	256	108	79
3	10	390	311	350	289	89	92
5	11	312	274	229	248	73	90
6	12	257	304	234	246	91	80
7		414		356		85	
Aver.		346	303	314	258	90	84
			Period of convalescence				
1	8	471	342	545	332	113	97
2	9	350	373	413	358	118	96
3	10	448	335	463	433	103	129
5	11	306	406	255	475	83	117
6	12	257	361	263	435	102	120
7		457		435		95	
Aver.		381	363	395	406	102	111

A—Infections of Upper Respiratory Tract.
B—Pneumonia.

TABLE II.
Iodine Numbers of Serum Fatty Acids in Acute Infections of Respiratory Tract of Infants with Eczema.

Case No.	Total fatty acids	Iodine absorbed	Iodine number
	Mg. per 100 cc. serum		
	Height of infection		
1	316	258	81
2	375	403	107
2	395	341	86
3	501	418	83
3	397	373	93
Aver.	396	358	90
	Afebrile periods		
1	402	364	90
1	342	331	97
2	432	389	90
3	513	488	95
3	444	467	105
3	540	600	111
Aver.	445	439	98

unsaturated during an acute illness. The already moderately low iodine number of serum fatty acids in the eczematous babies is still more reduced by acute respiratory disease, and there is a tendency for it to remain at low levels for some time. This slow return to higher levels was also observed in the infants without eczema. However, the pneumonia cases had a more rapid fall in the iodine

number with a rather quick return to normal levels after the pneumonia had completely resolved, leaving no complications.

Incidentally a very interesting observation was made in connection with eczematous babies. During the first half of the acute illness the eczema improved remarkably although no special treatment of the skin was instituted. Then at the end of the febrile period, the skin became worse, remaining in this condition for weeks. This period of improvement may coincide with the sudden flood of unsaturated fatty acids into the blood stream which Boyd[7] claims occurs at onset of fever. In one instance an eczematous infant fairly well under control developed an acute illness with fever, and the eczema became worse without the initial phase of improvement. The effect of acute infections on the course of infantile eczema is the subject of further investigations which are in progress.

8481 C

Water Metabolism of the Rat Following Removal of the Anterior Lobe of the Hypophysis.*

RICHARD I. PENCHARZ, JAMES HOPPER, JR., AND EDWARD H. RYNEARSON. (Introduced by H. M. Evans.)†

From the Institute of Experimental Biology, University of California, and the Division of Medicine, The Mayo Clinic, Rochester, Minn.

There is considerable evidence that experimental diabetes insipidus is neither strictly an endocrine disturbance nor entirely an involvement of the brain but probably the result of both. Rats can be rendered diabetic by removal of the posterior lobe or by a stab wound in the base of the brain, providing the latter injury is such that it completely severs the stalk from its attachment to the brain.[1,2]

Fisher, Ingram and Ranson,[3] availing themselves of the Horsley-

[7] Boyd, E. M., *Canadian Med. Assn. J.*, 1935, **32**, 500.

* Aided by a grant for the study of the metabolic relations of the anterior hypophysis contributed by Robert R. Presnell, Frank Tuttle, Arthur Stebbins, and Mrs. Gordon Kahn, of Los Angeles.

† The authors wish to thank Miss Frances Dobell for her valuable assistance in this work.

[1] Richter, C. P., *Am. J. Physiol.*, 1933, **106**, 80.

[2] Richter, C. P., *Am. J. Physiol.*, 1934, **110**, 439.

[3] Fisher, C., Ingram, W. R., and Ranson, S. W., *Arch. Neurol. and Psychiat.*, 1935, **34**, 124.