centres, ou bien aux extrémités des fibres nerveuses périphériques, des modifications cycliques qui ne concernent que les terminaisons nerveuses libres. Pas plus qu'ailleurs, le tissu nerveux ne montre à ce niveau des réseaux continus dans lesquels se perdraient les filaments les plus fins.

HORACE MICHELI

Laboratoire de neuro-histologie, Institut d'anatomie de l'Université de Genève, le 18 juillet 1951.

Summary

In the molecular layer of the cerebellum of the rat, alterations have been shown of the fine and free nervous endings, as described by WEBER. It seems, therefore, that there are no nervous reticular formations, but free endings in this layer of the cerebellum.

Examination of the Vitamin-E Barrier of the Placenta

According to previous clinical studies[1], prematurely born infants suffer from a deficiency in vitamin E in the first days of their life. Under pathological conditions the deficiency may often give rise to scleroedema. As this condition could promptly be influenced by administration of tocopherol, it has been supposed that scleroedema is a deficiency disease[2]. It seemed, therefore, of importance further to investigate the physiological conditions.

The level of tocopherol in human blood is, according to the literature, considerably variable (ABDERHALDEN[3], D'OLIVEIRA[4], MINOT[5], QUAIFE and HARRIS[6], VINET, ANDRÉE and MEUNIER[7]). The difference in the results is partly due to the fact that the blood level of tocopherol is greatly dependant on seasonal, nutritional and other factors, and partly that the results given by the different potentiometric, volumetric, ferro-colorimetric, and other methods, of assay used, cannot be compared with each other. Literary data on the tocopherol content of the blood of pregnant women are greatly different, sometimes even conflicting (ABDERHALDEN[8], VARANGOT[9], VARANGOT, CHAILLEY and RIEUX[10], STRAUMFJORD and QUAIFE[11]). According to ATHANASSIU[12], after delivery the tocopherol level increased during pregnancy returns to its original value. According to VARANGOT, CHAILLEY and RIEUX[13], the level drops from 320 γ per 100 ml to 220 γ; according to STRAUMFJORD and QUAIFE[14], from 1070 γ to 340 γ. ATHANASSIU[15] found tocopherol values of 1250 to 2000 γ per 100 ml in the venous blood of the umbilical cord while only 200 to 600 γ per 100 ml in the arterious blood. – According to MOYER[1], the tocopherol level in the plasma or serum of both term and premature infants amounts, with great variations, to 230 γ per 100 ml. In infants born on term, the level of tocopherol appears to be higher after five days, averaging 360 γ per 100 ml.

The investigations to be discussed were carried out during winter months.

For the examinations, blood was withdrawn from healthy women in labour whose clinical history mentioned neither abortion nor premature delivery. The first sample of blood was taken at the beginning of delivery, the second one immediately after delivery had been completed, both from a brachial vein of the mother. Simultaneously with the second withdrawal of blood, a third sample was also taken, this one from umbilical vein. The fourth sample of blood was taken from the newborn infant during the next few days. Determination of tocopherol was carried out from 2 to 4 ml of serum by a micro method devised by one of us. The method, the details of which will be published elsewhere[2], consists of the following procedures. From a measured amount of serum previously made alkaline, lipids together with tocopherol are shaken out with petrolether of a low boiling point. Then the petrolether is removed and Furter and Meyer's reaction performed with the residue[3]. The "tocopherol red" thus obtained is separated by shaking out with petrolether, then transformed with orthophenylenediamine into a phenazine derivative. After separation by means of absorption chromatography, the fluorescence of the substance is estimated. The fluorescence of the thus transformed and isolated phenazine derivative of tocopherol offers adequate means for the quantitative determination of vitamin E. The maximum deviation of the method amounts about to 10 to 15 %.

No. of case	Tocopherol content of serum in mg/100 ml			
	Sample 1st	Sample 2nd	Sample 3rd	Sample 4th
2	216	210	90	ø
3	270	230	246	ø
4	875	858	304	378
11	277	258	154	257
13	333	402	137	256
20	552	477	185	257
22	764	649	298	218
23	652	791	214	296
27	316	405	413	371
31	620	542	318	422
32	462	334	307	354
33	1156	890	405	510
34	707	948	362	ø
35	936	1095	529	564
M	*581*	*578*	*283*	*353*
σ M	76·6	78·4	32·4	33·6
P. E.	50·5	52·9	21·9	22·7
S	0·004		1·8	
M	579		318	
σ M	77·5		33	
P. E.	50·2		22·2	
S	4·6—4·7			

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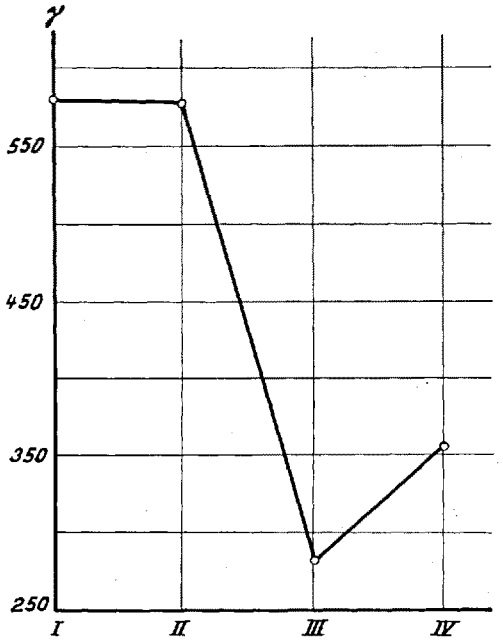
[1] W. T. MOYER, Pediatrics *6*, 893 (1950).

[2] B. BENCZE, Kémikusok Lapja (in print).

[3] B. BENCZE, Kémikusok Lapja *5*, 87 (1944); Hoppe Seyler's Zschr. *282*, 152 (1947).

A total of 53 determinations was performed in 14 patients. The results are presented in the table and the figure.



According to the results, the blood level of tocopherol in the mothers examined was remarkably inconstant, varying between 210 and 1090 γ per 100 ml. Cases Nos. 33, 34 and 35 were examined early in the spring, in the month of March. The exceptionally high tocopherol values yielded by them might perhaps be attributed to the more varied diet in that season. There was no significant difference in tocopherol content between the first and second samples of blood, those withdrawn from the mothers at different occasions. On the other hand, a notable difference was found to exist between the second and third samples, i. e. the blood of the mother and that of her newborn child, as the mother's blood contained about twice as much tocopherol as that of the umbilical vein. Further, the level of tocopherol in the blood of the umbilical cord was found to be lower than that of the newborn in 9 cases out of 11. The difference, which was not a significant one, seemed to be in connection with the onset of colostrum secretion. (According to WRIGHT, FILER, and MASON[1] the rapid increase of the tocopherol content in the blood of breast fed infants is due to the onset of the secretion of colostrum which has a high tocopherol level.) However, no final explanation can be offered before our serial observations of the variations of the level of tocopherol in the blood of newborn infants will have been concluded. The role of another factor, the dehydration of blood, possibly responsible for the increase of the tocopherol level in the newborn, will be clarified by observation of haematocrit values. A detailed study on these subjects is in the press.

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First Department of Pediatrics and Second Department of Obstetrics of the Budapest University Medical School, January 30, 1951.

Zusammenfassung

Wir haben an 14 Patienten insgesamt 53 Untersuchungen zur Bestimmung des Vitamin-E-Gehaltes im Blutserum durchgeführt, und zwar zur Bestimmung des Vitamin-E-Gehaltes im Blutserum der Mutter zu Beginn der Geburt (I) und unmittelbar nach der Geburt des Kindes (II); zur Bestimmung des Vitamin-E-Gehaltes der *Vena umbilicalis* der Nabelschnur (III) und schließlich zur Bestimmung des Vitamin-E-Gehaltes im Blutserum des Kindes (IV) in den ersten Lebenstagen des Säuglings. Die Bestimmungen beruhten auf Messung der Fluoreszenz des Phenazinderivates von Vitamin E (Tocopherol).

1. Zwischen dem Tocopherolgehalt des zu Beginn der Geburt (I) und unmittelbar nach der Geburt des Kindes, jedoch noch vor der Loslösung der Plazenta genommenen Mutterblutes (II) zeigte sich kein deutlicher Unterschied.

2. Zwischen dem Vitamin-E-Gehalt des von der gebärenden Mutter genommenen Blutes (II) und dem Vitamin-E-Gehalt des gleichzeitig der Nabelschnur entnommenen Blutes (Embryonalblut) war ein deutlicher Unterschied festzustellen: der Vitamin-E-Gehalt des Blutes der Mutter erwies sich im Durchschnitt doppelt so hoch als der des Nabelschnurblutes.

3. Während der ersten Lebenstage des Neugeborenen beobachteten wir eine schwache Tendenz zum Ansteigen des Vitamin-E-Gehaltes im Blutserum des Kindes.

4. Der Vitamin-E-Gehalt im Blute der Mutter ist auffallend schwankend.

[1] S. W. WRIGHT, JR. L. J. FILER, and K. E. MASON, Pediatrics *7*, 386 (1951).

Existence d'une relation fonctionnelle entre la glycémie et la vitesse d'absorption du glucose par l'intestin

Dans aucun mémoire nous n'avons trouvé l'hypothèse que la glycémie put intervenir pour régler la vitesse d'absorption du glucose par l'intestin. L'existence de ce mécanisme nous a été suggérée au cours d'un travail dans lequel nous avons obtenu les courbes *1* et *2* de la figure 1 qui représentent la cinétique de l'absorption intestinale du glucose chez le cobaye[1]. Cette idée s'est fortement imposée à notre attention lorsque, reportant sur le même graphique quelques-unes des données de FENTON[2] sur le rat, nous y avons retrouvé les particularités de nos propres courbes (courbe *3* de la figure 1).

Ces particularités sont l'existence de deux points d'inflexion correspondant aux abscisses 30 mn et 120 mn. Au premier point, l'absorption décroît; au deuxième, elle croît de nouveau. Que l'absorption s'accélère à la fin de l'épreuve, alors que la plus grande partie du glucose a disparu, paraît assez paradoxal. De toutes les explications que nous avons envisagées, une seule nous a paru mériter examen: celle d'une régulation de l'absorption par la glycémie elle-même. Celle-ci varie en même temps que l'absorption, mais en sens inverse: haute lorsque l'absorption est inhibée, normale ou basse dès que l'absorption recommence à croître[3].

L'expérience a confirmé notre hypothèse. Dans une brève note[4], nous avons signalé que l'on peut ralentir l'absorption d'une manière significative en élevant la glycémie dès le début de l'épreuve.

Dans ce travail nous nous sommes demandé s'il y a une relation quantitative et de quelle forme entre la glycémie et la vitesse d'absorption du glucose par l'intestin. Nous avons défini cette relation en envisageant uniquement le domaine des fortes glycémies, que nous avons obtenues, comme dans notre travail précédent[4],

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[4] M. LOURAU et O. LARTIGUE, C. r. Acad. Sci. Paris *232*, 1697 (1951).