and was completely free from all symptoms. She had greatly improved in strength at the time when she sought discharge.

Comments

Amongst all the vague and indefinite clinical details and numerous other drawbacks in the report of the above cases, two things emerge as definite and certain. They are: (1) the complaint-complex of the patients, and (2) the remarkable improvement in the clinical condition of the patients after the commencement of the course of streptomycin injections in micro-dosage. The lack of adequate facilities for detailed biochemical and other investigations has left us with the supreme clinical sense alone in the diagnosis and progress of the cases. As a matter of fact, we still lack convincing chemical tests to confirm the clinical diagnosis of early abdominal tuberculosis or cirrhosis of the liver. Abdominal tuberculosis can only be definitely diagnosed by laparotomy (peritoneoscopy not being available to us). But then, laparotomy itself would have been held as the major contributory factor in the improvement of the patients. As regards the various biochemical tests in the diagnosis of liver disease, it has been the general experience that, in a large number of cases, the entire battery of tests for liver function rarely takes the clinician a step further than what a thorough clinical examination can do. Liver biopsy would have clinched the diagnosis of cirrhosis, but it was believed to be a severe procedure for the debilitated cases. The presumptive clinical diagnosis in the cases described is by no means unquestionable. But whatever name may be given to the clinical condition it has improved on the described schedule of dosage of streptomycin.

The micro-dose schedule appeared to work only in early clinical cases suspected of abdominal tuberculosis. Three advanced cases of abdominal tuberculosis with œdema of feet, diarrhœa and marked emaciation, who were kept on this dose-schedule of streptomycin, did not appear to respond in the course of a week. They were later found to be refractory even to the usual doses.

A short explanation about some points arising from this paper is necessary:—

Selection of micro-dose.—This was done arbitrarily to suit the convenience of dilution. The dilution roughly approximates to between X and 2X of homœopathic potencies. *Average amount of streptomycin used for each case:* It is roughly 0.75 g. *Average number of injections given:* Twenty-five to 30, depending upon the clinical response.

Summary

Five cases with chronic abdominal complaints, including two with ascites, treated with daily injections of 0.025 g. of streptomycin, are reported. All the 5 cases showed remarkable clinical improvement during the course in the hospital and three for the short period of follow-up after their discharge, ranging from one to two months. An attempt is made to explain the probable mode of action of the unusually small dose of streptomycin.

I desire to express my thanks to Drs. Sule, Kelkar and Pendharkar working in my Unit, for their help.

REFERENCES

BINGALL, J. R., *et al.* *Brit. Med. J.*, *i*, 1224. (1950).

DESHMUKH, P. L. (1948). *Trans. Sixth All-India Tuberculosis Workers' Conference.* Tuberculosis Association of India, New Delhi.

EDITORIAL (1951) .. *J. Amer. Med. Assoc.*, **145**, 1269.

JAMES, L. A., SIDES, L. J., DYE, W. E., and DEYKE, V. F. (1951). *Amer. Rev. Tuberc.*, **63**, 275.

MARSHALL, E. K. (1951). *J. Amer. Med. Assoc.*, **146**, 1062.

MATTEI, C. C. (1951) . *Paris Med.*, **41**, 163. (Abstract—*J. Amer. Med. Assoc.*, **146**, 972.)

ASCORBIC ACID IN MILK AND PLASMA OF BENGALI WOMEN

By KALYAN BAGCHI

Assistant Professor of Nutrition and Dietetics, All-India Institute of Hygiene and Public Health, Calcutta

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Introduction.—Human milk is richer in ascorbic acid than cow's milk. The ascorbic acid in it has been determined by different investigators like Harris and Ray (1935), Selleg and King (1936), Gedda and Kjellberg (1939), etc., and the value varies from 1 to 10 mg. per 100 cc., but the majority of the investigators are of the opinion that with a satisfactory diet, the ascorbic acid varies from 4 to 8 mg. per 100 cc. of milk. Conclusive evidence has also been advanced to show that the ascorbic acid in milk depends on the dietary intake of the mother. Baumann and Rappolt (1937) consider 8 gm. per 100 cc. to be the maximum possible value for breast milk and that the ascorbic acid content of breast milk cannot be made to increase beyond that value, however great the intake of the mother.

The importance of ascorbic acid values of human milk lies on the fact that this is the only source of ascorbic acid for the infants, till they are weaned. Whether a breast-fed infant, without any supplement of ascorbic acid, can satisfy its requirement entirely through milk is a vital question. No work has been done in this line with Bengali women and the present investigation

was taken in hand for the clarification of this question.

The relationship between ascorbic acid in milk and that in plasma is also an interesting question. The source of ascorbic acid in milk is in all probability the plasma ascorbic acid. The natural conclusion is that the mammary gland receives the ascorbic acid from the plasma and secrete it after concentration. The possibility of concentration can easily be verified by having both the plasma and milk ascorbic acid values together. This was also explored in the present investigation.

Experimental.—Milk and plasma of 25 healthy lactating Bengali women were collected during different periods of lactation, varying from 1st to 7th month, at which period the infant is usually weaned. All these subjects came from the middle class and the ascorbic acid in their diet, as determined by questioning them on successive days, varied from 50 to 75 gm. per day. About 80 to 85 per cent of this ascorbic acid in their diet came from green leafy vegetables, which in the average Bengali middle class families, are taken in a cooked state, thus losing a large percentage of the vitamin. Citrous fruits were rarely taken. The infants of these mothers thrived entirely on breast milk. It was made sure that they were not given any supplement of ascorbic acid. The infants were examined and were found to be all healthy showing no clinical signs of ascorbic acid deficiency.

Milk and plasma were collected at the same time and almost always in the morning. The samples were collected in glass tubes with glass stoppers. The samples were brought to the laboratory immediately and analysed. In no case did the interval between the collection and the analysis exceed one hour. The samples were kept in the refrigerator as soon as they reached the laboratory. Both the plasma and the milk ascorbic acid were determined by titration with 2.6 dichlorophenolindophenol. For plasma, the method of Farmer and Abt (1936) was followed and for milk that of Richmond *et al.* (1940) was followed.

Table I shows the ascorbic acid values in human milk from 25 healthy Bengali women in different periods of lactation.

Table II shows the ascorbic acid values in both plasma and milk in the 25 subjects.

Discussion.—The average concentration of ascorbic acid in milk of healthy Bengali women is 6.7 mg. per 100 cc. with a standard deviation of 1.16 and the average concentration of ascorbic acid in plasma in this group of women is 0.78 mg. per 100 cc. with a standard deviation of 0.124. The figure for milk is the average for 1st to 7th month of lactation. The diet of the mother in this group is certainly not rich in ascorbic acid. In fact, the intake of the vitamin was either less than the recommended value or just on the border line. An infant, 5 months old for instance, weighing about 10 lb., would consume about 25 oz. of milk and would thus get about 48 mg. of ascorbic acid. According to the latest recommendation of the National Research Council of U.S.A., the requirement of an infant for ascorbic acid is 30 mg. per day. According to Hamil and co-workers (1938), the absolute

TABLE I

Serial number	Period of lactation	Ascorbic acid mg. per 100 cc. of milk
1	1st month	6.8
2	Do.	5.8
3	Do.	7.6
4	2nd month	6.7
5	Do.	8.2
6	Do.	8.6
7	Do.	7.3
8	3rd month	6.3
9	Do.	5.8
10	Do.	8.6
11	Do.	6.7
12	4th month	5.6
13	Do.	4.8
14	Do.	5.9
15	5th month	6.2
16	Do.	8.2
17	Do.	7.6
18	6th month	6.7
19	Do.	5.2
20	Do.	5.6
21	Do.	7.6
22	7th month	8.2
23	Do.	5.6
24	Do.	4.9
25	Do.	7.2

TABLE II

Serial number	Ascorbic acid mg. per 100 cc. of milk	Ascorbic acid mg. per 100 cc. of plasma
1	6.8	0.81
2	5.8	0.76
3	7.6	0.98
4	6.7	0.74
5	8.2	0.96
6	8.6	0.88
7	7.3	0.92
8	6.3	0.81
9	5.8	0.61
10	8.6	0.98
11	6.7	0.81
12	5.6	0.61
13	4.8	0.61
14	5.9	0.71
15	6.2	0.68
16	6.7	0.67
17	8.2	0.91
18	7.6	0.81
19	5.2	0.61
20	5.6	0.71
21	7.6	0.71
22	8.2	0.89
23	5.6	0.69
24	4.9	0.60
25	7.2	0.80

minimum of ascorbic acid required to protect infants under one year of age against scurvy is 10 mg. per day.

It is thus clear that an infant thriving on breast milk entirely requires no supplement of ascorbic acid, as the daily requirement is satisfied by breast milk. The position is quite different in case of cow's milk or any other artificial milk. The highest value obtained was 8.6 mg. per 100 cc., which goes against the view of Baumann and Rappolt (1937) that 8 mg. per 100 cc. is the maximum possible value. To all probability, an upper limit is there, as there must be a concentrating limit, but the maximum value is probably more than 8 mg. per 100 cc.

The relationship between the ascorbic acid of plasma and that in milk is noteworthy. On statistical analysis it was seen that the correlation between these two values is significant (+ 0.86). The relation between the two variants can be described, from this data, as follows :

$$y = 8.0970.x + 0.4506$$

where y .... ascorbic acid level in milk (mg. per 100 cc.).

x .... ascorbic acid level in plasma (mg. per 100 cc.).

The ascorbic acid level in milk can be predicted from this equation if the ascorbic acid value of plasma is known.

There is another factor which requires serious consideration. Until recently it was thought that only man, guinea-pigs and the primate suffered from scurvy and that other animals synthesized their ascorbic acid if necessary. There is now evidence that deer, cattle, swine, etc., may also be susceptible. The difference between the ability of different species to synthesize ascorbic acid may be one of degree only. It is possible that man can synthesize ascorbic acid, although not very effectively. Some observers believe that the human fœtus is able to synthesize ascorbic acid, because the pregnant woman rarely suffers from scurvy, even on a diet very poor in ascorbic acid. Ingalls, Draper and Teel (1938) suggested that as the nursing mother may secrete more ascorbic acid than she consumes in her food, it is quite probable that she may synthesize some quantity of ascorbic acid. The subjects in this group rarely consumed more than 40 to 50 mg. of ascorbic acid (excluding the loss in cooking procedures), but on the average they secreted in the later months of lactation about 40 to 50 mg. of the vitamin in their milk in addition to some in the urine. This fact, of course, is in favour of the above view that this vitamin can be synthesized in certain physiological states. It can be concluded also that in case of synthesis, the mammary gland takes no part in the process since the plasma and milk values are so consistent, showing that it is mainly a process of concentration that occurs in these glands. If the synthesis occurs at all, it is done in other tissues from which the vitamin enters the blood and then on reaching the mammary glands, the vitamin is concentrated and secreted in the milk.

Summary.—(*a*) The average concentration of ascorbic acid in milk of healthy Bengali woman is 6.7 mg. per 100 cc. of milk.

(*b*) The correlation between the ascorbic acid value of milk and plasma is significant and an equation has been given to show the relation between these two variants.

(*c*) The possibility of the synthesis of this vitamin has been discussed in lactation.

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REFERENCES

BAUMANN, T., and RAPPOLT, L. (1937). *Zeitschr. Vitaminforsch.*, **6**, 1.

FARMER, C. J., and ABT, A. F. (1936). *Proc. Soc. Exper. Biol. and Med.*, **34**, 146.

GEDDA, E., and KJELLBERG, K. (1939). *Acta Pediat.*, **26**, 177.

HAMIL, B. M., REYNOLDS, L., POOLE, M. W., and MACY, I. G. (1938). *Amer. J. Dis. Child*, **56**, 561.

HARRIS, L. J., and RAY, S. N. (1935). *Lancet, i*, 71.

INGALLS, T. H., DRAPER, R., and TEEL, H. M. (1938). *Amer. J. Dis. Child*, **56**, 1011.

RICHMOND, M. S., SATTERFIELD, G. H., GRINNELLS, C. D., and DANN, W. J. (1940). *J. Nutrition*, **20**, 99.

SELLEG, I., and KING, C. G. (1936). *Ibid.*, **11**, 599.

BIBLIOGRAPHY

NEUWEILER, W. (1937). *Zeitschr. Geburtsch. Gynak.*, **116**, 1.

THE EFFECT OF COBRA VENOM ON FROG INTESTINE

By T. H. RINDANI

(*From the Department of Physiology, Topiwala National Medical College, Bombay* 8)

THE paralysing effect of cobra venom in dilute solutions on frog's gastrocnemius has been reported recently by Sarkar and Maitre (1950). The inhibitory influence of dilute solutions of adrenaline on the pendular movements of intestinal segments has also been observed by Hoskins (1912). The action of drugs like acetylcholine and pilocarpine on isolated segments of small intestine has been studied by Bunting and his associates (1935),