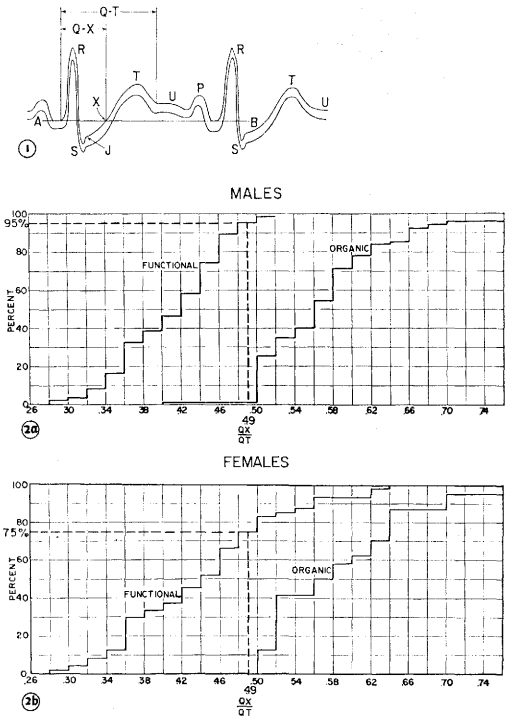

FIG. 1. Modified from Lepeschkin, E. and Surawicz, B., *New England J. Med.*, 258, 1958. RS-T segment is depressed. J is junction between end of QRS complex and beginning of RS-T segment. X is point of return of the depressed RS-T segment to isoelectricity. QX is time measured from beginning of ventricular activation to point X. QT interval (electrical systole)is measured from earliest deflection of QRS complex to end of T-wave. AB, the baseline or line of isoelectricity, is drawn through 2 successive QRS complexes.

FIG. 2a. Bar type cumulative frequency distribution curve in males indicates that 96% of individuals without heart disease have QX/QT ratios below 50%. With exception of one patient, all organic cases have values of 50% or more.

FIG. 2b. Cumulative frequency distribution bar graph of QX/QT ratio in women. All patients with organic heart disease have QX/QT ratios exceeding 50%. Among functional cases, 75% do not attain a 50% ratio.

There was no apparent relationship between QX/QT ratio and extent of RS-T depression in either organic or functional cases.

Conclusions. 1) There is a statistically valid difference in the nature of depressed RS-T segment after the "2-Step" test, among functional cases *vs.* those with ischemic heart disease. In presence of coronary heart disease, the depression persists for greater portion of ventricular complex than in functional cases. The QX/QT ratio is a simple and convenient expression of this characteristic of the RS-T segment and corrects for changes in heart rate. 2) A QX/QT ratio of 50% appears the optimal point at which to separate organic from functional cases. In 100 patients with known coronary heart disease, 99% demonstrate QX/QT ratios of 50% or more. Using this criterion, total incidence of false positive responses is 12% but with only 4.7% false reactors occurring among men. The overall accuracy of the criterion in both organic and functional cases with RS-T depression is 92.8% in our series. 3) It is suggested that criteria of an abnormal "2-Step" test should henceforth include a QX/QT ratio of 50% or more.

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Received November 5, 1959. P.S.E.B.M., 1960, v103.

Pantothenic Acid, Thiamine and Folic Acid Levels at Parturition.* (25504)

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Evidence showing fetal malformations and death from deficiencies of pantothenic acid (1), Vit. B_{12}(2), folic acid (PGA)(3), riboflavin(4), Vits. A, E, and K(5,6,7) have been

* This study was supported by U. S. Public Health Service Grants.

TABLE I. Pantothenic Acid, PGA and Thiamine Content of Maternal (M) and Infant (I) Serum at Parturition. All values given in mμg/ml.

Quartile	Pantothenic acid		PGA		Thiamine	
	M	I	M	I	M	I
	×100					
1st	.5- 1	1- 5	1- 4	3.5- 10	3 -10	19- 55
2nd	1 - 2.5	5-15	4- 7	10 - 34	10 -11.5	
3rd	2.5-10	15-19	7- 14	34 -100	11.5-15.5	55-100
4th	10 -22	19-68	16-100	100 -250	15.5-30	
Median	2.5	15	7	34	11.5	55
No. of patients	51	64	42	26	49	10

described. Before one can attempt to correlate the effects of vitamins on fetal survival and development, normal ranges must be established for serum levels of the various vitamins throughout pregnancy and parturition. Vit. B_{12}, PGA, and riboflavin and various amino acid concentrations of maternal serum were found to be lower than infant's at parturition(8,9,10,11,12). This is attributed to the avidity of the fetus for these vitamins. In the present report, a comparison of pantothenic acid, thiamine and PGA serum levels of the normal mother and infant at parturition is made so as to establish standard values.

Methods. Assays for pantothenic acid, thiamine and PGA on maternal and infant serum were carried out by methods previously described(13,14,15). Maternal blood was collected from an antecubital vein and infant blood was taken from the cord at time of delivery. Specimens were obtained from patients followed by the Obstetrical Service of Mount Sinai Hospital. The patients were in good nutritional state.

Results are given in Table I. The median value of 250 mμg/ml of pantothenic acid in maternal serum is substantially lower than 1500 mμg/ml in infant serum. The same relationship holds for thiamine and PGA levels; 11.5 mμg/ml for thiamine in maternal serum versus 55 mμg/ml for infants; 7 mμg/ml compared to 34 mμg/ml of PGA for mother and infant serum respectively. Median vitamin levels in infant serum are approximately 5 × greater than the maternal. Only 19% of the infant values for pantothenic acid fell below the maternal median. Only 23% of the infant PGA levels fell below the maternal median.

In reference to the range of these vitamins in nonpregnant subjects(13,14,15), the pantothenic acid levels of mothers fall within the normal range (100-1,000 mμg/ml); that of infants is elevated. The mother's PGA level is depleted, compared to the normal range of 8-24 mμg/ml, while the infants' are higher than normal. Finally, thiamine is lowered in maternal circulation; the infant appears to be within normal range (25-80 mμg/ml).

The PGA results confirm previous findings (9). Maternal PGA and B_{12} levels are lowered during pregnancy(8,9,11), suggesting that circulating maternal B_{12} and PGA levels are depleted by the fetus for use in cellular synthesis, a process catalyzed by these vitamines(9,16).

The maternal values for thiamine at parturition are lower than those reported for nonpregnant normals(14). In cases of pregnancy complicated by beriberi, thiamine deficiency was implicated as a factor responsible for toxemias(17). The maternal thiamine levels reported here (Table I), suggest that thiamine reserve is limited and that further reduction of maternal thiamine, as in nutritional deficiency, could be one of the factors leading to toxemias of pregnancy.

Summary. A comparison of pantothenic acid, thiamine, and PGA serum levels of mothers and infants at parturition shows an approximately 5-fold increase of these vitamins in the fetal circulation. The significance of these findings is discussed.

We are indebted to Dr. Alan F. Guttmacher, Dept. of Obstetrics and Gynecology, and his nursing staff for their cooperation.

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Received November 9, 1959. P.S.E.B.M., 1960, v103.

Plasma Protein VI. Catabolism *in vitro*.* (25505)

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It is apparent from numerous studies on turnover that serum albumin is being synthesized and degraded at a rapid rate in the mammal. The data of Cohen and coworkers (1) indicate that degradation occurs in the rabbit at 48 mg/hour. The present work was designed to determine the site of breakdown in the rat by carrying out *in vitro* studies with tissue slices and homogenates incubated with albumin which had been randomly labeled with C^{14}. Studies of this nature have already been carried out by Roberts and Kelley(2), who labeled rat albumin by administering phenylalanine-3-C^{14} to donor rats and then measured catabolism of labeled albumin in tissue slices from livers of the same species. They reported that albumin was rapidly broken down, a result that we have been unable to repeat. Katz and coworkers (unpublished) have likewise been unable to confirm results of Roberts and Kelley, using iodinated and S^{35}-labeled serum albumin. Other workers investigated the breakdown of albumin in liver either *in vivo* or by using perfusion technic (with or without reticuloendothelial blockade with india ink). Results of such experiments are not in good accord. Miller and coworkers (3) reported considerable breakdown of plasma protein by the perfused rat liver, whereas Gordon(4) found that perfused rat liver broke down albumin at a rate of only 10 to 15% of that calculated for the intact animal from turnover data. However, it has been reported that partial hepatectomy in rats (5) and mice(6) results in decrease in turnover rate of albumin. Following reticuloendothelial blockade various workers have reported conflicting results(6,7,8).

Methods. Slices or homogenates of rat tissues were incubated at 37° in buffer with plasma protein or albumin randomly labeled with C^{14}, made by feeding donor animals with labeled *Rhodospirillum rubrum*(9,10). Serum albumin was separated by salt fractionation. The method used was based on observations of Butler and Montgomery(11), and involved precipitation of globulins with 4 vol. 2.4 *M* phosphate buffer at pH 6.5. The solution was allowed to stand at room temperature for 24

* Aided by grant from Am. Cancer Soc.

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