

tory symptoms during 10 to 12 days of observation. Deaths occurred in both the infected and control litters. The majority of deaths occurred during the first experiment when the animals were housed in crowded quarters and the bedding was allowed to become wet. In one instance (litter No. 10) the mother deserted the sucklings. Avoidance of crowding in Exp. 2 and 3 limited non-specific deaths to the runts of the litters. None of the animals exhibited swollen nostrils, coryza, wheezing, or ruffled fur.

Discussion. The inocula used in these experiments had been stored at -70°C for varying periods of time, introducing a consideration of the stability of infectivity of the common cold virus at this temperature. Andrewes(6) found that filtrates of nasal secretions suffered no loss of infectivity when so stored for 2 years. Feller *et al.*(7) induced colds in volunteers with material that had been stored for 16 months. With 5 different secretions, Jackson(5) noted no loss of infectivity during periods of storage of from 40 to 49 weeks. While parallel transmission experiments were not done simultaneously in

both humans and hamsters, these considerations plus the evidence regarding the infectiousness for humans of N.S. #142 both before and after experiment 2, justify the conclusion that the negative results reported here are valid.

Summary. Nasal secretions from 5 different individuals with coryzal illnesses were inoculated intranasally in 138 suckling hamsters. No evidence was obtained to indicate that human colds can be transmitted to suckling hamsters.

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Blood Levels of 17-Hydroxycorticosteroids (17-OHCS) During Labor of Human Pregnancy.* (23318)

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It is now generally accepted that the activity of the adrenal cortex is increased in pregnancy(1-8). Blood levels of 17-OHCS have been found to increase progressively throughout the course of gestation reaching its maximum at the end of gestation and returning to low levels one week after delivery (2,3). These levels were higher in the primipara than in the multipara and equally higher in patients having vaginal deliveries than in

patients undergoing Cesarean section(3,9,10). These differences were attributed to the stress of labor which is more severe in primipara and during vaginal deliveries. This assumption received support from observations of high plasma levels of these hormones at the time of umbilical cord ligation(11).

The present study was aimed at assessing the effects of the various stages of labor and delivery on blood levels of 17-OHCS. This was done by following the same group of patients throughout labor until approximately one week after delivery.

Material and methods. Thirty-four pregnant subjects whose antenatal courses had

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been uneventful were selected at random. Ten patients were in the last trimester but not in labor and served as controls. Twenty-four patients were studied throughout their labors, deliveries and postpartum periods. Three periods during parturition were chosen for study: the first period encompassed the first stage of labor, the second period referred to the second stage of labor, and the third period represented the actual time of delivery. In addition, blood samples were taken at 30 minutes following delivery and on the 4th or 7th day postpartum. Although nearly all patients had samples taken when admitted early in labor, no more than 2 to 4 further samples were taken from any one patient. The methods for collecting blood samples and determining the concentration of 17-OHCS in the plasma have been described previously (8). Most patients were given 1 to 3 injections of intramuscular Demerol (100 mg) as required for analgesia, while anesthesia for delivery was usually local or pudendal block, with nitrous oxide and oxygen inhalation. Spinal anesthesia was given in a few cases. Two patients, Nos. 16 and 19 were delivered by Cesarean section.

TABLE I. Plasma Levels 17-Hydrocorticosteroid in Various Stages of Labor and Postpartum in 23 Patients.

1st stage	2nd stage	Delivery	$\frac{1}{2}$ hr post-partum	4th day post-partum
30.0	65.6	80.2	62.8	45.3
88.3		173.3	69.4	
	36.8			37.7
31.0		55.6		
67.5	115.0			64.2
67.0		83.4		25.8
72.6	75.9			61.1
29.5	58.7		46.6	37.9
33.1				
30.5	95.6			37.2
71.2	58.9			
33.9		82.9	43.5	22.0
79.8		77.2		23.7
59.6	111.4			22.0
19.2	78.6			
* 78.6		34.7		
43.3	98.3		105.0	
* 44.8		47.6		27.6
84.4		127.1		
65.8	79.0			40.6
	70.4			
53.9		69.3		19.2

* Cesarean section.

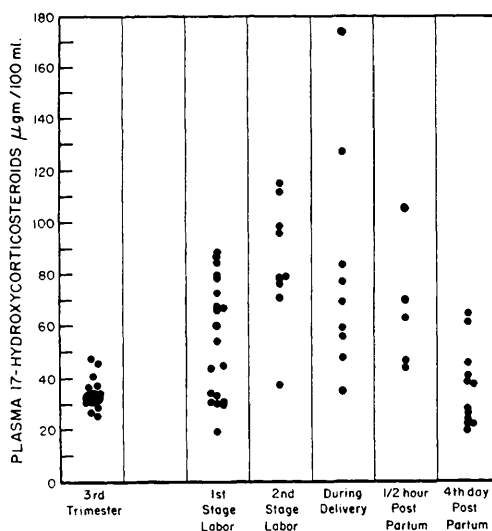


FIG. 1. Distribution of plasma volumes for 17-OHCS during last trimester of pregnancy (not in labor) and in various stages of labor and after delivery.

Results. Detailed data on blood levels of 17-OHCS during various stages of labor and after delivery are listed in Table I. Fig. 1 compares these values to those of a control group in the last trimester but not in labor. The values for plasma 17-OHCS in the control group not in labor ranged from 25.6-47.5 $\mu\text{g}\%$ with a mean value of 34.6 $\mu\text{g}\%$. These levels compare well with previous studies (2, 3, 5-8) and are well above nonpregnant values of 7 to 15 $\mu\text{g}\%$.

In the first stage of labor, plasma levels ranged from 19.2 to 88.3 $\mu\text{g}\%$ with a mean of 50.6. In the 2nd stage of labor the values ranged from 36.8 to 115 with a mean of 80.3 $\mu\text{g}\%$. The wide variation observed was probably due to differences in the reaction of patients to labor, duration of labor and some other subjective variables which could not be easily controlled. At the time of delivery, the values ranged from 34.7 to 173 $\mu\text{g}\%$. The highest value was observed in a patient with possible liver disease. Immediately after delivery the plasma levels of 17-OHCS were still high, ranging from 43 to 105 $\mu\text{g}\%$. Four days postpartum the levels had fallen somewhat but were still higher than those of nonpregnant patients (7-15 $\mu\text{g}\%$).

Statistical analyses of the data showed that the difference between the values obtained

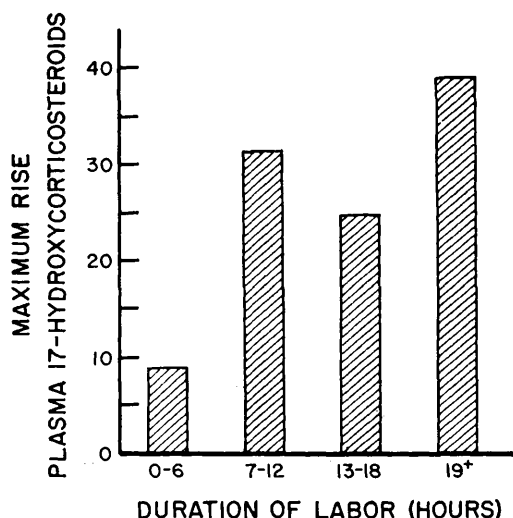


FIG. 2. Plasma levels of 17-OHCS in relation to length of labor. Labors lasting less than 6 hr had significantly lower values than labors lasting longer.

during labor and those obtained from the control series not in labor was significant ($P < .01$); the difference between first and second stage of labor was also significant ($P < .05$); and so was the difference between the values obtained during labor and those of the postpartum period ($P < .01$). Plasma levels of 17-OHCS were lower in patients whose labor lasted less than 6 hours (Fig. 2). Beyond 12 hours of labor there was little further increase in values.

Discussion. The data indicate a variable but consistent rise in plasma levels of 17-OHCS during parturition. Maximum elevation occurred at the stage of expulsion of the fetus.

Although mean values at the fourth or seventh postpartum day were below the initial values for patients in labor, they were in most cases well above normal. This could be caused by either a faulty metabolism of these hormones during the postpartum period or by the fact that the stimulus responsible for elevation of plasma levels of 17-OHCS during parturition had not subsided by the end of the first week after delivery.

The source of the excess of 17-OHCS observed during labor is difficult to determine. It has been shown previously(12) that the elevated levels of pregnancy were due to ex-

cessive production rather than to faulty metabolism of these hormones. It was further shown that despite the already elevated levels in normal pregnancy, the adrenal cortex was still capable of responding to additional stimulation with exogenous ACTH.

In view of the rise in 17-OHCS observed in the last stage of labor approximating those found after maximum ACTH stimulation in the last trimester of pregnancy(12) and in patients with Cushing's disease under similar stimulation(13), it is possible that labor imposes a maximum stimulus upon the adrenal cortex of the mother. This, however, does not rule out the possibility that the placenta might contribute in some way or another to these high levels.

The mechanisms by which labor could stimulate the adrenal cortex are obscure. Assuming that the maternal pituitary gland is the major site of adrenocorticotrophic hormones in pregnancy, it is probable that labor stimulates this gland to increase its release of ACTH. Several workers(14,15,16) have observed that certain posterior pituitary hormones may stimulate ACTH secretion. Martine *et al.*(17) have claimed that both vasopressin and oxytocin may act in this way, while others(18) have failed to confirm any significant action of either pitocin or syntocin in animals. If oxytocin stimulates ACTH release in human subjects, then it is reasonable to accept the idea that labor stimulates the pituitary gland of the mother through the action of oxytocin. This does not rule out the contribution of the stimulus provoked by the pain itself and by the muscular work involved in labor since these factors *per se* have been shown to lead to increased plasma levels of 17-OHCS(19,20). The contribution of each of these variables is now being studied.

Summary. 1. Blood levels of 17-OHCS were studied at intervals during parturition in normal pregnancy. 2. Early in labor, a rise in levels above those found in late pregnancy was noted, with further rise between first and second stages. These high levels were maintained until after the end of 3rd stage. In most cases studied, levels were still well above normal on 4th or 6th postpartum day. 3. There seems to be a relation between

length of labor and rise in plasma 17-OHCS. Labors of longer than 6 hours deviation were accompanied by high levels of these hormones. 4. Maximum values observed in labor approached those noted after exogenous ACTH stimulation during late pregnancy, indicating nearly maximal output of these hormones by the adrenal cortex. 5. The mechanisms by which labor stimulated the maternal pituitary gland have been discussed.

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Beneficial Effects of Alfalfa and Other Succulent Plants on Glucoascorbic Acid Toxicity in the Rat.* (23319)

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Woolley and Krampitz(1) have observed that immature mice fed a purified ration containing 5 to 10% glucoascorbic acid developed a severe condition characterized by failure of growth, diarrhea, subcutaneous hemorrhages, irritation and swelling of the anal region, alopecia and death. These effects were completely counteracted by concurrent feeding of dried grass(1) or alfalfa meal(2). Supplements of the known nutrients, in contrast to results obtained above, were without significant effect(2). In the present communication data are presented on comparative

effects of alfalfa and other materials of plant and animal origin on glucoascorbic acid toxicity in the rat.

Procedure. The basal ration consisted of sucrose, 62%; casein,[†] 24%; salt mixture,[‡] 5%; corn oil, 5%; and glucoascorbic acid,[§]

[†] Vitamin-free Test Casein, General Biochemicals, Chagrin Falls, O.

[‡] Hubbel, Mendel and Wakeman Salt Mixture, General Biochemicals, Chagrin Falls, O.

[§] The glucoascorbic acid was prepared by procedure described in U. S. Patent No. 2,206,374. The author is indebted to Dr. Robert Irwin for synthesizing this material and to Mr. Philip P. Gray of Wallerstein Laboratories, N. Y. City, for permission to employ patent procedure and his helpful suggestions in preparation of this product.

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