Table I. Length of the diestrus that followed reserpine treatment (mean ± S.E.)

Temperature	Jan.–Feb.	Aug.–Sept.	
Into the animal house (t. ⩾ 24 °C)	10 ± 1.05	4 ± 0.18	$p < 0.001$
Out of the animal house (environment t.)	9 ± 1.87	5 ± 1.10	$p < 0.02$

Data from 1970.

Table II. Difference in weight between traumatized and control uterine horn

	Weight difference (mg)		
	$\bar{X}$	S.E.	n
February–March	636.5	152	9
July	353.7	80.9	14
August	209.4	61	9

Data from 1971.

at the level of the catecholamine-PIF interaction. It would be of interest to explore if the seasonal variation is associated with different degrees of catecholamine depletion to localize the seasonally sensitive step to one of the two groups of events mentioned above.

Resumen. Se indujo pseudogestación por reserpina en ratas, cada mes del año, durante dos años. La longitud del diestro que siguió al tratamiento se tomó como estimación de la duración de la pseudogestación. Los resultados mostraron una clara variación estacional con una longitud mínima del diestro que siguió al tratamiento con reserpina durante los meses de Agosto y Setiembre (Hemisferio Sur). Una serie de experimentos en los cuales la pseudogestación se midio por medio de la formación de deciduomas confirmó los hallazgos en un tercer año.

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Plasma Renin Concentration at Delivery and During the Newborn Period in Humans

Numerous studies have been done on plasma renin activity (PRA) and plasma renin concentration (PRC) in adults in various pathophysiological conditions[1,2], whereas only a few investigations on renin in infants and mothers have been performed at delivery[3–7]; however, at the present time there is no information available on PRA or PRC in the newborn period. The reason for this is that large amounts of plasma were necessary for the determinations. The development of micromethods has made these studies possible. Recent investigations in newborn dogs suggested a stimulation of the renin-angiotensin system during the first weeks of extrauterine life[8,9].

Methods. PRC was measured with a micromethod[10] based on BOUCHER's[11] procedure; 0.1 to 1.0 ml plasma and 1.0 ml Dowex 50WX2-$(NH_4)^+$ were incubated at pH 7.0 with 150 mg sheep angiotensinogen, providing 12–15 ng angiotensin/mg protein, dissolved in 2.0 ml trisphosphate buffer, containing EDTA and NaN_3, for 15 h at 37 °C. PRC is expressed in ng angiotensin/ml plasma/h incubation, mean ± SE.

Twelve healthy women (no signs of toxemia, no sodium restriction, no administration of diuretic agents during pregnancy) were studied during uncomplicated vaginal delivery prior to administration of any oxytocic agent; in 4 of them PRC was measured 6 days post partum for the second time during resting conditions.

In 12 corresponding newborns PRC was determined in cord plasma taken before placental delivery and in peripheral venous plasma in the first 48 h post partum (3–48 h). The infants did not get any food for 24 h after delivery and then were kept on tea or tea and milk up to 48 h. In 8 of them PRC was measured 3–10 days after delivery also. In some newborns more than one measurement of PRC was done in the respective period; for calculation we used the maximum values in the 3–48 h period and the last determination in the 3–10 days period.

Statistical evaluation was done using Student's t-test and Student's paired t-test.

Results and discussion. The results are summarized in the Figure. a) The PRC in cord plasma (14.7 ± 3.1) was higher than in maternal plasma (7.7 ± 1.0). This result is in agreement with the studies of BROWN et al.[3] and WERNZE and SEKI[6] who described higher PRC resp. PRA in cord plasma than in maternal plasma; in contrast other investigators[4,5,7] did not find significant differences between fetal and maternal PRC and/or PRA. With regard to the higher PRC in cord plasma than in maternal venous plasma it seems likely that the renin in cord plasma derives from the fetus; furthermore, the increase of PRC after delivery b) shows that the infant can

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produce renin and suggests a fetal source of renin in cord plasma; however, we cannot rule out that part of the renin in cord plasma; derives from uterine structures, especially from the placenta which contains a high amount of renin[12]. In this context it is of interest that PRC in amniotic fluid exceeds PRC in fetal and maternal plasma by far[3].

b) 3 to 48 h after delivery PRC in peripheral venous plasma of the infants was higher (27.0 ± 2.5) than in cord plasma. In 3 infants 2 determinations of PRC were done in the first 48 h after delivery; PRC between 5 to 7 h post partum was higher than between 23 to 48 h (mean decrease 17%). At the present time we have no explanation for the high PRC at delivery. Water loss[13] and reduction of extracellular volume[14] may account for the high PRC in the postnatal period; the 24 h fasting period to which the infants were submitted might intensify these effects and thus mediate the shortlasting further increase of PRC in the first 48 h after delivery; however, in our study the maximum of PRC did not coincide with the maximal weight loss of the newborns. 3 to 10 days post partum PRC was significantly lower (8.4 ± 2.8) than during the 3–48 h period. In infants between 3–24 months[15] PRC had further decreased (3.42 ± 0.50; $n = 46$; $p < 0.0005$ vs. 3–10 days period). It is of interest that in a recent report the aldosterone plasma level has been found extremely high in cord plasma and in peripheral venous plasma of infants in the first 3 days after delivery[16]. The high plasma aldosterone might be secondary to the increased PRC.

c) In mothers, PRC during labor was significantly higher (7.7 ± 1.0; $p < 0.0005$) than in normal subjects (recumbent 0.98 ± 0.12; upright 2.12 ± 0.22; $n = 22$; age 20–35 years; no significant differences between males and females). In 4 women studied 6 days after delivery PRC had decreased to 3.2 ± 1.5 ($p < 0.025$ vs. PRC during labor). Our findings are consistent with previous studies reporting elevated PRC[3,17] and PRA[4,6] during labor and immediately post partum[5]; PRC had returned to the normal range 2–4 days after delivery[17]; 7 days post partum PRA had normalized in one study[5] and was found still elevated in another report, returning to normal within 6 weeks[4]. It seems likely that, besides methodological reasons (PRC-PRA), small differences in sampling and postpartal conditions (diet, nursing) cause the differential results[18].

Zusammenfassung. Die Plasma-Renin-Konzentration (PRC) Neugeborener ist höher als die ihrer Mütter, verdoppelt sich innerhalb 48 h und sinkt bis zum 10. Tag unter den Geburtswert. Die PRC der Mütter ist bei der Entbindung sowie am 6. Tag post partum trotz Absinkens auf 50% des Ausgangswertes höher als bei nicht-graviden Kontrollen.

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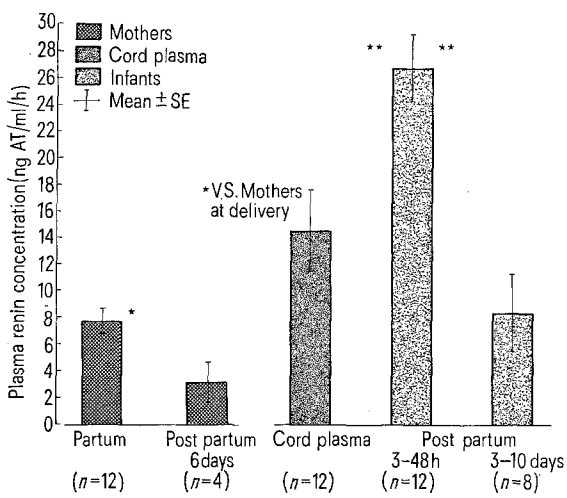

Plasma renin concentration (PRC) at delivery and during the newborn period in humans. $*p < 0.025$; $**p < 0.0005$.

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Oxidative Metabolism of the Limbic System in Prepuberal Rats

The limbic system presents variations in the consumption of oxygen in female rats in relation to the sexual cycle. SCHIAFFINI et al.[1] have shown that it is high during the estrous phase and that it decreases during the diestrous, whereas the contrary happens in the hippocampus. In males, the consumption of the amygdala is inferior to that of the hippocampus, and studies realized in vitro suggest a possible relationship between the oxidative metabolism of the limbic system and the pituitary gonadotrophins[2].

The oxidative metabolism of the limbic system in females is affected by the ovariectomy[3], experimental diabetes[4] and postnatal treatment with testosterone[5]. In this paper we have studied the oxidative metabolism in prepuberal rats of both sexes as well as their alterations after modifying those mechanisms that control the secretion of gonadotrophins by administering testosterone to the females or castrating the males in the first days of life[6].

Material and methods. We have studied the oxidative metabolism of the amygdala, hippocampus, and the hypothalamus of 21-day-old Wistar rats, pertaining to the following experimental groups: a) control females; b) control males; c) females, 5 days old, treated with 100 µg of testosterone propionate, in oil solution; d) males castrated 2 or 7 days after birth.