

TREATMENT OF THYROID HORMON IN PREMATURES

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INTRODUCTION:

Function of thyroid hormon

- Increase the basal metabolic rate,
- Affect protein synthesis,
- synergy with growth hormone, and neural maturation
- Increase the body's sensitivity to catecholamines (such as adrenaline) by permissiveness.
- Regulate protein, fat, and carbohydrate metabolism.
- Inhibit neuronal activity
- Severe drop in body temperature.

Fetal thyroid function: 8-10 weeks

Second trimester and then T₄ steadily increase.

24 - 34 weeks, serum T₄ levels plateau.

STUDIES:

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High Incidence of Thyroid Dysfunction in Preterm Infants
J Korean Med Sci. 2008 Aug; 24(4): 627-631.



High Incidence of Thyroid Dysfunction in Preterm Infants

Hye Rim Chung, Choong Ho Shin, [...], and Jung Hwan Choi

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Abstract

To determine the validity of a repeat thyroid function test for preterm infants, and to investigate factors that influence thyroid function of preterm infants, thyroid functions of 105 infants born at <32 weeks'

gestational age were evaluated. Initial serum free thyroxine (fT4) and thyrotropin (TSH) levels were measured during the first 10 days of life, and repeated tests were performed more than 2 weeks apart. We analyzed the effects of gestational age, systemic diseases, and nutrition on the development of thyroid dysfunction. Thirty-one infants (30%) had low fT4 levels (<0.7 ng/dL) in the absence of elevated TSH levels (<7 μ U/mL). Thirteen infants (12%) had hypothyroidism (fT4 <0.7 ng/dL, TSH \geq 10 μ U/mL) and mean age at diagnosis was 28 \pm 17 days. Twelve infants had moderately elevated TSH (TSH 10-30 μ U/mL) with normal fT4 levels after 1 week of postnatal life. The history of undergone surgical procedure which needed iodine containing disinfectants was significantly frequent in the infant with hypothyroidism and transient TSH elevation. Repeated thyroid function tests are necessary for preterm infants, even though they initially show

105 premature infants (28-34 weeks)

TSH (TSH 10-30 $\mu\text{U}/\text{mL}$) with normal fT4

levels after :

1 week of postnatal life(12%)

2 weeks of postnatal life(32%)

3 weeks of postnatal life(54%)

➔ Repeated thyroid function tests are necessary for preterm infants. Treatment with thyroid hormone in premature infants is unnecessary.

P&S Journal: Fall 1996, Vol.16, No.3

Research Reports

Premature Babies, Thyroid Hormone, and CP

Premature infants with low levels of thyroid hormones are significantly more likely to develop cerebral palsy (CP) than are infants with normal hormone.

Abstract

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Pediatr Res. 1998 Nov;44(5):642-9.

Plasma thyroid hormones in premature infants: effect of gestational age and antenatal thyrotropin-releasing hormone treatment. TRH Collaborative Trial Participants.

Ballard PL¹, Ballard RA, Ning Y, Cnann A, Boardman C, Pinto-Martin J, Polk D, Phibbs RH, Davis DJ, Mannino FL, Hart M.

Author information

Abstract

Thyroid hormones are important for both perinatal adaptation and long-term psychomotor development; however, there is limited information on the effects of extreme prematurity and antenatal TSH-releasing hormone (TRH) treatment on pituitary-thyroid function. In this study we assayed plasma triiodothyronine (T3) and TSH in infants who were part of a collaborative trial of antenatal maternal TRH therapy. Within the control population (n = 166), infants of 24-28-wk and 28-32-wk gestational age had comparable levels of T3 (0.94 and 1.06 nmol/L, respectively) and TSH (5.7 and 7.2 mU/L) at birth, but the increases at 2 h and subsequent T3 levels were less in the 24-28 wk versus 28-32-wk gestation infants. In the TRH-treated group (n = 131), T3 was lower in the first day for infants delivered 7-72 h after antenatal TRH compared with control infants. TSH at birth was approximately 3.5-fold greater for infants delivered at 0-6 h after the last TRH dose compared with the control group and was suppressed in infants delivering at 7-36 h. T3 and TSH levels were not different between control and TRH-treated groups at 3-28 d of age. In TRH stimulation tests on d 28, control and TRH-treated groups had similar peak levels of TSH and incidence of exaggerated response (TSH > or = 35 mU/L). We conclude that extremely premature infants have a reduced postnatal surge in TSH and T3 and maintain lower T3 concentrations, probably reflecting tertiary hypothyroidism. The stimulatory and suppressive effects of antenatal TRH treatment observed at birth are transient and do not affect pituitary-thyroid responsiveness at 28 d of age.

PMID: 9803444 [PubMed - indexed for MEDLINE]



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The stimulatory and suppressive effects of antenatal TRH treatment observed at birth are transient and do not affect pituitary-thyroid responsiveness at 28 d of age.

Thyroid hormones for preventing neurodevelopmental impairment in preterm infants

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Dates

Date edited: 23/08/2001
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Date next stage expected //
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Review first published: Issue 2, 1999

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Nine studies were identified that compared thyroid hormone treatment and nontreatment.

This review does not support the use of thyroid hormones in preterm infants to reduce neonatal mortality, improve neurodevelopmental



Postnatal thyroid hormones for preterm infants with transient hypothyroxinaemia

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Osborn DA, Hunt RW

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There is insufficient evidence to determine whether use of thyroid hormones for treatment of preterm infants with transient hypothyroxinaemia results in changes in neonatal morbidity and mortality, or reductions in neurodevelopmental impairments. Further research is required.

Results of controlled double- x

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Pediatrics. 1984 Mar;73(3):301-5.

Results of controlled double-blind study of thyroid replacement in very low-birth-weight premature infants with hypothyroxinemia.

Chowdhry P, Scanlon JW, Auerbach R, Abbassi V.

Abstract

The nature of hypothyroxinemia in sick very low-birth-weight (VLBW) infants was evaluated by assessment of the hypothalamic-pituitary axis and by the clinical response to thyroxine (T4) therapy. Twenty-three very low-birth-weight infants of gestational age 26 to 28 weeks, whose serum T4 concentrations were 4 micrograms/dL on two occasions, and thyrotropin less than 20 microU/mL, were included in a double-blind study. Following a thyrotropin-releasing hormone stimulation test, babies were given either T4 or placebo. Nine babies were thyrotropin-releasing hormone tested prior to therapy; four babies, two from each group, were tested 1 to 2 weeks after therapy. In 11 untreated babies, mean base line serum thyrotropin of 7.0 +/- 1.4 rose to 23.7 +/- 4.1 microU/mL in 30 minutes. This response was not significantly greater than the observed response in full-term babies, 23.7 +/- 4.1 v 16.6 +/- 0.97 microU/mL, respectively, P greater than .05. In two babies treated with T4 the thyrotropin response to thyrotropin-releasing hormone was completely suppressed. Serial serum T4 determinations showed normalization in both groups after a similar time interval. There was no beneficial effect of T4 therapy on growth of head circumference, length, or weight. Developmental data revealed no significant differences in the mental, motor, or gross neurologic outcome in the treated and nontreated infants after 1 year of follow-up. These observations imply that hypothyroxinemia in sick preterm infants is not a direct consequence of hypothyroidism. Despite the lack of demonstrable short-term beneficial effects of T4 therapy, follow-up studies are necessary to resolve the question of long-term benefits.

PMID: 6366725 [PubMed - indexed for MEDLINE]

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Developmental data revealed no significant differences in the mental, motor, or gross neurologic outcome in the treated and nontreated infants after 1 year of follow-up.

Abstract Send to:

N Engl J Med. 1996 Mar 28;334(13):821-7.

The relation of transient hypothyroxinemia in preterm infants to neurologic development at two years of age.

Reuss ML¹, Paneth N, Pinto-Martin JA, Lorenz JM, Susser M.

Author information

Abstract

BACKGROUND: Transient hypothyroxinemia, a common finding in premature infants, is not thought to have long-term sequelae or to require treatment. We investigated whether hypothyroxinemia in premature infants is a cause of subsequent motor and cognitive abnormalities.

METHODS: In this historical cohort study, we retrieved blood thyroxine values, obtained on routine screening in the first week of life, from state screening records on children who weighted 2000 g or less at birth, who were born at 33 weeks' gestation or earlier, and who were enrolled in a population-based study of the late sequelae of neonatal brain hemorrhage. We investigated the relation of these values to the odds for disabling cerebral palsy among 463 subjects for whom data were available and to the mental-development score on the Bayley Scales of Infant Development or the Stanford-Binet Intelligence Scales for Children at the age of two years in 400 subjects. The effects of severe hypothyroxinemia, defined as a blood thyroxine value more than 2.6 SD below the mean for New Jersey newborns, were assessed before and after adjustment for gestational age and potentially confounding variables.

RESULTS: In analyses adjusted for gestational age, infants with severe hypothyroxinemia had a risk of disabling cerebral palsy that was nearly 11 times that of infants without hypothyroxinemia (odds ratio, 10.8; 95 percent confidence interval, 3.0 to 39.3) and a mean mental-development score at the age of two that was 15.4 points lower (95 percent confidence interval, 8.1 to 22.6 points) than the mean score of children with normal neonatal blood thyroxine concentrations. After adjustment for gestational age and multiple prenatal, perinatal, and early and last neonatal variables, severe hypothyroxinemia was still associated with an increased risk of disabling cerebral palsy (odds ratio, 4.4; 95 percent confidence interval, 1.0 to 18.6) and a reduction of nearly 7 points (95 percent confidence interval, 0.3 to 13.2 points) in the mental-development score.

CONCLUSIONS: Severe hypothyroxinemia in preterm infants may be an important cause of problems in neurologic and mental development detected at the age of two years.

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Severe hypothyroxinemia in preterm infants may be NOT an important cause of problems in neurologic and mental development detected at the age of two years.

CONCLUSIONS:

- Should follow up thyroid hormone in premature infants.
- Treatment thyroid hormone in premature infants is unnecessary