Motor nerve conduction velocity and somatosensory evoked potentials in the newborn and young child in relation to thyroid function

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Chapter 1

General introduction

Thyroid hormone is essential for normal brain development. Proliferation of astrocytes and myelination are known to be regulated by thyroid hormones. A deficiency during brain development, as in congenital hypothyroidism, can cause problems in motor and cognitive development. In addition, transient hypothyroxinemia, which is commonly found in preterm infants, is associated with an increased risk of neurodevelopmental dysfunction. Material thyroid disease can occur in the newborn period. Transient hypothyroxinemia of the preterm infant may be caused by immaturity of the hypothalamo-pituitary-thyroid axis, interruption of maternal-fetal transfer of thyroid hormones, sick euthyroid syndrome or a combination of these factors. It is still uncertain whether transient hypothyroxinemia in preterm infants causes a shortage of thyroid hormone at the cellular level in the nervous system, as is the case in congenital hypothyroidism, and thus requires L-thyroxine supplementation. Until now, six prospective studies in preterm infants have been performed to examine the effect of thyroid hormone supplementation on mortality, morbidity and neurodevelopmental outcome. None of these studies provides sufficient evidence to implement thyroid supplementation in preterm infants in clinical practice. In addition to these clinical studies, more insight could be obtained by investigating the effect of hypothyroxinemia or maternal thyroid disease on the developing nervous system itself. For this purpose, neurophysiologic methods such as measuring motor nerve conduction velocities and latencies of somatosensory evoked potentials, may be used. In patients with congenital hypothyroidism prior to treatment, slow nerve conduction velocity was reported. Also in patients with congenital hypothyroidism, prolonged latencies of somatosensory evoked potentials during the first year of life were found and interpreted as a sign of a transient shortage of thyroid hormone in the nervous system. For preterm infants an association between low serum thyroxine concentrations and a delay in neural maturation was reported. Measuring motor nerve conduction velocities and latencies of somatosensory evoked potentials may be utilized to study the effect of transient hypothyroxinemia and maternal thyroid disease on the maturation of the developing nervous system. These measures may also be employed when studying the effect of intervention strategies such as thyroid hormone supplementation in very preterm infants.
Chapter 1

General Introduction

Obtaining the necessary state of relaxation with no phasic movements of body, head and eyes in these children was a quite challenging task. A rather intense human relation involving explanations, jokes, story telling and reading comic books was maintained throughout the session by both the experimenter and an expert nurse (Madame Stage).

— John E. Desmond et al., 1976
General introduction

Thyroid hormone is essential for normal brain development. Proliferation of axons and dendrites, synapse formation, gliogenesis and myelination are known to be regulated by thyroid hormone.\(^1\) Thyroid hormone deficiency during brain development, as in congenital hypothyroidism, can cause problems in motor and cognitive development.\(^2\) In addition, transient hypothyroxinemia, which is commonly found in preterm infants, is associated with an increased risk of neurodevelopmental dysfunction.\(^3,5\) Maternal thyroid disease can also interfere with normal brain development.\(^6,10\) Transient hypothyroxinemia of the preterm infant may be caused by immaturity of the hypothalamo-pituitary-thyroid axis, interruption of maternal-fetal transfer of thyroid hormones, sick euthyroid syndrome or a combination of these factors.\(^11,14\) It is still uncertain whether transient hypothyroxinemia in preterm infants causes a shortage of thyroid hormone at the cellular level in the nervous system, as is the case in congenital hypothyroidism, and thus requires L-thyroxine supplementation.\(^15-21\) Until now, six prospective studies in preterm infants have been performed to examine the effect of thyroid hormone supplementation on mortality, morbidity and neurodevelopmental outcome.\(^22-27\) None of these studies provides sufficient evidence to implement thyroid supplementation in preterm infants in clinical practice. In addition to these clinical studies, more insight could be obtained by investigating the effect of hypothyroxinemia or maternal thyroid disease on the developing nervous system itself. For this purpose, neurophysiologic methods such as measuring motor nerve conduction velocities and latencies of somatosensory evoked potentials, may be used. In patients with congenital hypothyroidism prior to treatment, slow nerve conduction velocity was reported.\(^28,29\) Also in patients with congenital hypothyroidism, prolonged latencies of somatosensory evoked potentials during the first year of life were found and interpreted as a sign of a transient shortage of thyroid hormone in the nervous system.\(^30-33\) For preterm infants an association between low serum thyroxine concentrations and a delay in neural maturation was reported.\(^34\) Measuring motor nerve conduction velocities and latencies of somatosensory evoked potentials may be utilized to study the effect of transient hypothyroxinemia and maternal thyroid disease on the maturation of the developing nervous system. These measures may also be employed when studying the effect of intervention strategies such as thyroid hormone supplementation in very preterm infants.
Scope of the thesis
This thesis describes the use of motor nerve conduction velocities (MNCVs) and
latencies of median nerve somatosensory evoked potentials (SEPs) in the newborn
and young child in order to study the effect of thyroid function on the maturation
of the developing nervous system. Literature reviews on MNCV and on median
nerve SEPs are given in chapters 2 and 3, respectively. Until now, in very preterm
infants insufficient reference values are available. Reference values for MNCV
in ulnar and posterior tibial nerves in very preterm infants are described in
chapter 4. Reference values for latencies of median nerve SEPs are described in
chapter 5. In chapter 6 the neurological development of children born to mothers
with known thyroid disease during pregnancy, is described. In these infants
MNCVs and latencies of median nerve SEPs were measured as well. Chapter 7
describes an MNCV study of an L-thyroxine supplementation trial in a group of
200 infants born at less than 30 weeks’ gestation. In chapter 8, a study of
latencies of median nerve SEPs in the same L-thyroxine supplementation trial
is described. The main results of the various studies and their implications are
summarized in chapter 9.

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