

344 Thyroid Disorders

Definition/Cut-Off Value

Thyroid dysfunctions that occur in pregnant and postpartum women, during fetal development, and in childhood are caused by the abnormal secretion of thyroid hormones. The medical conditions include, but are not limited to, the following:

Thyroid Dysfunction	Definition
Hyperthyroidism	Excessive thyroid hormone production (most commonly known as Graves' disease and toxic multinodular goiter).
Hypothyroidism	Low secretion levels of thyroid hormone (can be overt or mild/subclinical). Most commonly seen as chronic autoimmune thyroiditis (Hashimoto's thyroiditis or autoimmune thyroid disease). It can also be caused by severe iodine deficiency.
Congenital Hyperthyroidism	Excessive thyroid hormone levels at birth, either transient (due to maternal Grave's disease) or persistent (due to genetic mutation).
Congenital Hypothyroidism	Infants born with an under active thyroid gland and presumed to have had hypothyroidism in-utero.
Postpartum Thyroiditis	Transient or permanent thyroid dysfunction occurring in the first year after delivery based on an autoimmune inflammation of the thyroid. Frequently, the resolution is spontaneous.

Presence of condition diagnosed, documented, or reported by a physician or someone working under physician's orders, or as self reported by applicant/participant/caregiver. See Clarification for more information about self-reporting a diagnosis.

Participant Category and Priority Level

Category	Priority
Pregnant Women	I
Breastfeeding Women	I
Non-Breastfeeding Women	III, IV, V or VI
Infants	I
Children	III

Justification

The thyroid gland manufactures three thyroid hormones: thyroxine (T_4), triiodothyronine (T_3), and calcitonin. The thyroid hormones regulate how the body gets energy from food (metabolism). Iodine is an essential component of the T_4 and T_3 hormones (1) and must come from the diet. (Note: In nature, iodine does not exist as a free element; rather, it forms compounds such as sodium iodide (2, 3). For more information see Clarification section.) Iodine is available from various foods, and is present naturally in soil and sea water. A dysfunctional thyroid gland can become enlarged (goiter) as a result of an overproduction of thyroid hormones (hyperthyroidism) or conversely, from insufficient thyroid hormone production (hypothyroidism). Thyroid hormones influence virtually every organ system in the body.

Maternal needs for dietary iodine and thyroid hormone medication (if prescribed) increase during pregnancy as maternal thyroid hormones and iodine are transferred to the fetus along with an increased loss of iodine through the maternal kidneys (3). Concurrently, the fetus is unable to produce thyroid hormones during the first trimester and is entirely dependent on the maternal supply of thyroid hormones. As a result, maternal production of T_4 must increase by at least 50% during pregnancy (4). If the pregnant woman is receiving thyroid hormone therapy, often a 30% - 50% increase in thyroid hormone medication is also needed.

Hyperthyroidism

Hyperthyroidism is a condition in which the thyroid gland is overactive, manufacturing too much thyroid hormone (T_4 and T_3). An excessive consumption of iodine ($> 1000 \mu\text{g}/\text{d}$) may cause fetal and maternal hyperthyroidism (5). In other circumstances, the thyroid might develop nodules which secrete excessive amounts of thyroid hormone regardless of iodine status (5). Enlargement of the thyroid gland (goiter) is a common symptom, as well as weight loss, fatigue, muscle weakness and an irregular heartbeat.

Hyperthyroidism is relatively uncommon in pregnancy (4). However, when it occurs, uncontrolled hyperthyroidism (especially in the second half of pregnancy) may result in infection, miscarriage, preterm delivery, preeclampsia, or congestive heart failure. Fetal complications may include prematurity, small for gestational age, fetal or neonatal thyrotoxicosis, or death (6). Postpartum maternal hyperthyroidism is likely in women with prenatal hyperthyroidism (7).

The primary medical therapy for hyperthyroidism is radioactive iodine therapy which is contraindicated during pregnancy and lactation (7). If hyperthyroidism occurs during this period, low doses of thiomide (antithyroid drug) are given instead.

Hypothyroidism

Hypothyroidism is a condition in which the thyroid gland does not make enough thyroid hormone. Maternal and fetal hypothyroidism may occur when preconception maternal iodine stores are insufficient and there is inadequate maternal iodine intake in early pregnancy. In this instance, the maternal iodine balance may become negative and may never be restored, even with eventual iodine supplementation (4).

Mothers with iodine deficiency during the first half of pregnancy may produce offspring with severe, irreversible brain damage (8). Maternal thyroid deficiency has been associated with neonatal developmental problems which may cause lasting changes in the brain structure and cognitive function.

Uncontrolled hypothyroidism in the second half of pregnancy can cause maternal complications such as anemia, preeclampsia, miscarriage, premature delivery, and postpartum thyroid disease. Fetal or neonatal

complications include prematurity, low birth weight, congenital anomalies, poor neuropsychological development, and stillbirth (6).

When iodine nutrition status is adequate, autoimmune thyroid disease (AITD) – also called Hashimoto’s thyroiditis - is the most common type of hypothyroidism during pregnancy (4). Pregnant women with AITD are at increased risk of miscarriage and postpartum thyroid disease (including thyroiditis, hyperthyroidism and hypothyroidism). There is an increased risk of permanent and significant impairment in cognitive function for their infants (9).

Congenital Hyperthyroidism and Hypothyroidism

Congenital hyperthyroidism is rare in neonates. Transient congenital hyperthyroidism is caused by maternal Graves disease. Thyroid stimulating immunoglobulin passes from the mother to the fetus via the placenta and causes thyrotoxicosis in the fetus and subsequently, the neonate. After the baby is born, improvement is rapid if the condition is treated using antithyroid drugs and the hyperthyroidism will subside within several weeks (10). Persistent congenital hyperthyroidism is a familial non-autoimmune disease. It is caused by a genetic mutation resulting in an increase in the constitutive activity of the TSH receptor (11).

Congenital hypothyroidism due to maternal iodine deficiency is a leading cause of preventable mental retardation (10). Over-treatment of thyroid hormone, during pregnancy, as well as prolonged maternal iodine therapy (more than two weeks of therapy or more than 1000 µg/iodine) can also cause congenital hypothyroidism (6). The condition is exacerbated by coexisting selenium and vitamin A deficiencies or iron deficiency (5). Treatment for neonatal hypothyroidism should be started as soon as possible, as every day of delay may result in loss of IQ. Unless treated shortly after birth (within the first 18 days of life), the resulting mental retardation will be irreversible (10).

Postpartum Thyroiditis

Postpartum thyroiditis, an autoimmune inflammation of the thyroid, occurs within the first year after delivery or sometimes after termination of pregnancy. It can be a transient thyroid dysfunction with a brief thyrotoxic phase followed by hypothyroidism, usually with a spontaneous resolution (10). Smoking is a significant precipitating factor in the onset of postpartum thyroiditis (9). Women with a past history of postpartum thyroiditis have a risk of long-term permanent hypothyroidism and recurrence of postpartum thyroiditis in subsequent pregnancies (12). Tests for this condition consist of radioactive products necessitating a temporary cessation of breastfeeding (usually up to 3 days).

Implications for WIC Nutrition Services

Individuals with thyroid disorders can benefit from WIC foods and WIC nutrition services can reinforce and support the medical and dietary therapy prescribed by the participants’ health care provider. The following nutrition education messages may be appropriate depending on the type of thyroid disorder:

- Encourage iodine sufficiency, unless contraindicated, with an adequate intake of foods high in iodine such as iodized table salt, bread, saltwater fish, kelp, egg yolks (because of iodine supplementation in chicken feed), milk and milk products (because of the treatment of cows with supplemental dietary iodine) (5). It is important to note that the salt used in manufactured foods is not iodized.
- Advise women to review the iodine content of their prenatal supplement. It is recommended that all prenatal vitamin-mineral supplements for use during pregnancy and lactation contain at least

150 micrograms of iodine a day (13). Currently, less than 50 percent of prenatal vitamins on the market contain iodine (5, 7).

- Promote breastfeeding, as there are no contraindications to breastfeeding and thyroid hormone replacement therapy as long as normal thyroxine levels in the maternal plasma are maintained. Breast milk provides iodine to the infant and is influenced by the dietary intake of the pregnant and lactating mother (14). Hyperthyroidism can develop for the first time during the postpartum period, but the mother's ability to lactate is not affected. However, if a woman with untreated hypothyroidism breastfeeds, her milk supply may be insufficient. In such instances, replacement thyroid hormone therapy is necessary to help increase milk production.
- Weight management - hyperthyroidism: The elevated plasma levels of thyroid hormones may cause increased energy expenditure and weight loss along with increased appetite. Following medical treatment, individuals with hyperthyroidism usually regain their typical body weight with a concurrent decrease in appetite (4). Therefore, the monitoring of weight status and dietary adequacy are recommended.
- Weight management – hypothyroidism: Many individuals with hypothyroidism experience an increase in weight due to both a decrease in basal metabolic rate and an excessive accumulation of water and salt. Most of the weight gained is due to the excess water and salt retention. After medical treatment, a small amount of weight may be lost, usually less than 10% of body weight (15). Once hypothyroidism has been treated and thyroid hormones are within normal levels, it is less likely that the weight gain is solely due to the thyroid. If an overweight condition persists, weight control therapy may be necessary.
- Recommend the cautionary use of soy formula and the avoidance of foods or supplements rich in soy, fiber, or iron when therapeutic thyroid medications are prescribed, since soy, iron, calcium, fiber and phytates may interfere with the absorption of oral thyroid hormone therapy (16, 17).
- Discourage smoking as the compound thiocyanate found in tobacco smoke inhibits iodine transport (9).

References

1. National Academy of Sciences. Institute of Medicine. Food and Nutrition Board. Dietary reference intakes: The essential guide to nutrient requirements. 2001.
2. Los Alamos National Labs Chemistry Division Periodic Table. <http://periodic.lanl.gov/elements/53.html>. Accessed December 2009.
3. WebElements: The periodic table on the web. <http://www.webelements.com/iodine/>. Accessed December 2009.
4. Smallridge RC, Glinoe D, Hollowell JG, Brent G. Thyroid function inside and outside of pregnancy: What do we know and what don't we know? *Thyroid*. 2005;15(1):54-59.
5. Lee SL, Ananthakrishnan S, Pearce EN. Iodine deficiency. [updated July 27, 2006]. Available from: <http://www.emedicine.com/med/topic1187.htm>.
6. Nguyen PH. Autoimmune thyroid disease and pregnancy. [updated December 21, 2004;cited 2007 Sept 7]. Available from: <http://www.emedicine.com>.

7. American Association of Clinical Endocrinologists (AACE) Thyroid Task Force. AACE Medical guidelines for clinical practice for the evaluation and treatment of hyperthyroidism and hypothyroidism. 2006 Amended Version. *Endocrine Practice* 2002;8(6):457-469.
8. LaFranchi SH, Haddow JE, Hollowell JG. Is thyroid inadequacy during gestation a risk factor for adverse pregnancy and developmental outcomes? *Thyroid* 2005;15(1):60-71.
9. Muller AF, Drexhage HA, Berghout A. Postpartum thyroiditis and autoimmune thyroiditis in women of childbearing age: Recent insights and consequences for antenatal and postnatal care. *Endocrine Reviews*. 2001 Oct; 22(5):605-630.
10. Association for Clinical Biochemistry, British Thyroid Association, British Thyroid Foundation. UK guidelines for the use of thyroid function tests. 2006 July;1-86.
11. Polak M, Legac I, Vuillard E, Guibourdenche J, Castanet M, Luton D. Congenital hyperthyroidism: The fetus as a patient. *Horm Res*. 2006;65:235-242 (DOI: 10.1159/000092454)
12. O'Malley B, Hickey J, Nevens E. Thyroid dysfunction – weight problems and the psyche: The patients' perspective. *J Hum Nutr Dietet*. 2000 May;13:243-248.
13. American Thyroid Association. American Thyroid Association statement on early maternal thyroidal insufficiency: Recognition, clinical management and research directions. Consensus statement #2. *Thyroid*. 2005;15(1):77-79.
14. Riordan J. *Breastfeeding and human lactation*, 3rd ed. Jones & Bartlett Publishers, Inc., 2005; p. 157, 461.
15. American Thyroid Association. Thyroid and weight, 2005. Available from: <http://www.thyroid.org>
16. American Academy of Pediatrics. American Thyroid Association, Lawson Wilkins Endocrine Society. Update of newborn screening and therapy for congenital hypothyroidism. *Pediatrics*. 2006; Jun;117(6): 2290-2303.
17. American Academy of Pediatrics Committee on Nutrition. Use of Soy protein-based formulas in infant feeding. *Pediatrics*. 2008;121(5):1062-1068 (doi:10.1542/peds.2008-0564).

Additional References

Hashimoto's Thyroiditis online reference:

http://www.medicinenet.com/hashimotos_thyroiditis/article.htm

Clarification

Self-reporting of a diagnosis by a medical professional should not be confused with self-diagnosis, where a person simply claims to have or to have had a medical condition without any reference to professional diagnosis. A self-reported medical diagnosis (“My doctor says that I have/my son or daughter has...”) should prompt the CPA to validate the presence of the condition by asking more pointed questions related to that diagnosis.

Iodine (I₂) is an element. In the ambient temperature, it is volatile and forms blue-violet gas. In nature, it does not exist as free element. Instead, it forms compounds, such as sodium **iodide** (NaI), and potassium **iodide** (KI). To prevent iodine deficiency, potassium iodide is added to the salt (most commonly to table salt) to form iodized salt (2, 3).