

Local Guideline



John Hunter
Children's Hospital
CHILDREN, YOUNG PEOPLE AND FAMILIES



Health
Hunter New England
Local Health District

Thyroid Disorders in the Neonate

Sites where Local Guideline applies	JHCH NICU and JHH Maternity Services
Target audience:	All clinical staff that provide care to neonates.
Description	To provide information about thyroid disorders in the neonate to enable appropriate care for the infant as well as education for the mother about thyroid disease.
This Local Guideline applies to:	
1. Adults	Yes
2. Children up to 16 years	No
3. Neonates – less than 29 days	Yes Approval gained from the Children Young People and Families Network on
Keywords	Graves' Disease, hormones, hyperthyroidism, hypothyroidism, NBST, thyroid, T ₄ , T ₃
Replaces Existing Local Guideline and Procedure	No
Related Legislation, Australian Standards, NSW Health Policy Directive, NSQHS Standard/EQuIP Criterion and/or other, HNE Health Documents, Professional Guidelines, Codes of Practice or Ethics:	
<ul style="list-style-type: none"> NSW Health Policy Directive 2014_036 Clinical Procedure Safety 	
Prerequisites (if required)	Nil
Local Guideline Note	This document reflects what is currently regarded as safe and appropriate practice. The guideline section does not replace the need for the application of clinical judgment in respect to each individual patient but the procedure/s requires mandatory compliance . If staff believe that the procedure/s should not apply in a particular clinical situation they must seek advice from their unit manager/delegate and document the variance in the patient's health record.
Position responsible for the Local Guideline and authorised by	Dr Paul Craven, Director of Newborn Services, JHCH
Contact Person	Jennifer Ormsby, NICU Guideline Development Coordinator, NICU, JHCH
Contact Details	Phone: 02 4985 5304 Email: Jennifer.Ormsby@hnehealth.nsw.gov.au
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This Local Guideline contains advice on therapeutics	Yes Approval gained from Local Quality Use of Medicines Committee on June 2016
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RISK STATEMENT

This local guideline has been developed to provide guidance to clinical staff in NICU to assist in assessment and management of thyroid disorders in the neonate. It ensures that the risks of harm to the infant whilst being assessed and managed for a thyroid disorder are identified and managed.

Any unplanned event resulting in, or with the potential for injury, damage or other loss to

infants/staff/family as a result of this management must be reported through the Incident Information management System and managed in accordance with the Ministry of Health Policy Directive: Incident management PD2007_061. This would include unintended injury that results in disability, death or prolonged hospital stay.

RISK CATEGORY: Clinical Care & Patient Safety

OUTCOMES

1	Clinical care and support given to infants with thyroid disorders and their mothers and infants of mothers with thyroid disease
2	Guidance and education provided to clinical staff of NICU, the post-natal ward and delivery suite and other staff caring for infants with thyroid disorders and infants of mothers with thyroid disease.

ABBREVIATIONS & GLOSSARY

Abbreviation/Word	Definition
AABR	Automated auditory brainstem response
APEG	Australasian Paediatric Endocrine Group
CP	Cerebral palsy
IQ	Intelligence quotient
IUGR	Intrauterine growth restriction
IVH	Intraventricular haemorrhage
NBST	Newborn screening test
NICU	Neonatal Intensive Care Unit
^{99m} Tc pertechnetate	Radiopharmaceutical of choice for thyroid gland imaging
OAE test	Otoacoustic emission test (hearing test)
PTU	Propylthiouracil – medication to inhibit thyroid hormone synthesis and inhibit conversion of T ₄ to the active form T ₃
RDS	Respiratory Distress Syndrome
T ₄	Thyroxine
fT ₄	Free (not bound to thyroglobulin) thyroxine
T ₃	Triiodothyronine (three to four times more potent than T ₄)
TBG	Thyroglobulin (thyroid hormone binding protein)
TRH	Thyrotropin-releasing hormone
TSH	Thyroid-stimulating hormone
TSH Rc-Ab	TSH receptor antibody

Thyroid Disorders in the Neonate – One Page Summary and Checklist

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Summary of Screening a Newborn for Thyroid Dysfunction After Birth

Maternal thyroxine therapy is not an automatic indication for doing neonatal thyroid function tests.

All babies have a Newborn Blood Screening Test done after 48 hours that screens for primary congenital hypothyroidism

Babies born < 1500 gm require a second NBST done at 1 month of age.

Maternal Hashimoto's Disease: NBST screening alone is adequate.

Maternal Dyshormonogenesis: NBST screening plus TSH and fT4 once at 2-5 days.

Maternal thyroid absence or ectopic gland: NBST screening alone is adequate.

Maternal Graves' disease:

- Assess thyroid antibodies in 3rd trimester of pregnancy
 - (Titres > 5 X normal indicate high risk for neonate).
- Check TSH, fT4 and thyroid antibodies (TSH receptor stimulating) on cord blood
- For high risk babies repeat TSH and fT4 at 2-5 days
- For high risk babies repeat TSH and fT4 at 7-14 days or sooner if symptomatic

Maternal Anti-thyroid Medication Therapy:

- Check TSH, fT4 on cord blood
- Repeat TSH and fT4 at 2-5 days

Repeat TSH and fT4 at 10-14 days or sooner if symptomatic

GUIDELINE

This Guideline does not replace the need for the application of clinical judgment in respect to each individual patient.

Thyroid disorders in neonates**Introduction**[Top](#)

Thyroid hormones are essential for the developing brain. They are also essential in thermogenesis, metabolism and growth and affect function in nearly every body system.

There are two biologically active thyroid hormones: T_3 and T_4 . T_3 and T_4 are products of the thyroid gland (the ratio of T_4 to T_3 released into the blood is roughly 20 to 1). T_3 is mainly produced by de-iodination of T_4 in the periphery. T_3 is the active thyroid hormone that regulates metabolism. Reverse T_3 (rT_3) hormone is also produced by conversion from T_4 . rT_3 is generally inactive and eliminated quickly but may build up under certain circumstances. rT_3 can bind to T_3 receptors to block T_3 action and confuse diagnosis if only normal T_3 & T_4 are assayed.

Iodine is essential for normal thyroid function. The fetus is dependent on maternal iodine intake throughout pregnancy and iodine is transferred across the placenta for fetal thyroid hormone production.

Thyrotropin-releasing hormone (TRH) produced in the hypothalamus stimulates the pituitary gland to produce thyroid-stimulating hormone (TSH), which in turn stimulates the thyroid gland to produce thyroid hormones. When T_3 and/or T_4 concentrations are low, the production of TSH is increased and, conversely, when T_3 and/or T_4 concentrations are high, TSH production is decreased. This is an example of a negative feedback loop, shown diagrammatically below.

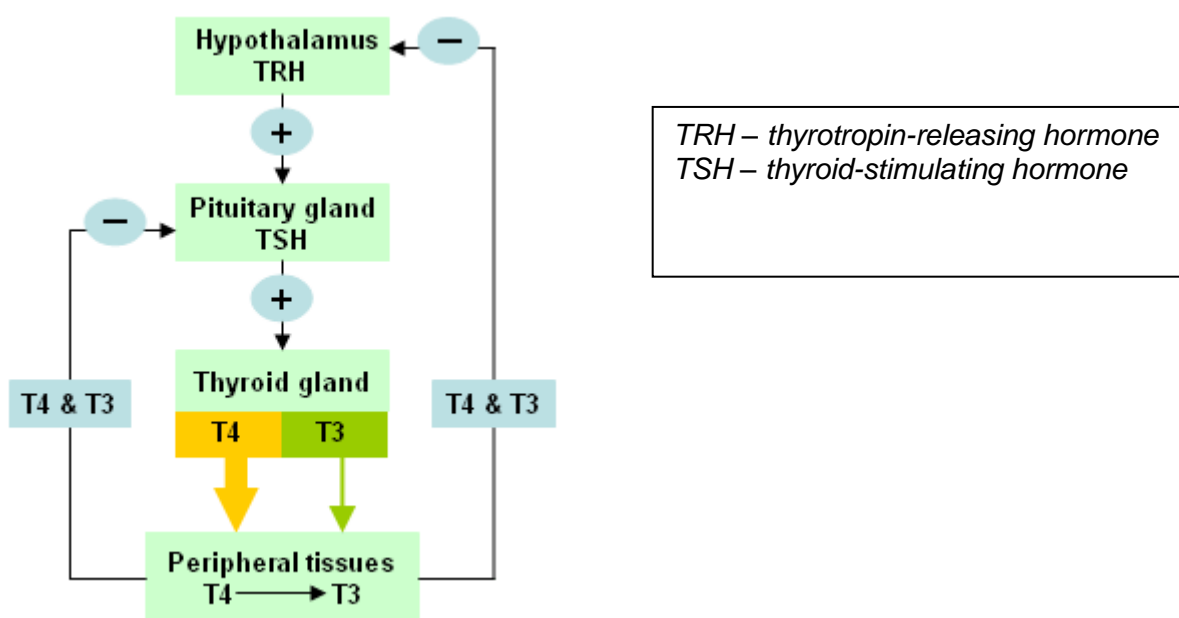


Image1: Hypothalamic-pituitary-thyroid axis (from BMJ Best Practice).

A number of conditions may impact on neonatal thyroid function, including gestation, postnatal age, non-thyroid illness, drugs, maternal thyroid disease, maternal iodine deficiency, iodine excess in baby and various causes of congenital hypothyroidism.

Thyroid function in the newborn

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Term infant

The normal infant born at term has a marked surge of TSH in the first 30 minutes following birth. This surge continues until about 24 hours of age, after which TSH decreases to close to adult concentrations over the next 4 weeks.

The neonatal brain requires T_3 ; however only T_4 (and not T_3) can cross the blood brain barrier and is then converted to T_3 . Hence, oral or subcutaneous administration of T_3 alone is not adequate treatment for hypothyroidism.

Preterm infants

The thyroid axis in a preterm infant is immature but in general shows the same changes after birth as a normal term infant with less magnitude.

When a preterm baby is born, the concentration of T_4 is lower than that of a term baby but correlated with gestational age and birth weight. Concentrations of TSH and T_3 are normal to low, free T_4 is low and thyroglobulin (TBG) concentrations are high. This reflects an increase in the production of iodinated thyroid hormone precursors in the thyroid gland. Responses to TSH and fT_4 are normal, indicating that the site of immaturity is in the hypothalamic-pituitary axis.

In preterm infants of more than 30 weeks gestation, T_4 rises to its peak at 1 week of life and then falls. The concentrations then overlap those of a term infant. In the more preterm infants i.e., less than 30 weeks gestation, T_4 falls in the first week of life before gradually rising to overlap that of a term infant by 3–6 weeks of age.

Preterm infants with respiratory distress syndrome (RDS) or other medical issues tend not to have the expected postnatal increases in TSH, T_3 and T_4 (termed sick euthyroid syndrome). Hence concentrations of these hormones may remain low until the infant recovers and/or begins to gain weight.

The hypothyroxinaemia observed in premature infants is multifactorial and usually considered physiological. Reasons for this hypothyroxinaemia include immaturity of the hypothalamic-pituitary axis, loss of maternal T_4 contribution, decreased responsiveness of the thyroid gland to TSH and immaturity of peripheral tissue deiodination. In preterm infants there is an association between adverse outcomes and more severe hypothyroxinaemia. In its severe forms, it has been associated with an increase in perinatal mortality and morbidity, prolonged requirement for supplemental oxygen therapy, mechanical ventilation and longer hospital stays as well as increased risks for intraventricular haemorrhage (IVH), decreased IQ and cerebral palsy (CP). This increased risk persists even when hypothyroxinaemia is corrected although potential confounders include gestation, fetal growth and illness severity.

Thus, although severe hypothyroxinaemia is associated with neonatal morbidity and developmental disability, it is not recommended that these infants should be supplemented with thyroxine; indeed this may in fact be detrimental to long-term neurological outcome.

Newborn screening test (NBST)

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Newborn screening is done at on all neonates (with consent from parents) at close to 48 hours after birth. As mentioned, after birth there is an initial physiological surge of TSH which may be modestly elevated in the first few days hence the >48 hour after birth timing of the test. Refer to NICU CPG "[Newborn Screen Test in NICU](#)" JHCH NICU 16.02.

In Australia the primary screening for congenital hypothyroidism is a TSH assay. This detects neonates with primary hypothyroidism but not those with a deficiency in TSH. Infants with hypothyroidism and TSH deficiency are rare and most commonly have hypopituitarism. They often present with hypoglycaemia and a small phallus in males.

If there is a borderline elevation of TSH on the NBST a repeat specimen is requested. All neonates with high concentrations on the first sample or a persistently elevated TSH on the second sample will need urgent biochemical and clinical assessment. The referring doctor will be contacted via phone followed by written notification from the screening laboratory.

Infants born prematurely and are < 1500 grams birth weight should have a repeat newborn screening test done at 1 month of age.

Infants of mothers with thyroid disease

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Maternal hypothyroidism

Hypothyroidism, both overt and subclinical, is common in women of reproductive age and during pregnancy, with frequencies ranging from 0.3% to 2.5%. Hypothyroidism has adverse effects on the course of pregnancy and development of the fetus. A women who has hypothyroidism needs to increase her thyroxine dose when she takes the oral contraceptive pill or becomes pregnant because her TBG concentrations increase and this results in decreased free T₃ and T₄ Failure to increase the dose will cause the fetus to have low thyroid hormone levels in the 1st trimester of pregnancy and can have severe consequences.

Hashimoto's thyroiditis causing hypothyroidism

Hashimoto's thyroiditis is the most common cause of maternal thyroid disorder. Pregnancy complications include preterm delivery, intrauterine growth restriction, post-partum haemorrhage and impaired foetal brain development. However, there are usually no significant effects on the neonatal thyroid gland.

Treated Graves' disease causing hypothyroidism

Graves' disease may be managed medically, by surgical resection or radio-iodine ablation. All treatments have the potential to render the mother hypothyroid. Importantly, the mother may still be producing TSH receptor antibodies after treatment so there is potential for neonatal thyrotoxicosis in the infant caused by trans placental transfer of those antibodies. It is recommended to assay for TSH receptor antibodies during the 3rd trimester in order to

assess risk for neonatal thyrotoxicosis. Titres of 5 times normal indicate a significant risk for development of neonatal thyrotoxicosis.

Maternal congenital hypothyroidism causing hypothyroidism

Most frequently the cause of congenital hypothyroidism in the mother is gland ectopia or agenesis. Less commonly, dysmorphogenesis may be the cause. In all such cases, hypothyroidism in the newborn will be detected by elevation of the TSH on the NBST. In the rare case of hypothalamic-pituitary disorders, TSH will be low and hypothyroidism will NOT be detected by the newborn screening test.

In the case of suspected dysmorphogenesis, a family history of thyroid dysfunction or Pendred syndrome (a genetic disorder leading to congenital, bilateral sensorineural hearing loss and goitre with euthyroid status or mild hypothyroidism) a hearing test should be performed. The Australasian Paediatric Endocrine Group (APEG) suggests AABR or OAE testing at 4–8 weeks with three monthly testing for at least the first year in such cases.

Maternal hyperthyroidism

Graves' disease causing hyperthyroidism

Graves' disease is an auto-immune disorder. A small percentage of the neonates born to women with active Graves' disease will develop neonatal thyroid disease. This is caused by the trans-placental passage of TSH receptor antibodies (TSH Rc-Ab). Mothers with the highest titres are most prone to have their infants develop neonatal thyrotoxicosis.

It is therefore recommended that **all women with active Graves' disease or a history of previously treated Graves' disease have the TSH Rc-Ab titres checked at 28–32 weeks gestation.** Babies born to mothers with Graves' disease may be hyperthyroid, hypothyroid or euthyroid at birth depending on the balance of maternal stimulating or inhibitory antibodies and anti-thyroid drug effects.

All babies born to hyperthyroid women need to have their thyroid function and TSH receptor antibody status checked at birth or shortly thereafter (day 3 and 7).

Neonatal thyroid disease

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Congenital hypothyroidism

Congenital hypothyroidism is due to dysgenesis of the thyroid gland in 80–90% of cases. The incidence in Australia is about 1:3600 live births. Transient hypothyroidism, due to maternal antibodies/drugs, iodine exposure etc. is less common.

The infant should be evaluated as soon as possible including history (maternal drugs and diet need consideration) and clinical examination for evidence of hypothyroidism, including a goitre and growth parameters. Look for evidence of other congenital malformations, especially congenital heart disease.

Investigations for congenital hypothyroidism

Serum TSH and fT₄

- Confirms the result from the NBST and determines the degree of hypothyroidism

Plasma bilirubin

- Jaundice is associated with hypothyroidism

Maternal and infant anti-thyroid antibodies if history of maternal autoimmune thyroid disease

- TSH receptor antibodies are preferred. Anti-thyroglobulin and anti-thyroid peroxidase antibodies may provide indirect evidence of maternally transmitted thyroid auto-immunity. High titres correlate with significant neonatal disease. If maternal antibodies are known prior to delivery, these tests can be done on cord blood.

Thyroid scan (^{99m}Tc pertechnetate)

- Provides diagnostic information and is performed within 5 days of starting therapy. The scan should not delay starting treatment. An ultrasound to determine the presence of a thyroid gland can be performed if there is no uptake on the thyroid scan. If the gland is present on ultrasound this indicates the possible presence of blocking antibodies and transient hypothyroidism or dyshormonogenesis but does not change initial management.
- Potential scan findings
 - Absent isotope uptake:
 - Agenesis of thyroid gland or maternal blocking antibodies
 - Reduced uptake or in an abnormal position:
 - Hypoplastic or ectopic gland
 - Increased uptake in normal position:
 - Inherited defect of thyroxine biosynthesis, dyshormonogenesis or excessive iodine exposure

X-ray of the knee

- There is a positive correlation between T₄/T₃ and the size of the knee epiphyses. Markedly delayed development may indicate a worse intellectual prognosis.

Hearing tests

- Important for all infants diagnosed with hypothyroidism. Initially done at 4–8 weeks then repeated 3 monthly for the first year and yearly until school age

Treatment of congenital hypothyroidism

Thyroxine should be started as soon as possible. **Recommended starting dose is 10 microgram/kg/day.** Doses need to be adjusted at follow up visits with an aim to keep free T₄ at the upper end of the normal range and TSH in the normal range (usually maintenance dose is 8–12 microgram/kg/day, max 15 microgram/kg/day). It may take

several months for the TSH to normalise. However slow resolution should raise concern about inadequate dose and or administration.

Thyroxine tablets require storage in the refrigerator. A more expensive less potent more temperature resistant preparation is available but usually not recommended. Tablets are generally loosely crushed (not ground with mortar and pestle) and administered with milk (not soy formula as it impairs absorption). As the active thyroxine T₃ is the product of deionisation of thyroxine (T₄) it takes some time for steady state to be achieved. This also means that the dose per week is more important than daily dose and it is acceptable to vary dose day to day if this aids compliance. E.g. half a tablet on all days of the week except Wednesday and Saturday when a full tablet is given. Use of a pill box should be strongly encouraged to improve reliability of medication delivery.

Thyroid function tests should be taken just prior to a dose of thyroxine as the serum concentrations of free thyroxine may increase significantly following an oral dose of thyroxine while TSH and fT₃ are not affected.

If there is overtreatment with thyroxine, the child may develop craniosynostosis, accelerated growth and maturation, diarrhoea, disturbed sleep patterns, behavioural problems and effects on temperament.

If it is suspected that the congenital hypothyroidism is transient, the child can safely be trialled off therapy when 3 years old. If this is strongly suspected i.e., if there is maternal thyroid disease or thyroid receptor antibodies are present, this can be trialled earlier by very gradually weaning the dose. Close monitoring during this time is essential.

Follow up of congenital hypothyroidism

Follow up of these patients is vital to ensure normal growth and development. The aim is for the TSH to be in the normal range and free T₄ in upper range of normal prior to each visit. Formal developmental assessment may be indicated and the recommended frequency of follow up visits is:

- 2 weeks after starting therapy
- 6 weeks of age
- 3 months
- Then 2–3 monthly for the first year of life
- Then 3 monthly
- After 3 years of age every 4–6 months

Regular hearing tests should be performed if the child has (or is suspected to have) dysmorphogenesis.

There is no need to check bone age if growth patterns are normal.

Compliance with medication

Compliance with treatment is vitally important to ensure the best possible outcome for these patients and the importance of this must be stressed to the parents. Poor compliance may be suggested by a high TSH and high/normal fT₄. This occurs when thyroxine has been given regularly in the days prior to the blood test, but erratically over the previous weeks to months.

Transient hypothyroidism

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Transient congenital hypothyroidism is diagnosed by high TSH and low free T₄ soon after birth but with spontaneous resolution over time. The major causes of transient congenital hypothyroidism are iodine deficiency, iodine excess and passive transfer of maternal thyrotropin receptor blocking antibodies (see maternal section).

Iodine deficiency

Iodine is essential for normal thyroid function. Iodine deficiency is common in the developing world as well as in Australia. Up to 26% of women in Australia are found to be iodine deficient. Iodine supplementation to meet recommended daily intake recommendations is often provided in bread, salt and other foodstuffs. The World Health Organisation recommends a daily iodine intake of 150 microgram/day, increasing to 200–250 microgram/day during pregnancy and lactation as pregnancy is a state of increased thyroid hormone demand.

Iodine excess

Infants exposed to excess iodine are at risk of iodine overload resulting in transient hypothyroidism. The risk factors for neonatal iodine overload include maternal exposure to iodine (skin disinfectants, amiodarone) and post-natal exposure to iodine, including antiseptics and iodinated contrast material for radiological purposes.

Congenital hyperthyroidism/neonatal thyrotoxicosis

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Neonatal thyrotoxicosis is rare, but may be associated with significant morbidity and mortality (12–20%, usually from heart failure) if unrecognised or inadequately treated. It should be considered a medical emergency. The prevalence of Graves' disease in pregnant women is approximately 0.2% and 1–5 % of these women's babies will have hyperthyroidism.

The usual cause of hyperthyroidism in the newborn is maternal Graves' disease. More rarely, but transient, is Hashimoto's thyroiditis (with trans-placental transfer of thyroid stimulating immunoglobulins from the mother). The third cause of neonatal thyrotoxicosis is due to an activating mutation of the TSH receptor and this is not transient. As this is a dominantly inherited disorder, activating mutations of the TSH receptor should be suspected if the mother has had thyrotoxicosis since birth or there are other first degree relatives with thyrotoxicosis.

Clinical features of neonatal thyrotoxicosis

The clinical symptoms and signs are related to the degree of maternal thyrotoxicosis with complications being increased in mothers who remain hyperthyroid during the second half of pregnancy.

Signs can be detected in the fetus, such as tachycardia, arrhythmias, hydrops, intrauterine growth restriction (IUGR), advanced bone age and goitre. There is an increased risk of intrauterine death.

In the neonate, clinical symptoms and signs may be overt from birth but may take several (up to 10) days to develop, either due to maternal antithyroid medications or the effect of coexisting blocking antibodies. Most infants will have a goitre.

Central nervous system signs include irritability, restlessness and jitteriness.

They may have eye signs such as exophthalmos or lid retraction.

Cardiovascular signs include tachycardia and arrhythmias, but may proceed to cardiac failure which is the most common cause of death in these infants.

They may also have signs of hyper-metabolism such as increased appetite, diarrhoea and sweating.

Other signs include persisting acrocyanosis, hepatosplenomegaly and petechiae secondary to thrombocytopenia. Advanced bone age, craniosynostosis and microcephaly may be evident in both the fetus and newborn.

Investigation and treatment of neonatal thyrotoxicosis

As mentioned above, trans-placental transfer of TSH receptor antibodies from the mother with Graves' disease is the most common cause of neonatal thyrotoxicosis.

The diagnosis of the infant should be confirmed with serum TSH and free T₄. TSH antibodies should also be measured and can be collected from cord blood if the mother is known to have Grave's disease before the birth.

Babies at high risk of thyrotoxicosis are

- those who have had evidence of thyrotoxicosis in utero,
- those who have mothers with Graves' disease and are receiving antithyroid treatment and
- those whose mothers have high titres of thyroid stimulating immunoglobulins.

These infants may require close observation in hospital for the first few days after delivery. Although maternal thyroid stimulating immunoglobulins are usually five or more times the upper limit of normal much lower concentrations may also result in fetal and neonatal thyrotoxicosis.

Neonatal thyrotoxicosis is treated with thioamides – carbimazole or propylthiouracil (PTU). Both medications inhibit thyroid hormone synthesis by preventing the thyroid peroxidase enzyme from coupling and iodinating the tyrosine residues on thyroglobulin, hence reducing the production of the thyroid hormones T₃ and T₄. Propylthiouracil also (unlike carbimazole) inhibits tetraiodothyronine 5' deiodinase, which converts T₄ to the active form T₃.

Carbimazole: Initially 0.75–1 mg/kg (maximum 45 mg) daily in 2 or 3 doses, when euthyroid reduce to a maintenance dose of 30–60% of the initial dose

Propylthiouracil: Initially 2.5–5 mg/kg, 12 hourly, when euthyroid reduce to a maintenance dose of 30–60% of the initial dose

Use of carbimazole in pregnancy may be associated with aplasia cutis congenita in the infant. In children, carbimazole is usually preferred to propylthiouracil as it is less toxic.

A clinical response should not be expected until colloid stores are depleted. Adjunct treatments may include iodine containing solutions (e.g., potassium iodide, Lugol's solution), β -blockers (for tachycardia), diuretics/digoxin (for cardiac failure) and prednisolone. Glucocorticoids suppress thyroid hormone release and decrease the peripheral deiodination of T_4 to T_3 .

The treatment can usually be withdrawn after several weeks as the maternal antibodies are cleared. Cessation of treatment is determined by clinical features and TSH and fT_4 concentrations.

Suspected or confirmed cases should be promptly referred to/discussed with a paediatric endocrinologist.

Figures 2 and 3 show a suggested pathway for the investigation and management of infants at risk of thyrotoxicosis.

Figure 2 – Investigations of infants at risk of hyperthyroidism

Investigation of infants at risk of hyperthyroidism
At birth: Clinical examination and take cord blood: TSH, fT_4 , Thyroid stimulating immunoglobulins
Age 3 & 7-10 days: If high risk: repeat TSH, fT_4 and examination

Figure 3 – Action based on thyroid function tests

Thyroid function test results		
Normal	Hypothyroid	Hyperthyroid
↓	↓	↓
No treatment	Repeat TSH, fT_4	Consider treatment (Carbimazole/Propylthiouracil \pm others)
If the child remains hypothyroid on repeat testing or is hyperthyroid contact paediatric endocrinologist.		

Breastfeeding advice

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- Maternal hypothyroidism – there are no contraindications to breastfeeding for mothers using thyroxine
- Maternal hyperthyroidism – there are no contraindications to breastfeeding for mothers using propylthiouracil (PTU) or carbimazole. However, PTU is excreted in lower concentration in breast milk than carbimazole, therefore PTU would be preferable. **Radioiodine is an absolute contraindication to breastfeeding**

Summary

- All infants in NSW will have TSH measured through the NBST done after 48 hours (unless parents decline the test). This will pick up most infants with hypothyroidism except those who have central hypothyroidism
- Infants born prematurely < 1500 grams birth weight should have a repeat NBST done at 1 month of age
- The infants who require further thyroid function tests (\pm TSH receptor antibodies) in addition to the standard NBST are those whose mothers are hyperthyroid, have treated Graves' disease with remaining high maternal TSH receptor antibody titres, congenital hypothyroidism secondary to activating TSH receptor mutation or Pendred syndrome. The tests should be done at birth (cord blood is adequate but results can be difficult to interpret) and at day 3 and day 7-10 if considered high risk
- There is no need to do further testing or examination other than the standard NBST in infants whose mothers have autoimmune hypothyroidism/Hashimoto's thyroiditis
- Referral to a paediatric endocrinologist in all cases of neonatal hyperthyroidism and hypothyroidism

References[Top](#)

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AUTHOR Dr Kristin Gotaas Mansour, Paediatric registrar, JHCH

REVIEWERS Dr Chris Wake, Neonatologist, NICU, JHCH
 Dr Paul Craven, Neonatologist, NICU, JHCH
 Dr Donald Anderson, Paediatric Endocrinologist
 Susanne Wooderson Liaison CNC Newborn Services JHCH
 Mandy Hunter Clinical Midwife Consultant Maternity JHH
 Dr Koert de Waal Neonatologist NICU JHCH
 Michelle Jenkins Senior Pharmacist JHCH
 Karen Takats CMC Aboriginal Maternal & Perinatal Health/Acting Women’s Health & Maternity Network Coordinator

APPROVED:

NICU Executive Committee	1 st June 2016
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FEEDBACK

Any feedback on this document should be sent to the Contact Officer listed on the front page.