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North Bristol

Thyroid Function Testing

1. First line thyroid testing

TSH will be measured on all samples as a first line test and subsequent actions determined by the result.

In the absence of non-thyroidal illness and interfering medications TSH is a reliable screening tool, with a sensitivity 89-95% and specificity 90-96%. It has a high predictive value in ruling out thyroid disease and is cost effective. TSH may be within the normal range with hypopituitarism but as this is a rare presentation it is accepted as a screening tool.

	Result	
TSH	0.27 – 4.2mU/L	Result indicates patient is euthyroid. No further tests will be performed and a report issued with the result and the reference range only.
TSH	> 4.2mU/L-	Result indicates possible subclinical hypothyroidism
	10mU/L	Free T ₄ will be measured and a report issued containing both results
		and if appropriate an interpretative comment.
TSH	>10mU/L	Result indicates possible subclinical or overt primary hypothyroidism
		Free T ₄ will be measured and a report issued containing both results
		and if appropriate an interpretative comment.
TSH	<0.2mU/L	Interpretation depends on whether the patient is taking thyroxine.
		Free T4 will be measured and a report issued containing both results
		and if appropriate an interpretative comment.
		Consider adding FT3 if not on thyroxine or if pregnant.

2. Exceptions

1		
1	Treated thyrotoxic patients	All such patients will have FI_4 measured in addition to
		TSH. The results will be reviewed and if appropriate
		an interpretative comment added to the report.
2	On amiodarone	Ideally, these patients should have a FT4, FT3 and
		TSH.
3	Known or possible	All such patients will have FT ₄ measured in addition to
	hypopituitarism	TSH. The results will be reviewed and if appropriate
		an interpretative comment added to the report.
4	Children (<18 years)	All such patients will have FT ₄ measured in addition to
		TSH and if appropriate an interpretative comment
		added to the report.
5	Endocrinology Clinic patients	As many patients have complex problems the requester
	(Dr A Johnson, Dr V Parfitt,	will specify which tests are needed.
	Dr E Cheyne, Dr F Chau, Dr K	
	Lonnen)	

Any other request for additional tests will require discussion with the duty biochemist

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3. Thyroid antibody measurement

Thyroid peroxidase antibody (TPOAb) measurement should be considered in the following patients:

• Hyperthyroidism – to distinguish between autoimmune disease, thyroiditis and toxic multi nodular goitre.

• If risk factors are present for autoimmune thyroid disease.

• If subclinical hypothyroidism is confirmed by repeat TFT results, thyroid peroxidase antibodies should be requested by the requesting physician.

• Risk factors for hypothyroidism such as lithium, amiodarone, interferon-alpha and interleukin-2.

Thyroid antibodies should not be added routinely on hospital inpatients. (Repeat thyroid function tests should be requested after discharge).

Repeat testing of thyroid antibodies is not indicated.

TSH receptor antibody should be considered in the following patients:

- Hyperthyroid patients of uncertain aetiology.
- Euthyroid Grave's Ophthalmopathy
- Pregnant and past or present history of Grave's disease.
- Neonates with transient hypothyroidism due to TSH receptor blocking antibody

4. Thyroid function in pregnancy

Normal thyroid function is essential for fetus development.

In hypothyroid patients their thyroxine requirements increase, thereby requiring increases in thyroxine which can usually be reduced by 2-4 weeks post-partum.

Any woman who is thyrotoxic or taking anti-thyroid medication should be seen by a specialist prior to conception.

TFT's should be checked at diagnosis or antenatal booking in women with:

- Current thyroid disease
- Previous history of thyroid disease
- Type 1 Diabetes mellitus
- Goitre
- Symptoms of hypothyroidism

All pregnant women with thyroid pathology should have shared care with the obstetrician and endocrinologist who will set out a timetable for further testing.

Thyroid function testing during pregnancy should comprise both TSH and FT4 TPO-Ab should also be considered as this has predictive value for both post-partum thyroiditis and fetal impairment.

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It should be noted that trimester specific reference ranges need to be applied. In accordance with NBT endocrine and biochemistry departments the following reference ranges in pregnancy should be used:

	1 st Trimester	2 nd Trimester	3 rd Trimester
TSH mU/L	0.02-3.7	0.2-3.7	0.27-4.2
FT4 pmol/l	12.1-19.6	9.6-17.0	8.4-15.6

(TSH reference ranges quoted by the American Thyroid Association and FT4 reference ranges supplied by Roche).

5. Thyroid function in children

FT4 is analysed automatically in all patients under 18 years. Note pre- and full term infants show a rapid increase in TSH during the first 24 hours. Reference ranges agreed between all Bristol Trusts for TSH, FT4 and FT3 are detailed below:

	0 – 6d	6-14d	>14 days	
TSH (µIU/l)	0.7 - 15.2	0.72 - 11	0.27- 4.2	
fT4 (pmol/l)	11 – 32	11.5 – 28.3	12.0 - 22.0	
fT3 (pmol/l)	2.6 - 9.6	3-9.2	3.1-6.8	

6. Thyroid Function Testing in Thyroid Cancer

Following a total thyroidectomy and thyroid ablation treatment for thyroid cancer replacement thyroxine will be commenced. In general, Thyroxine is prescribed at a supra-physiological dose (usually 175-200mcg a day) to suppress TSH to less than 0.1mU/ll. However, for some low risk patients a TSH < 0.5mU/l may suffice. Full guidance is available at:

http://bahno.org.uk/wp-content/uploads/2014/03/UK-Head-and-Cancer-Guidelines-2016.pdf

Lifelong monitoring is required due to late reoccurrence being common. Annual monitoring should include:

- Clinical examination (and USS thyroid if available)
- TSH and thyroglobulin
- Calcium to assess possible hypocalcaemia post total thyroidectomy

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Interpretation

1. Sick euthyroid or Non-thyroidal illness

There is no thyroid pathology but the TFT's are disturbed by the influence of inflammatory cytokines on the thyroid gland.

TSH can often be suppressed during the acute phase of an illness but then there can be a rebound elevation on recovery.

FT3 is often well below the reference range and FT4 can be normal or elevated.

Routine testing of thyroid function in patients admitted acutely to hospital is NOT warranted unless specific clinical indications exist.

2. Hypothyroidism

TSH > 10mU/l, FT4 < 12.1 pmol/l

Most common aetiology in the UK is chronic autoimmune disease (Hashimoto's thyroiditis) or following surgical or radioactive iodine treatment for hyperthyroidism.

A TSH >10mU/l is associated with dyslipidaemia, subfertility, altered menstrual cycles, fetal loss and symptoms which can be improved by treatment.

Subclinical Hypothyroidism

TSH > 4.2 mU/l, FT4 and FT3 within the (low) normal range.

A repeat should be arranged in 3-6 months to exclude developing hypothyroidism.

TSH 4.2-10mU/l is not associated with an increased risk of mortality or ischaemic heart disease. Treatment should be considered with a rising TSH, pregnancy and the presence of goitre. It may progress to overt hypothyroidism so requires monitoring:

•If TPO antibody is positive monitor once a year.

•If TPO antibody negative monitor every 3 years as less likely to progress to overt hypothyroidism.

Once TSH > 10 mU/l there is an association with dyslipidaemia, cardiovascular mortality and symptoms. These improve on treatment and therefore thyroxine therapy is recommended.

Secondary hypothyroidism

Low TSH, Low FT4, Low FT3 Usually due to hypopituitarism. Consider additional pituitary function tests (FSH, LH, Cortisol/ACTH, IGF-1, Prolactin) and referral to endocrinology.

Congenital hypothyroidism

Is an important cause of treatable mental retardation and therefore is part of the Newborn screening heel prick test.

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3. Hyperthyroidism

TSH <0.02mU/l, raised FT4 or FT3 (or both)

The most common causes in the UK are Grave's disease and toxic multi-nodular goitre, although thyroiditis and toxic adenoma are also frequently seen.

It is associated with atrial fibrillation, subfertility, fetal loss, altered menstrual cycles, osteoporosis and vascular mortality.

When the TSH is suppressed <0.02mU/l and the FT4 >50pmol/l or FT3 >25pmol/l then an urgent referral to endocrinology is advised via FAX 0117 414 8129.

A routine referral is advised when TSH <0.02mU/l and FT4 is raised but <50pmol/l. It is advisable to have repeated TFT's to rule out a transient picture and to have tested thyroid peroxidase antibodies (available on ICE).

T3 Toxicosis

TSH < 0.02 mU/l, isolated raised FT3 and normal FT4.

The risks of hyperthyroidism are still present and require similar treatment.

The causes are an isolated toxic adenoma, early toxic multi-nodular goitre or early Grave's disease.

Thyroiditis

Transient TSH <0.02mU/l, Raised FT4/FT3

Thyroid cell destruction results in the release of preformed hormones.

Resolves in 6-8 weeks but can be followed by transient hypothyroidism and rarely more chronic hypothyroidism.

- Sub-acute (De Quervain's) is associated with a history of viral illness, a tender thyroid and raised viscosity.
- Autoimmune
- Post-partum (autoimmune in aetiology, more likely if positive thyroid antibodies during pregnancy)
- Riedel's fibrosis of the gland is of unknown aetiology

Subclinical Hyperthyroidism

Categories:

• TSH < 0.1mU/l, FT4 and FT3 normal.

There is an association with increased vascular mortality and atrial fibrillation therefore, if persistent, these patients should be referred to endocrinology.

Initially TFT's should be rechecked. The timing will depend on the clinical setting. Those who are elderly, who have symptoms or who have underlying vascular disease will require monitoring in 1-2 months but all others in 3-6 months.

If the subclinical hyperthyroidism is left untreated monitoring 6-12 monthly is required.

• TSH is reduced but >0.1mU/l, FT4 and FT3 normal

There is no evidence for any increased risk and a referral is not indicated.

Monitor every 6-12 months in case overt hyperthyroidism develops.

It should be noted that a low serum TSH may reflect 'non-thyroidal illness' or be seen with certain medications. This is a frequent finding in hospitalised patients but in primary care a TSH <0.1mU/l is more likely to indicate mild thyroid hormone excess.

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4. Drugs used in treating hyperthyroidism

Carbimazole (or propylthiouracil) aims to suppress thyroid hormone synthesis and conversion from FT4 to FT3. It can be used in isolation to partially suppress the thyroid, leaving enough endogenous thyroid hormone production or as a "block and replace" regime when the thyroid is totally suppressed. In this case exogenous thyroxine is given to maintain adequate thyroid hormone supply. Once carbimazole has been started TSH and FT4 should be measured 4-6 weekly for the first few months.

Propranolol can be used to control thyrotoxic sympathetic nervous system symptoms.

Radioactive iodine is used as thyroid ablation as a more definitive treatment.

When requesting thyroid function tests on patients on any of the above, they should have FT4 performed also. Monitoring is suggested 4-6 weekly after initiation of treatment.

5. Medications which may cause interference

Drugs may interfere with the production, secretion, transport and metabolism of thyroid hormones.

Certain agents will impair the absorption of thyroxine from the gut and patients on thyroxine therapy should be advised to take their thyroxine at least 4 hours apart from these medications.

Patients taking thyroxine are likely to require an increase in replacement dose if drugs such as phenytoin or carbamazepine are prescribed that increase hepatic metabolism of T4.

Decrease in TSH	Decreased Thyroid	Increased thyroid	Decreased thyroidal	Displacement of Hormone from	Impaired T4 to T3 Conversion
Secretion	Hormone	Hormone	synthesis*	Plasma	Conversion
	Secretion	secretion	-	Proteins	
		Decreased			
		thyroidal			
Dopamine	Lithium	Iodide	Methimazole	Frusemide	Beta antagonists
Dopaminergic	Iodide	Amiodarone	Carbimazole	Fenclofenac	Glucocorticoids
agents	Amiodarone	Lithium (rare)	Propylthiouracil	Salicylates	Amiodarone
Glucocorticoids			Lithium	Mefenamic acid	Propylthiouracil
Cytokines				Carbamazepine	Iopanoic acid
Octreotide				Non-steroidal	Radiocontrast dyes
				AIDs	
Increase TBG,	Decrease TBG,	Increased	Impaired	Alter	Modify Thyroid
TT3, TT4	TT3, TT4	Hepatic	Absorption of	autoimmunity**	Hormone Action
		Metabolism of	Thyroxine **	*	
		T4			
Oestrogens	Androgens	Phenytoin	Cholestyramine	Interleukin 1	Amiodarone
Tamoxifen	Anabolic	Carbamazapine	Cholestapol	Interferon α	
Heroin	steroids	Barbiturates	Aluminium OH	Interferon β	
Methadone	Glucocorticoids	Rifampacin	Ferrous S04	TNF α	
Clofibrate			PPI's		
Raloxifene			Calcium C03		



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Amiodarone and thyroid function

Amiodarone causes a wide spectrum of effects on the thyroid. TFT testing is required prior to starting amiodarone, every 6 months whilst on treatment and up to 12 months after.

In summary:

- 1. Serum T4 levels rise by 20-40% during the first month of therapy and then gradually fall toward high normal.
- 2. Serum T3 levels decrease by up to 30% within the first few weeks of therapy and remain slightly decreased or low normal.
- 3. TSH levels usually rise after the start of therapy but return to normal in 2-3 months.

Amiodarone inhibits 1 5'-deiodinase, thereby decreasing the peripheral conversion of T4 to T3 and reducing the clearance of both T4 and reverse T3 (rT3). Consequently, the serum levels of T4 and rT3 increase and the serum levels of T3 decrease by 20-25%.
Amiodarone inhibits entry of T4 and T3 into the peripheral tissue. Serum T4 levels increase by an average of 40% above pretreatment levels after 1-4 months of treatment with amiodarone. This, in itself, does not constitute evidence of hyperthyroidism.
Inhibition of 2 5'-deiodinase in the pituitary due to feedback regulation is seen in the first 1-3 months and leads to an increase in thyroid-stimulating hormone (TSH) levels. This is not an indication for T4 replacement in these patients. Serum TSH levels return to normal in 2-3 months as T4 concentrations rise sufficiently to overcome the partial block in T3 production. The response of TSH to thyroid-releasing hormone (TRH) may be reduced.
Amiodarone and its metabolites may have a direct cytotoxic effect on the thyroid follicular cells, which causes a destructive thyroiditis.

•Amiodarone can act as a competitive antagonist of T3 at the cardiac cellular level.

Amiodarone induced thyrotoxicosis:

Thyrotoxicosis may develop rapidly and there may be marked cardiac manifestations. All should be referred urgently to endocrinology although amiodarone should be continued until endocrinology can review as withdrawal may exacerbate hyperthyroidism and the half-life is long so there will be no immediate benefit anyway.

•Type 1 usually affects patients with latent or pre-existing thyroid disorders and is more common in areas of low iodine intake. Type 1 is caused by iodine-induced excess thyroid hormone synthesis and release (amiodarone contains 75mg per 200mg dose).

This form may be treated with propylthiouracil or potassium perchlorate.

•Type 2 occurs in patients with a previously normal thyroid gland and is caused by a destructive thyroiditis that leads to the release of preformed thyroid hormones from the damaged thyroid follicular cells.

This form may be treated with glucocorticoids.

Amiodarone induced hypothyroidism:

Amiodarone-induced hypothyroidism is more common in iodine-replete communities (up to 20%) and related to the presence of thyroid autoimmunity. It is managed in the same way as normal hypothyroidism and amiodarone may not always need withdrawing.

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Lithium and thyroid function

TFT testing is required prior to starting lithium and every 6-12 months whilst on treatment. The presence of TPO antibodies may indicate those in whom thyroid effects may be more likely.

Lithium commonly causes goiter, subclinical and hypothyroidism. Hyperthyroidism is less common

6. Thyroid test interference

All assays can be affected by interference which may cause falsely low or elevated results. If the thyroid results do not fit the clinical picture it may be worth discussing with the duty biochemist. For patients on thyroxine dosing and compliance issues should be ruled out first.

Within the laboratory the duty biochemist may consider the following options:

If normal FT4 with inappropriate TSH;

• TSH can be evaluated by a method comparison using the Abbott platform at Newport Laboratory.

If TSH confirmed to be inappropriate

• SHBG, other pituitary hormones and alpha subunit if TSHoma suspected

• Referral to Supra-regional assay service. Cambridge offer TSH method comparison on Centaur, PEG and dilution, equilibrium dialysis and gel filtration chromatography for macroTSH.

If raised FT4 with normal TSH

• Consider referral to supra-regional assay service. Cambridge offer method comparison on a 2 step assay DELFIA, PEG and dilution, TT4/TBG to calculate FT4 and Equilibrium dialysis.

Prior to sending discuss with Cambridge to establish which tests to perform as choice and costs will vary depending on clinical picture.

7. Inappropriate TSH

FT4 or FT3 are elevated with an inappropriate TSH that is detectable or elevated.

This picture may be seen when a patient on thyroxine therapy is poorly compliant. Discussion with the duty biochemist is advised as it can be a difficult area to explore. However, if the patient is thought to be compliant the following issues may be considered:

- 1. Assay interference (As above).
- 2. Thyroid Hormone Resistance

Autosomal Dominant trait but has variable presentations. In generalised resistance the patient is most often euthyroid but may have a goitre or tachycardia. There is a defect in the beta subunit of the thyroid hormone receptor which causes peripheral tissues to be resistant to FT4 and FT3.

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FT3/FT4 are increased and TSH is normal or high. The TSH responds to a TRH test. Confirmation of the diagnosis can be made by the thyroid hormone receptor β -gene sequence analysis available through the Supraregional Assay Service. The decision to treat these patients is often made on clinical grounds rather than the biochemical abnormality

- TSH secreting adenoma
 A rare cause of hyperthyroidism is a pituitary tumour that releases TSH.
 Biochemistry will show a raised TSH, with raised FT3 and FT4.
 There may be an elevated SHBG and alpha subunit. Other pituitary hormones should be assessed.
 Pituitary imaging usually is diagnostic.
- 4. Familial dysalbuminaemic hyperthyroxinaemia.

Autosomal dominant trait in which there is an albumin variant with an increased affinity for T4. Therefore Total T4 is increased, with a normal TSH. The patient is euthyroid. In the past when total T4 was measured it would often cause diagnostic confusion. Now it is noted less often because first line screening uses TSH alone and if appropriate free T4 is measured not total. However, occasionally free T4 can also be raised.

Diagnosis is made through taking a family history, screening with IEF electrophoresis and genetic testing (both via supraregional centres).

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Thyroid coded comments

Euthyroid TEU	Euthyroid picture
Taking thyroxine: TARE	Results suggests dose of thyroxine is adequate.
Sick Euthyroid TSIC	Sick euthyroid syndrome. Routine testing of thyroid function in patients who are acutely unwell or hospitalised is not warranted unless specific clinical indications exist. Repeat TFT's when clinically well.
Hypothyroidism THPO	Hypothyroid picture
SCHB	Borderline Hypothyroidism. If symptomatic a trial of thyroxine is recommended however if asymptomatic suggest excluding concurrent illness or interfering drugs then repeat in 2-3 months.
Taking thyroxine: TUR	If a regular dose of thyroxine has been taken for at least 8 weeks, results may reflect under-replacement, non-thyroidal illness, poor compliance or thyroxine malabsorption (e.g. due to drugs that interfere with thyroxine absorption such as FeSO4).
СОМ	This picture is most often seen with non-compliance.

Subclinical hypothyroidism

Automatic comments applied when TSH 4.2-10mU/l

Consistent with subclinical hypothyroidism provided patient is not acutely unwell and with no relevant history or causative medications. If pattern persists, and not checked previously, antithyroid peroxidase antibodies may be of value to assess risk of progression. Note: if patient on thyroxine, a TSH within the reference range indicates optimal replacement and good compliance, assuming dose stable for at least 8 weeks.

SCH0 Possible subclinical hypothyroidism. Suggest exclude concurrent illness or interfering drugs and repeat in 3-6 months, or sooner if clinical concern. SCH1 If TPO are negative then repeat TFT's every 3 years. However, if TPO positive there is a higher risk of developing overt hypothyroidism and yearly TFT's are indicated.

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SCH2	Subclinical hypothyroidism with negative TPO antibodies. Suggest repeat testing in 3 years.
SCH3	Subclinical hypothyroidism with positive TPO antibodies. Suggest repeat testing in 1 year.
SCH4	Current Royal College of Physician Guidelines recommend thyroxine replacement at TSH greater than or equal to 10mU/l, if the patient is pregnant, has a goitre or if TSH is rapidly increasing.
Hyperthyroidism	
ΤΟΧ	Hyperthyroid picture
TOX1	Hyperthyroid. Please repeat after 2-3 weeks to exclude transient thyroiditis and send TPO antibodies. If persistently elevated FT4/FT3 a routine endocrinology referral is indicated.
TOX2	TSH,0.02mU/L and FT4>50pmol/L or FT3>25pmol/L Thyrotoxicosis. Suggest FAX urgent referral to endocrinology on 0117 4149448. Repeat TFT's with TPO antibodies.
Taking thyroxine:	
TOR	TSH<0.1mU/L Results suggest over-replacement. A reduction in FT4 should be considered providing the patient has not had thyroid cancer and requires suppressive therapy.
TLO	TSH 0.1-0.4mU/L Results may indicate over-replacement. Suggest exclude concurrent illness and interfering drugs and repeat in 3 months.
TLR	TSH Persistent 0.1-0.4mU/L TSH ideally should be within the reference range on replacement (unless the patient has had thyroid cancer and requires suppressive therapy).
Subclinical hypert	hyroidism
TSH1	TSH<0.4mU/L and normal FT4 and FT3 May suggest subclinical hyperthyroidism although other causes include concurrent illness and interfering medications. Suggest repeat in 3-6 months or sooner if elderly, symptomatic or underlying vascular disease.
TSH2	Persistent TSH 0.1-0.4mU/L Marginally low TSH, which may fit with subclinical hyperthyroidism. Suggest repeat in 6-12 months.

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TSH3	Persistent TSH <0.1mU/L Subclinical hyperthyroidism. If other causes have been excluded such as
	concurrent illness or interfering medications then an endocrine referral is
	indicated as there is a risk of AF, osteoporosis and increased vascular risk.
TSH4	Recurrent TSH <0.1mU/L and previous advice to refer
	Previously documented subclinical hyperthyroidism. If the patient has been reviewed by endocrine and no further treatment was advised then the British Thyroid Association suggests monitoring every 6-12 months.
Thyroid cancer	
TC1	TSH suppression may be appropriate in patients with a previous history of thyroid cancer. Patients should be managed according to individual Oncology guidance.
TC2	Thyroxine treatment seems appropriate in a post thyroid cancer patient.
ТНҮХ	Please note that this sample has not been referred for thyroglobulin analysis. Analysis of thyroglobulin is required only in the monitoring of patients post-thyroidectomy for thyroid carcinoma. This sample will be stored for 2 weeks, please contact the duty biochemist should you wish to discuss (via 323 8383).

Pregnancy

TPŘ

In pregnancy the following trimester specific reference intervals are recommended.

	1 st Trimester	2 nd Trimester	3 rd Trimester
TSH mU/L	0.02-3.7	0.2-3.7	0.27-4.2
FT4 pmol/l	12.1-19.6	9.6-17.0	8.4-15.6

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