

best tests

MARCH 2010

INVESTIGATING THE GUT:

Faecal inflammatory markers

Pernicious anaemia

Helicobacter pylori

Coeliac disease

Steatorrhoea

Lactose intolerance

QUIZ FEEDBACK:

Troponin & Urea Testing



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CONTENTS



2 Investigating the Gut

Faecal inflammatory markers

Clinical assessment of intestinal inflammation can be problematic. Faecal calprotectin can be useful in differentiating between IBS and IBD, but it is not funded or widely available.

Pernicious anaemia

There are a number of causes of low vitamin B12 levels with pernicious anaemia being among the most important to identify. Parietal cell and intrinsic factor antibodies have an important role to play when investigating the possibility of pernicious anaemia.

Helicobacter pylori

The choice of test for initial testing for *H. pylori*, is determined by prevalence. In areas where the local prevalence is >30%, serology tests are best. In areas of low prevalence, stool antigen testing is the better option.

Coeliac disease

Tissue transglutaminase antibodies are the most useful preliminary step for testing people with a clinical picture suggestive of coeliac disease.

Steatorrhoea

In the past, the faecal fat test was sometimes used to aid the diagnosis of steatorrhoea. This practice is no longer recommended. There are other tests available that can provide more valuable information.

Lactose intolerance

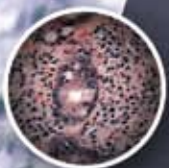
The role of laboratory tests in diagnosing lactose intolerance in primary care is limited. In most cases the diagnosis can be made on clinical grounds and by a trial of a lactose-free diet.



11 QUIZ FEEDBACK: Troponin & Urea Testing

INVESTIGATING THE

Gut



1

Inflammatory bowel disease – the role of faecal calprotectin

Clinical assessment of intestinal inflammation can be problematic. Clinicians often under or over-estimate the degree of inflammation present due to the subjective nature of many gastrointestinal symptoms.

When inflammatory bowel disease (IBD) is suspected clinically, laboratory testing can help rule out other causes of diarrhoea and abdominal pain. However, a definitive diagnosis of IBD is made histologically by bowel biopsy.

Faecal calprotectin

- Reasonably new biomarker, unfunded (NZ\$90 per assay)¹
- Usually a specialist request
- Can be useful in differentiating between irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD), in symptomatic patients with only slightly raised CRP
- Does not help determine the cause of inflammation¹
- A single faecal calprotectin of <60 µg/g is a good negative predictor for inflammatory change²

This test is currently not widely available. Most gastroenterologists will proceed directly to colonoscopy and biopsy if there are suggestive symptoms.

Antibody tests

Antibody tests are sometimes requested by specialists to help differentiate between ulcerative colitis and Crohn's disease. None of the antibody tests are specific or sensitive enough to be used to diagnose either condition but may provide some additional information. The current view is that they have little role in primary care.³

Tests for inflammatory bowel disease

Faecal calprotectin	✗	not widely available
pANCA, ASCA, Anti-CBir1, Anti-Omp C, Anti-I-2	✗	

References

1. Geary R, Barclay M, Florkowski C et al. Faecal calprotectin: the case for a novel non-invasive way of assessing intestinal inflammation. NZMJ May 2005.Vol 118;1214.
2. Dolwani S, Metzner M, Wassell J et al. Diagnostic accuracy of faecal calprotectin estimation in prediction of abnormal small bowel radiology. Aliment Pharmacol Ther 2004 Sep; 20(6):615-21.
3. Personal communication Assoc. Professor Alan Fraser, Mercy Specialist Centre, Auckland.

2

Pernicious anaemia

There are a number of causes of low vitamin B12 levels (see Table 1 below), but pernicious anaemia is among the most important to identify. Pernicious anaemia is rare in people under 30 years.

The diagnosis of pernicious anaemia identifies the need for lifelong vitamin B12 treatment and is associated with other autoimmune endocrinopathies, particularly thyroid disease and diabetes. There is also a small, increased incidence of stomach cancer.

Parietal cell and intrinsic factor antibody tests should be requested for a patient with low vitamin B12, and signs/symptoms consistent with pernicious anaemia. Ninety percent of people with pernicious anaemia will test positive for one or both of these tests.¹

Lab tests:^{1,3,4}

- Intrinsic factor antibodies:
 - Very specific and virtually diagnostic for pernicious anaemia but sensitivity is low (approximately 60%)
 - Absence does not rule out a diagnosis of pernicious anaemia
- Parietal cell antibodies:
 - High sensitivity (85–90%) meaning most patients with pernicious anaemia will have positive parietal cell antibodies, but low specificity, yielding higher number of false positives
 - Incidence of these antibodies in healthy individuals increases from 2.5% of those in their twenties, to 10% of those in their seventies. The test may be positive in 20–30% of first degree relatives of patients with pernicious anaemia as well as in patients with other autoimmune endocrine disorders.
- Schilling test:
 - Rarely used and difficult to perform (involves radio-labelled vitamin B12)
 - Has been superseded by antibody testing

Initial testing once low vitamin B12 levels detected

Intrinsic factor antibody	✓
Parietal cell antibody	✓
Schilling test	✗

Table 1: Possible causes of low vitamin B12:²

- Nutritional deficiency – the main dietary sources are meat and dairy products therefore elderly patients with “tea and toast” diets, chronic alcoholics and strict vegans are especially at risk
- Gastric causes e.g. pernicious anaemia, gastrectomy
- Intestinal causes e.g. ileal disease/resection
- Severe pancreatic insufficiency
- Drugs e.g. oral contraceptives, metformin, long term proton pump inhibitor therapy



Interpreting results

	Intrinsic factor antibody (IFA) ⊖ Negative	Intrinsic factor antibody (IFA) ⊕ Positive
Parietal cell antibody (PCA) ⊖ Negative	Pernicious anaemia unlikely	Immunological evidence of pernicious anaemia
Parietal cell antibody (PCA) ⊕ Positive	<ul style="list-style-type: none"> • Not diagnostic • PCA positive in 85–90% of patients with pernicious anaemia • Negative IFA does not exclude pernicious anaemia (only present in 50% or less) 	Immunological evidence of pernicious anaemia

References:

1. Diagnostic Medlab – A handbook for the interpretation of laboratory tests. 4th edition.
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3. Lahner E, Annibale B. Pernicious anemia: New insights from a gastroenterological point of view. World J Gastroenterol 2009;15(41): 5121-5128.
4. The Pernicious Anaemia Society. Intrinsic factor and parietal cell antibodies.
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3

Helicobacter pylori testing: Serology and stool antigen testing

Helicobacter pylori is a carcinogen

As knowledge of *Helicobacter pylori* (*H. pylori*) has grown over the last twenty years, the implications of infection have become more apparent. It is estimated that people who test positive for *H. pylori* have a 10–20% increased lifetime risk of developing peptic ulcer disease and a 1–2% increased risk of developing distal gastric cancer. As a result, *H. pylori* has been declared a Class 1 carcinogen by the World Health Organisation.¹

H. pylori infection is usually acquired in early childhood, and does not resolve spontaneously. There is a higher rate of infection associated with lower socioeconomic living conditions. As living conditions have improved in New Zealand, *H. pylori* infection rates have decreased. As a result *H. pylori* infection is more common in older people, as a result of acquisition in childhood.²

Prevalence of *H. pylori* in New Zealand

There is incomplete data on *H. pylori* infection rates throughout New Zealand, however it is known that rates are significantly higher in Māori and Pacific people compared to those of European descent.

The NZGG Dyspepsia Guidelines contains the following statements about *H. pylori* infection rates:³

- Rates in the South Island are well below 30%
- Rates tend to be >30% in adult Māori and Pacific people, and people with lower socio-economic status
- Rates in adults living in Auckland have been generally found to be greater than 30%

Initial testing for *H. pylori* (first time ever) in the presence of dyspepsia

Serology (for antibodies)	✓ If local prevalence > 30%
Stool antigen	✓ If local prevalence < 30%

Who should be tested for *H. pylori*?

1. Patients from a high prevalence (>30%) group presenting with dyspepsia without “alarm symptoms” (weight loss, dysphagia, signs of anaemia, blood loss). Identifying and treating this patient group may reduce the requirement for endoscopy. A useful rule-of-thumb is to ask where they were born. A place of birth and early life in a high prevalence area is predictive of infection in adulthood.
2. Patients with a past or present history of peptic ulcer disease and no record of treatment of *H. pylori*.
3. Patients with a family history of gastric cancer.

Testing for *H. pylori*⁴

- Serology tests are appropriate where the prevalence of *H. pylori* infection is greater than 30%.
 - A negative *H. pylori* serology test confirms the absence of infection in the majority of cases
 - Patients can test positive for months to years after eradication, making it difficult to discern if it is a current or past infection. For this reason serology testing is unsuitable if a patient represents at a later date with symptoms suggestive of subsequent *H. pylori* infection.
- Stool antigen tests are appropriate where the prevalence of *H. pylori* infection is less than 30%.
 - A positive *H. pylori* stool antigen test is highly predictive of the presence of *H. pylori* infection
 - *H. pylori* disappears quite quickly from the stool after eradication, therefore positive results indicate persisting active infection
 - To avoid false negative results, patients should be off antibiotics for at least four weeks and off PPIs and bismuth for at least two weeks. H2 receptor antagonists and antacid preparations (e.g. mylanta) are allowed.⁵

Confirmation of *H. pylori* eradication

The majority of patients do not require post-eradication testing for *H. pylori*. The eradication rate is over 85% with the “triple therapy” combination of omeprazole, amoxicillin and clarithromycin.

Confirmation of eradication of *H. pylori* is only required in those with a peptic ulcer complication, important comorbidity factors, symptom recurrence or residence in isolated areas.³

In these cases, *H. pylori* stool antigen testing may be used for confirmation of eradication at least 4 weeks after stopping treatment. For people taking PPIs, perform at least two weeks after cessation of the PPI.

References:

1. Centers for Disease Control and Prevention (CDC) – *Helicobacter pylori*.
Available from: <http://www.cdc.gov/ncidod/aip/research/hp.html>
2. Fraser, A. 2004. *Helicobacter pylori*: A historical perspective 1983–2003. N. Z. Med. J. 117:U896.
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4. Health Protection agency (HPA) 2009. *Helicobacter pylori*.
http://www.hpa.org.uk/webw/HPAweb&HPAwebStandard/HPAweb_C/1195733787873?p=1191942127576
5. Diagnostic Medlab – A handbook for the interpretation of laboratory tests. 4th edition 2008.



4

Coeliac disease

Prevalence in New Zealand

Coeliac disease has a prevalence of approximately 1% in the general New Zealand population. Prevalence in those with a first-degree relative with coeliac disease is about 1 in 10.

Who to test

Testing is recommended for all symptomatic children and adults as well as asymptomatic people at increased risk.

People at increased risk include:¹

- Siblings of any index case
- Those with type I diabetes and other systemic autoimmune disorders
- Patients with IgA deficiency
- Children with Down syndrome

Antibody tests are less reliable for excluding coeliac disease in very young children. Where there is strong clinical suspicion of coeliac disease and negative serology it is recommended that the child be referred for specialist opinion.

In New Zealand, screening of asymptomatic people is not recommended.

What tests to use

Serological tests are the most useful preliminary step for testing symptomatic people and those with an increased risk of coeliac disease.^{1,2} People must have consumed adequate amounts of gluten (equivalent to four slices of bread daily) for 4–6 weeks prior to testing.³ Negative

Initial testing for coeliac disease

Tissue transglutaminase antibodies	✓
Endomysial antibodies	✗
Gliadin antibodies	✗
HLA typing (HLA-DQ2 and DQ8)	✗

results can not exclude coeliac disease if the patient has had a significantly reduced gluten intake.

IgA tissue transglutaminase antibodies (TTG)

- The preferred initial test for detecting coeliac disease
- The TTG test detects IgA antibodies; consequently, tests can be negative in patients with coeliac disease with a coexisting IgA deficiency (more common in people with coeliac disease). To detect IgA deficiency, laboratories routinely test for total serum IgA whenever a TTG test is requested. If an IgA deficiency is detected, the IgG TTG test is performed.

Endomysial antibodies

- May be useful to confirm an equivocal TTG or for monitoring compliance
- When TTG antibodies are negative, the additional value of performing endomysial antibodies is low

Gliadin antibodies

- Unnecessary for the diagnosis of either coeliac disease or "gluten sensitivity"
- Should no longer be requested

HLA typing (HLA-DQ2 and DQ8)

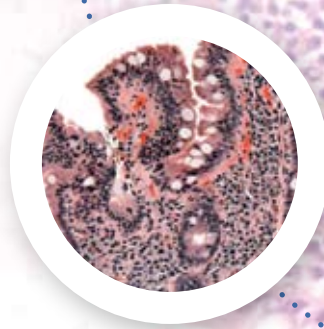
- Virtually all patients with coeliac disease are either HLA DQ2 or DQ8 positive, compared with 20–30% of the general population. In the rare circumstances that serological testing is equivocal, the absence of these HLA haplotypes can help exclude the diagnosis of coeliac disease

Who requires biopsy?

Biopsy is recommended to confirm the diagnosis of coeliac disease. Serological testing may not correlate with mucosal damage and it is important to determine the degree of inflammation and villous atrophy in the gut as well as excluding other small bowel disease. A biopsy test may be appropriate if there is strong clinical suspicion, even if the TTG antibody test is negative. Serological testing may not be accurate in children under 5 years of age.

References:

1. Diagnostic Medlab – A handbook for the interpretation of laboratory tests. 4th edition 2008.
2. BPAC. Best Practice Journal. Issue 9. Coeliac Disease. Available from www.bpac.org.nz keyword "coeliac disease"
3. BPAC. Best Tests Best Tests March 2007. Testing for Coeliac disease. Available from www.bpac.org.nz keyword "coeliac disease"



5

Steatorrhoea

Steatorrhoea is the result of fat malabsorption, with diagnosis usually ascertained from patient history. The hallmark of steatorrhoea is the passage of pale, bulky, and malodorous stools, which often float and are difficult to flush.

In healthy individuals, less than 6 g of fat is excreted daily in the stools. This amount remains stable irrespective of intake.

The most important first step in deciding whether to investigate possible steatorrhoea is to determine if the overall clinical picture suggests fat malabsorption. Fat malabsorption can have many causes including pancreatic insufficiency, diarrhoeal illnesses and coeliac disease. A history of oil drops that separate from the main stool mass, and become whitish and firm after cooling (non-hydrolysed triglycerides), strongly points toward pancreatic insufficiency as the cause.

Faecal elastase test¹

- Most sensitive test for pancreatic insufficiency
- Elastase levels correlate with other pancreatic enzymes such as lipase, amylase and trypsin

Faecal fat test^{2,3}

- Not recommended
- Low sensitivity for pancreatic insufficiency
- Very unpleasant test

Fat soluble vitamins³

- In the first instance, the measurement of fat soluble vitamins is not indicated when investigating steatorrhoea, but may be recommended by a specialist
- Ongoing steatorrhoea can be associated with deficiency of fat soluble vitamins (A, D, E, K), as they are “trapped” by the unabsorbed fats

Investigating steatorrhoea

Diagnosis made clinically	✓
Faecal elastase	✓
Faecal fat	✗
Fat soluble vitamins	✗

Further assessment at a specialist level will often be directed towards making a specific diagnosis rather than proving fat malabsorption.

References:

1. Loser C, Mollgaard A, Folsch UR. Gut 1996;39:580-586
2. RCPA The Royal College of Pathologists of Australasia version 5
<http://www.rcpamannual.edu.au/default.asp>
3. Diagnostic Medlab – A handbook for the interpretation of laboratory tests. 4th edition

6

Lactose intolerance

The role of laboratory tests in diagnosing lactose intolerance is limited. The diagnosis can usually be made on clinical grounds and by trial of a lactose-free diet (see over page).

A laboratory diagnosis is not generally helpful as results do not always correlate with clinical symptoms. Approximately 10–30% of proven lactose intolerant subjects (i.e. people who lack normal lactase levels) report no symptoms with lactose challenges, while over half of patients who report symptoms of lactose intolerance, actually have normal intestinal lactase levels.¹

In addition, the amount of lactose ingested and the combination of other foods influences whether symptoms may occur, e.g. ice cream is generally better tolerated because the increased fat content delays gastric emptying.²

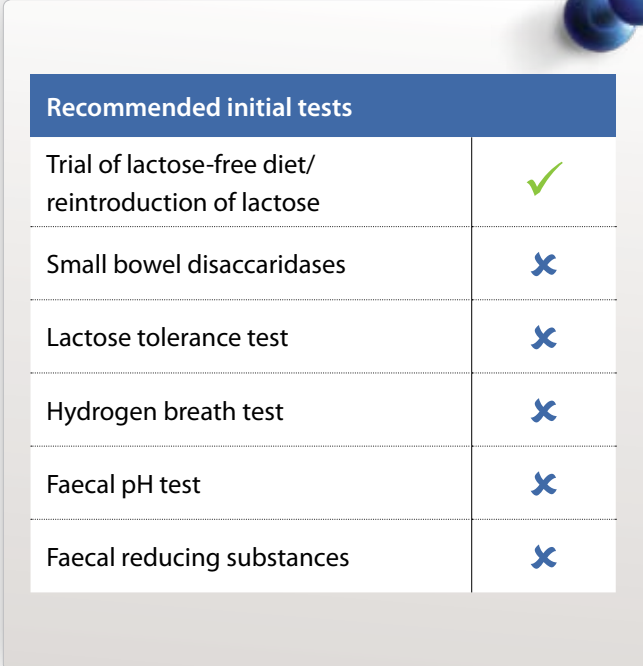
A number of laboratory investigations are available^{1,3} but these are most often reserved for use in secondary care.

Small bowel disaccharidase testing

- Remains a very good test for lactose intolerance, but is an invasive test that requires small bowel biopsy
- Cannot differentiate between primary and secondary lactase deficiency

Lactose tolerance test

- Rarely performed due to poor sensitivity (about 75%)
- Causes unpleasant symptoms such as diarrhoea and abdominal pain



Recommended initial tests	
Trial of lactose-free diet/ reintroduction of lactose	✓
Small bowel disaccharidases	✗
Lactose tolerance test	✗
Hydrogen breath test	✗
Faecal pH test	✗
Faecal reducing substances	✗

Hydrogen breath test

- An alternative to the lactose tolerance test where breath samples rather than blood samples are analysed
- Not widely available
- Preferable to lactose tolerance tests in children

Faecal pH test

- Limited value and no longer recommended

Faecal reducing substances

- Unreliable and not recommended
- False positives can occur following antibiotic therapy or in any condition in which the gut transit time is more rapid than usual. False negatives can also occur e.g. if the offending sugar has not been ingested recently.³

Lactose intolerance is usually diagnosed by dietary challenge

Step 1: Rule out other causes

Step 2: Remove lactose from the diet for two weeks, then reintroduce

Step 3: Referral if symptoms warrant and dietary challenge inconclusive

Gastrointestinal symptoms of lactose intolerance usually occur between 30 minutes and two hours after the ingestion of lactose. The symptoms of lactose intolerance are generally non-specific, highly individual and mild. Vomiting is rare. Severe gastrointestinal symptoms are an indication to investigate other causes.

When lactose intolerance is suspected, the American Academy of Paediatrics recommends that a lactose-free diet is trialled for two weeks. During this trial it is important that all sources of lactose are eliminated – food labelling should be closely studied. Lactose intolerance can be diagnosed if symptoms resolve over the two week period and then return with the subsequent reintroduction of lactose containing foods.² This diagnosis can be made by a GP and further investigation is rarely needed.³

If the dietary challenge is inconclusive, then a referral may be required.



References

1. DML – Diagnostic Medlab: A handbook for the interpretation of laboratory tests, 4th edition, 2008.
2. Swagerty D, Walling A, Klein R. Lactose intolerance. Am Fam Physician 2002;65(9):1845-50.
3. RCPA The Royal College of Pathologists of Australasia version 5. Available from: <http://www.rcpamanual.edu.au/default.asp>
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5. Bhatnager S, Aggarwal R. Lactose intolerance. BMJ 2007;334(7608):1331-2.

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QUIZ FEEDBACK

Troponin & Urea Testing



Introduction

This quiz feedback provides an opportunity to revisit the last “Best Tests” document and accompanying quiz which focused on the role of troponin testing in primary care, and provided an update on whether we should still be measuring urea.

This feedback provides an overview of the key messages of the articles, in particular the benefits and pitfalls of the tests, and the best use of the tests. The most important message, of the troponin article is that if it is suspected that a patient has had an MI, they should be referred

immediately to hospital. Admission should not be delayed while waiting for a troponin result.

The urea test has fallen from favour over the last decade, as eGFR and creatinine have become the better choice when assessing renal failure.

All general practitioners who responded to this quiz, will receive personalised online feedback and CME points. The quiz can still be completed online. Currently, there are over 30 interactive quizzes available which provide an ongoing opportunity for the accumulation of points. Visit www.bpac.org.nz

1. In which of the following situations would testing troponin levels be useful?		
	Your peers	GP panel
<input type="checkbox"/> To confirm a suspicious ECG	27%	✗
<input type="checkbox"/> Use to rule out MI, in someone presenting with acute chest pain	13%	✗
<input type="checkbox"/> Screening for people with high cardiovascular risk	0%	✗
<input type="checkbox"/> Delayed presentation of suspected MI	97%	✓

Comment:

Almost all of GPs correctly responded that one of the key situations in which troponin is useful in primary care, is for patients presenting 24–72 hours after a single episode of chest pain e.g. the “Monday morning” consultation. Measurement of troponin and ECG will establish whether or not the chest pain was due to a MI. If there has been a MI, troponin is likely to remain elevated for up to 10 days. A positive troponin result is indication for immediate referral. The use of troponin to confirm a suspicious ECG or to rule out an MI in someone presenting with acute chest pain is not recommended. These patients should be referred immediately to secondary care.

2. For a patient presenting a few days after a single episode of chest pain, how can troponin be helpful?		
	Your peers	GP panel
<input type="checkbox"/> To establish if chest pain was due to MI, or other causes	94%	✓
<input type="checkbox"/> Help determine risk of future events	3%	✗
<input type="checkbox"/> For someone with atypical symptoms	81%	✓
<input type="checkbox"/> If no ECG changes	79%	✓

Comment:

The majority of GPs correctly identified that troponin is most helpful when there is delayed presentation, absence of ECG changes or the presence of atypical symptoms. In these situations, troponin can provide reassurance that no MI has occurred, or provide sufficient evidence to refer the patient to hospital.



3. Which of the following are true about the use of troponin as a screening test?		
	Your peers	GP panel
<input type="checkbox"/> Can provide additional information than just lipids and glucose for CVD risk assessment	1%	✗
<input type="checkbox"/> Troponin is being requested more frequently as a screening test	24%	✓
<input type="checkbox"/> Troponin testing is only indicated if there is some suspicion of MI	69%	✓
<input type="checkbox"/> There is no rationale for using troponin as a screening test	90%	✓

Comment:

There has been some concern expressed by Cardiologists that troponin may be being used for CVD risk assessments in primary care. However the responses to this question indicate general practitioners are well aware troponin does not provide any additional information for CVD risk assessment and that there is no indication for troponin testing in asymptomatic patients.

4. A negative troponin can only be used as a rule out test if it is:		
	Your peers	GP panel
<input type="checkbox"/> Laboratory measured	59%	✓
<input type="checkbox"/> From a point-of-care analyser	2%	✗
<input type="checkbox"/> Negative 4 hours post onset symptoms	3%	✗
<input type="checkbox"/> Negative 10 hours post onset symptoms	96%	✓

Comment:

Most respondents correctly identified that a negative troponin result is an appropriate “rule out” test for MI. But fewer recognised that a laboratory method must be used if the initial symptoms occurred more than 10 hours ago. Most point-of-care methods for troponin testing do not have sufficient sensitivity to “rule out” acute MI.

5. What is the appropriate action following a positive troponin test?		
	Your peers	GP panel
<input type="checkbox"/> Immediate referral to secondary care	99%	✓
<input type="checkbox"/> Confirm with ECG	3%	✗
<input type="checkbox"/> Perform repeat troponin test in 4 hours	2%	✗
<input type="checkbox"/> A positive troponin from a point-of-care analyser, should be confirmed with a laboratory tested specimen	11%	✗

Comment (Question 5):

A positive result for troponin, whether from the laboratory or from a point-of-care analyser, is significant and the patient should be referred immediately to secondary care. This should not be delayed by repeating the test (either by point-of-care or in the laboratory) or by confirming with ECG.

6. Which of the following are true about the interpretation of a troponin test?		
	Your peers	GP panel
<input type="checkbox"/> A negative troponin at the time of presentation is a useful rule out test	5%	✗
<input type="checkbox"/> It takes 3–4 hours for troponin levels to begin to rise	86%	✓
<input type="checkbox"/> Troponin is useful for a delayed presentation, since troponins can remain elevated for up to 2 weeks	81%	✓
<input type="checkbox"/> In patient with no ST changes, but elevated troponin, it is worth considering other causes	48%	✗

Comment:

A patient with no ST changes but elevated troponin should be assumed to have had a MI and referred immediately to secondary care. There may be rare circumstances in which the troponin is elevated for a reason other than MI but it is not appropriate to delay referral.

7. Which of the following are true about the use of urea as a 'renal function test'?		
	Your peers	GP panel
<input type="checkbox"/> Has been superseded by eGFR and creatinine	96%	✓
<input type="checkbox"/> Is generally an insensitive marker of renal failure	75%	✓
<input type="checkbox"/> Urea levels can be altered by a number of non-renal causes	95%	✓
<input type="checkbox"/> Urea:creatinine ratio remains a useful means of distinguishing between pre-renal and renal causes of renal failure	4%	✗

Comment:

Urea is generally an insensitive marker of renal failure as levels can vary for a number of reasons such as high/low protein diet, tissue breakdown, GI haemorrhage and liver disease. Note: using the urea:creatinine ratio to distinguish between pre-renal and renal causes is now considered unreliable; eGFR and creatinine are preferred instead.

8. Indicate the situations where urea may have a role:		Your peers	GP panel
<input type="checkbox"/>	Management of a patient on dialysis	94%	✓
<input type="checkbox"/>	Occasionally for the assessment of dehydration in the frail elderly	89%	✓
<input type="checkbox"/>	Monitoring changes in renal function for people on ACE-inhibitors or diuretics	1%	✗
<input type="checkbox"/>	For areas of New Zealand that do not calculate eGFR	6%	✗

Comment:

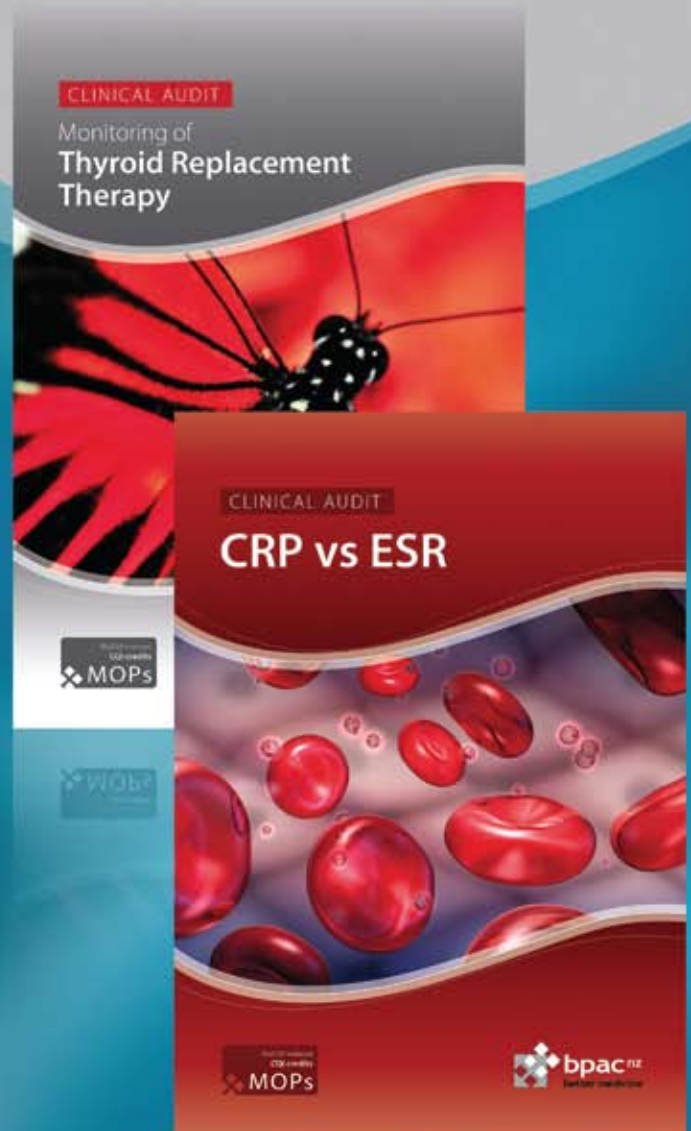
Urea has a limited role in primary care, although it may be useful in some circumstances for assessing hydration status in frail elderly.

eGFR is now routinely reported by all laboratories throughout New Zealand, when creatinine is requested.



Please note: We no longer send out printed personalised quiz feedback. Personalised feedback is now available from www.bpac.org.nz. GPs who completed this quiz will receive an email with access instructions.

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