

# QML PATHOLOGY

newsletter August 07

## >> Basal Cell Carcinoma: The Most Common Malignant Neoplasm, Important Histologic Subtypes and Management Guidelines

**Dr John Pauli, Pathologist, Histology**

Basal cell carcinoma (BCC) is the most common malignancy in Caucasian people. In Australia, the incidence is up to 2% per year, which is about ten times higher than in the USA. Although very rarely metastatic with less than 300 well-documented cases reported in the literature, BCC produces significant morbidity<sup>1-4</sup>.

Treatment options for BCC are numerous and many guidelines for management of BCC have been published, including those of the NHMRC (2003)<sup>5</sup> and British Association of Dermatologists (1999)<sup>6</sup>. What follows is an outline of the epidemiology of BCC and important histologic subtypes, together with a brief review of the literature providing suggested recommendations for treatment of primary and recurrent BCC. A tabulated summary of the NHMRC 2003 guidelines is also provided.

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**QML Pathology.**



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### Risk Factors and Significance of BCC

Risk factors for development of BCC include factors common to other cutaneous malignancies linked to ultraviolet (UV) light exposure, exposure to arsenic and immunosuppressive therapy. Several genetic conditions are also associated with BCC as part of their clinical expression. These include albinism, xeroderma pigmentosa and Gorlin's syndrome (naevoid basal cell carcinoma syndrome). Several genetic polymorphisms have been identified that are associated with increased susceptibility to BCC, including a cytochrome P-450 genotype associated with multiple often clustering lesions, and BCC occurring on the trunk (often in younger age group) associated with alterations in modifier genes including that of the Vitamin D receptor and tumour necrosis factor<sup>7,8</sup>.

BCC is a marker for future BCC and for squamous cell carcinoma (SCC), and malignant melanoma. The 3-year cumulative risk for developing a further BCC after initial diagnosis is between 33 and 77% with truncal tumours at increased risk. The risk of developing SCC is 6% at 3 years after initial BCC, and the risk of developing malignant melanoma after diagnosis of BCC is increased with risk ratios in the literature ranging between 2.2 and 6.0<sup>9,10</sup>.

### Clinical Features

The diverse clinical appearances of BCC would be well appreciated by clinicians in Australia with approximately 80% occurring on the head and neck, and the remainder predominantly on the trunk and legs, more commonly seen in women. The classic clinical variant is the ulcerated BCC 'rodent ulcer'. Nodulocystic BCC (solitary, shiny, red nodule with large telangiectatic vessels) occurs most commonly on the face, while superficial BCC is most commonly found on the trunk. Approximately 2-5% of BCC are pigmented (more common in black races and Japanese) and these may mimic malignant melanoma. The clinically aggressive subtypes of BCC can present late, and be difficult to diagnose, as they often do not have characteristic clinical appearances and often have ill-defined margins. BCC can also complicate other lesions, which results in a more varied clinical appearance. These include chronic ulcers, scars, skin graft sites, dilated pore of Winer, port wine stain, arteriovenous malformations, rhinophyma, pilonidal sinus and lupus vulgaris.

BCC can be extensively locally destructive but they have a very limited metastatic potential, with metastatic rates in the literature ranging between 1 in 1000 to 1 in 35000<sup>2</sup>. The metastatic rate appears significantly greater in BCC of the scrotum. Other features associated with metastatic BCC include large size, ulceration, deep infiltration and recurrent tumours.

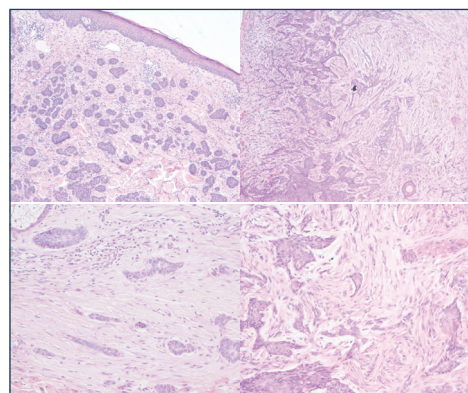
### Histologic Subtypes of BCC

The following histologic subtypes of BCC are of no special clinicopathologic significance. The list highlights the very diverse range of appearances that BCC can assume:

- Solid/nodular (approximately 70% of cases), cystic, adenoid, pigmented, keratotic, infundibulocystic, pleomorphic/giant cell, fibroepithelioma (of Pinkus)
- Miscellaneous variants including BCC with clear cell, sebaceous, pilar, tricholemmal, signet-ring, Schwannian, adamantinoid, myoepithelial, granular cell or neuroendocrine differentiation.

### Aggressive Histologic Types of BCC (Variants with Clinicopathologic Significance):

- Multifocal superficial
- Micronodular (fig 1a)
- Infiltrative (fig 1b)
- Sclerosing (morphoeic) (fig 1c & 1d)
- Metatypical (basosquamous).



**Figure 1**

1a (top-left) micronodular BCC

1b (bottom-left) infiltrative BCC

1c & 1d (right) sclerosing BCC, note prominent desmoplastic/sclerotic stroma.

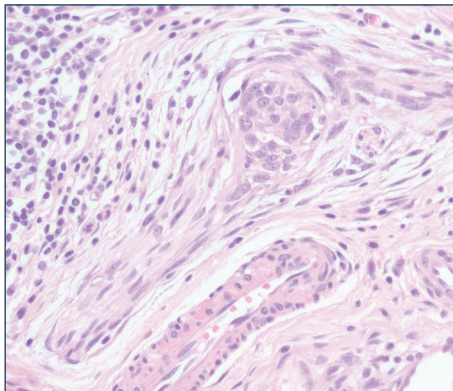
Multifocal superficial BCC is often poorly-defined clinically and as the name implies, multifocal in nature, which makes complete excision and pathologic assessment of margins difficult. As the lesion is discontinuous a nest of BCC may be clear of the excision margin in the slide being examined but the possibility of further nests of BCC in the adjacent skin (not clinically recognised) beyond the excision exists. Due to the high-risk of residual BCC, multifocal superficial BCC is considered one of the high-risk BCC subtypes.

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Infiltrative, sclerosing and micronodular BCC often diffusely infiltrate the dermis and extend into the subcutis making complete excision difficult, and as a result are associated with a higher local recurrence rate than the other subtypes. Perineural infiltration is also more common in these BCC subtypes and this is an independent prognosticator for local recurrence (fig 2). Infiltrative and sclerosing BCC can also be problematic because they may mimic other lesions including microcystic adnexal carcinoma, desmoplastic SCC and desmoplastic trichoepithelioma.

Metatypical BCC shows hybrid basal cell and squamous differentiation, and may in fact represent a collision tumour between BCC and SCC that would account for its higher metastatic potential when compared with all other BCC subtypes.



**Figure 2**

BCC within the perineural space of a mid-dermal nerve deep to the remainder of the BCC.

### Treatment Modalities

The destructive methods of treatment (cryotherapy, curettage, diathermy/cautery, topical agents and photodynamic therapy) are best suited only to 'low-risk' BCC i.e. small (<1.0cm), well-defined, primary (not recurrent), not occurring in high-risk sites such as the nose, nasolabial folds, around the eyes, of non-aggressive histologic type BCCs.

The topical immunomodulatory agent imiquimod and photodynamic therapy (PDT) using topical application of aminolaevulinic acid derivatives, which are absorbed by the tumour rendering it subject to photo-destruction by exposure to light at 620-670nm wavelength, offer cure rates comparable to excision in appropriately selected BCC. Imiquimod is PBS listed and application 5 days per week for 6 six weeks achieves approximately 90% clinical clearance of superficial BCC and 70% clinical clearance of small nodular BCC. PDT has the benefit of once-off treatment with curettage used primarily to de-bulk the tumour, followed by application of the photosensitising agent and then 3-6 hours later application of

a red light. PDT is approved in Australia for primary treatment of superficial and/or nodular BCC where surgery is considered inappropriate. It achieves approximately 70-80% histologic clearance of superficial and thin primary nodular BCC<sup>11</sup>.

Radiotherapy has a role in primary therapy for BCC, particularly in extensive lesions where surgical excision is not possible because of tumour site or patient morbidity. Radiotherapy also has a role in treatment of recurrent BCC and as an adjuvant in cases with perineural invasion not amenable to surgical excision. Radiotherapy has the disadvantage of chronic radiation changes that may mask recurrence and make treatment of recurrences difficult. As a result radiotherapy should be avoided in younger patients (<50 years of age) if possible.

Surgical excision remains the primary treatment modality for BCC. The main advantage of surgical excision is that histologic margins can be examined for tumour clearance. Mohs' micrographic surgery offers high cure rates for BCC at high-risk sites (nose, nasolabial folds, around eyes), of aggressive histologic type, for tumours with perineural spread and recurrent tumours. It is labour intensive and expensive but invaluable in these situations.

Surgical excision of primary (previously untreated) BCC is very effective. It is estimated that for BCC less than 20mm in maximum dimension, 3mm clinically assessed margin clearance will result in cure in 85% of cases. This increases to 95% cure with 4mm margin clearance. Large(>20mm) and aggressive histologic type BCC require wider surgical excision with 66% cure at 3mm margin clearance, 82% cure at 5mm margin clearance and >95% cure with >13mm margin clearance<sup>12,13</sup>.

In practice, the histologic clearance is often much less than the clinically assessed margin clearance. In this situation what is the risk of recurrence? Table 1 below, adapted from Dixon et al. 1993 shows how margin clearance and histologic subtype alter the recurrence rate of BCC after primary excision.

**Table 1. Recurrence rate (%) for BCC treated by surgical excision varies with increasing histologic margin clearance and with histologic type<sup>14</sup>.**

Histologic margin/BCC type	<0.38mm	0.38-0.75mm	>0.75mm
Solid/nodular	41%	11%	4%
Superficial multifocal, infiltrative, sclerosing	82%	44%	22%

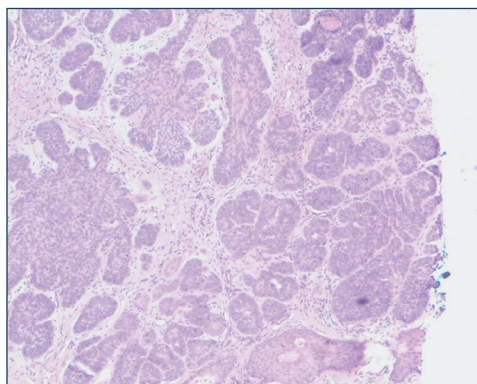
Dixon et al. concluded that a minimum histologic clearance of 0.75mm for solid BCC limited recurrences to 4% of cases, but that for aggressive histologic types a wider histologic clearance is required.

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Another commonly encountered scenario is selecting the appropriate treatment for recurrent (previously treated) BCC. Silverman et al. 1991 examined over 5000 BCC treated between 1955 and 1982 and calculated overall 5-year recurrence rates of 17% for BCC treated by destructive methods (electrodissection and curettage) and 6.8% for those treated by surgical excision<sup>14</sup>. Lesions of the central face, lesions >20mm and those of aggressive histologic type are overrepresented among recurrent BCC. Results of available published series reveal that re-recurrence rates (recurrence after treatment of already recurrent BCC) are higher than these values suggesting that considerably wider surgical margins are required to achieve a cure. As a result, peripheral excision margins for recurrent BCC of 5-10mm have been suggested<sup>16</sup>. Frozen section margin control or Moh's micrographic surgery is particularly useful in management of recurrent BCC.

Another common scenario is what to do in the case of incompletely excised BCC (fig 3). Some evidence suggests that total removal of some BCC may not be necessary to achieve cure, with up to 65% of incompletely excised BCC showing no evidence of recurrence<sup>16</sup>. This may be explained by the fact that only just over half of 78 re-excision specimens for BCC reported with positive margins on the initial excision contained residual BCC. This figure correlates well with prospective studies showing 41-58% of incompletely excised BCC recurred during a two-year follow-up period<sup>17, 18</sup>. The recurrence risk also appears related to which surgical margins are involved. The risk of recurrence is lower if only lateral margin involvement is present, intermediate rates of recurrence occur if there is deep margin involvement and the highest rates of recurrence are encountered with both lateral and deep margin involvement.



**Figure 3. H & E stained photomicrograph showing micronodular BCC present at the green-inked transverse margin of excision at the level of the mid-dermis (right).**

Despite this data, an expectant policy of management would not generally be considered appropriate in the primary care setting. The NHMRC 2003 guidelines for treatment and management of non-melanoma skin cancer in Australia state that histologic confirmation of complete removal of the tumour is important. The Medical Board of Queensland (MBQ) has also recently stated that it is vital that excision margins of a lesion are histologically clear of pathology and that this is adequately documented. The MBQ Bulletin 2007 goes on to say that the Board will continue to look unfavourably on registrants who do not follow-up inadequate excision margins with re-excision themselves or referral for further excision by a surgical colleague. Because of the NHMRC and MBQ statements, expectant management of residual BCC should probably be reserved for patients under specialist care and follow-up.

### Summary of NHMRC 2003 Treatment Recommendations for BCC

#### Definitions:

- 1. Clinically favourable:** Absence of any of the following features; >2cm, recurrent, incompletely excised, ill-defined margin, aggressive histologic type, perineural invasion, high-risk site
- 2. High-risk sites:** Nose, eyelids, nasolabial grooves, pre and post auricular
- 3. Poor prognosis BCC:** T4 primary, >2cm recurrent lesions, multiple recurrences, aggressive histologic type, perineural invasion, nodal metastases.

#### Follow-up

Evidence suggests that most recurrences will occur within 5 years. Some authors recommend follow-up for life for patients with multiple BCC or high-risk tumours such as truncal BCC. The increased risk of other skin malignancy including SCC and malignant melanoma in patients with BCC is probably of more significance in follow-up.



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	Primary BCC	Persistent/Recurrent BCC
<b>Cryotherapy</b>	<ol style="list-style-type: none"> <li>1. Histologic confirmation of diagnosis and analysis for clinically unfavourable features strongly recommended.</li> <li>2. Not recommended for high-risk sites, ill-defined lesions, infiltrative or sclerosing BCC.</li> <li>3. 3-5mm clinical margins recommended.</li> <li>4. Long-term follow-up for recurrence essential.</li> </ol>	Contraindicated
<b>Curettage/diathermy</b>	<ol style="list-style-type: none"> <li>1. Effective for small (&lt;1cm) BCC.</li> <li>2. Useful on legs as alternative to grafting.</li> <li>3. Not appropriate on very thin skin.</li> <li>4. Relatively contraindicated for high-risk sites, especially for lesions &gt;5mm.</li> <li>5. Contraindicated for infiltrative and sclerosing BCC.</li> </ol>	Relatively contraindicated
<b>Topical agents</b>	Consider for superficial and some thin nodular BCC	Contraindicated
<b>Photodynamic therapy</b>	<ol style="list-style-type: none"> <li>1. Consider in patients with bleeding disorders, pacemakers, multiple lesions.</li> <li>2. Suitable for superficial BCC and nodular BCC.</li> </ol>	Contraindicated
<b>Simple surgical excision</b>	<ol style="list-style-type: none"> <li>1. Remains the treatment of choice.</li> <li>2. Excise clinically favourable lesions with minimum 3mm clinical margin.</li> <li>3. Confirm histologic margins clear.</li> </ol>	<ol style="list-style-type: none"> <li>1. Re-excision to achieve histologic margin clearance mandatory (see MBQ Bulletin 2007).</li> <li>2. Consider XRT if re-excision not possible.</li> <li>3. For previously treated BCC resect scar, macroscopic tumour and all adjacent previously treated skin.</li> </ol>
<b>Moh's micrographic surgery or excision with frozen section margin assessment</b>	Time consuming and costly. Limit to situations where risk of persistent BCC is high.	First line treatment for persistent or recurrent BCC where wide re-excision is difficult or clinically unfavourable features present.
<b>Specialist referral</b>	<ol style="list-style-type: none"> <li>1. BCC with perineural spread.</li> <li>2. Consider referral for clinically unfavourable BCC.</li> <li>3. Cases with palpable regional lymph nodes.</li> </ol>	Consider specialist referral for all recurrent BCC especially those at high-risk sites.
<b>Radiotherapy (RT)</b>	<ol style="list-style-type: none"> <li>1. Histologic confirmation required prior to RT.</li> <li>2. Consider for stage T4 lesion.</li> <li>3. Avoid RT in younger patients if possible.</li> </ol>	<ol style="list-style-type: none"> <li>1. Consider for persistent and recurrent BCC not amenable to re-excision.</li> <li>2. Useful for perineural invasion not amenable to surgical excision.</li> <li>3. Consider adjunctive XRT after salvage re-excision for poor prognosis BCC.</li> </ol>

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# clinical data Aug 07

## Infectious Diseases Report - Geographic Distribution - July 2007

SEROLOGY	Regions (as per key below)															Total			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Jul	Jun	May	Apr
Adenovirus (not typed)	2	4	2				7		1		3	1				20	10	12	7
Adenovirus (typing pending)		1					1		2	1	2		1			8	5	5	2
Barmah Forest virus	3	1	1	2			1		9	5	3	10	1	2	4	42	45	58	69
Bordetella pertussis	2	6	3				6		11	3	17	7	3			58	39	51	32
Brucella species										1	1			1		3	1	0	1
Campylobacter jejuni																0	0	0	0
Chlamydia pneumoniae																0	0	0	0
Chlamydia trachomatis, not typed	57	60	21	19	3		73		22	25	99	51	4	16	12	462	442	525	423
Coxiella burnetii			4						1	2	3	2	4			16	12	10	7
Cryptococcus species							1					1				2	0	3	1
Cytomegalovirus (CMV)	3	7	2	2			4		4		9	4	1	2		38	48	39	23
Entamoeba histolytica																0	0	0	1
Enterovirus - not typed		1													1	2	4	6	8
Epstein-Barr virus (EBV)	6	17	4	1	1		23		13	4	17	8	4	4	2	104	117	141	135
Flavivirus unspecified	4	3							1	1		1				10	11	19	18
Hepatitis A virus			5													5	3	1	2
Hepatitis B virus	9	2	3	3			10		3	1	43	4		1	1	80	69	83	67
Hepatitis C virus	10	52	16	2	2		30		10	3	40	26	3	7	3	204	214	274	205
Hepatitis D virus																0	0	0	1
Hepatitis E virus																0	0	0	0
Herpes simplex Type 1	18	32	9	3	1	2	55		35	8	52	33	4	17	3	272	255	215	197
Herpes simplex Type 2	15	30	8	6	1	1	29		18	4	39	21	1	2	2	177	161	190	142
Herpes simplex virus - not typed	1	16	2	2	1		8	1	9	2	12	7		2	4	67	51	54	46
HIV-1							1				1	1	1			4	5	9	4
HTLV-1																0	0	0	0
Influenza A virus	2	15	12				123		35	9	78	12	3	3	1	293	22	7	7
Influenza B virus											1					1	0	0	2
Legionella species																0	1	0	0
Leptospira species	1									1						2	4	12	2
Measles virus		1	1													2	2	0	1
Mumps virus									1							1	2	0	2
Mycoplasma pneumoniae	2	11	5	2			14	4	9	5	10	12	2		4	80	71	77	79
Neisseria gonorrhoeae	6	1		1			5			1	11	1		2		28	39	44	25
Parainfluenza virus Type 1											1					1	2	0	0
Parainfluenza virus Type 2		1														1	2	4	3
Parainfluenza virus Type 3		2					2		1		1					6	5	4	1
Parvovirus		2	1				5		2	3	11	3	1			28	25	29	10
Pneumocystis carinii																0	2	4	3
Respiratory Syncytial virus		7	8				27		5	5	18	8	1	4		83	76	58	53
Ross River virus	4	6		1			2		8	6	11	5			3	46	43	161	177
Rubella virus							1									1	2	1	0
Salmonella paratyphi A																0	0	0	0
Salmonella paratyphi B																0	0	0	1
Salmonella typhi																0	0	0	1
Shigella dysenteriae																0	0	0	0
Shigella flexneri																0	0	0	0
Streptococcus Group A	9	6	5				10	19	5	5	20	8	2	4	1	94	90	116	84
Toxoplasma gondii							1									1	0	3	1
Treponema pallidum	14	9	5		2		21	1	3	3	31	9	5	6		109	118	159	122
Trichomonas vaginalis	1										1			1		3	12	10	12
Varicella Zoster virus	9	27	15	1	1	5	21		12	6	49	26	3	4	2	181	141	156	137
Yersinia enterocolitica																0	0	0	0
TOTAL	178	320	132	45	12	8	481	25	220	104	584	261	44	78	43	2535	2151	2540	2114

### REGIONS

1 Cairns  
2 Gold Coast/Northern Rivers  
3 Ipswich

4 Mackay  
5 Mount Isa  
6 New England  
7 North Brisbane Suburbs

8 Northern Territory  
9 Redcliffe  
10 Rockhampton  
11 South Brisbane Suburbs

12 Sunshine Coast  
13 Toowoomba  
14 Townsville  
15 Wide Bay/Burnett

June 2007 and further historical clinical data can be obtained by contacting your local Medical Liaison Officer

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## Adverse Effects Associated with the Glucose Tolerance Test

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The glucose tolerance test or one of its variations is a frequently performed test with laboratories of our size performing and reporting over a hundred daily. As such, it is generally viewed as a safe test. However we occasionally come across adverse responses to the test. Most are trivial but as clinicians, we should be aware of the breadth of possible effects and if necessary, discuss these with the patient at the time that the test is being considered.

In the late 1970s, WHO published guidelines to standardise the performance and interpretation of glucose tolerance tests for diagnosing diabetes mellitus. The test has undergone several revisions since then (predominantly with respect to interpretation of results).

The day-to-day variation of tolerance within any one patient is well known and contributes to concerns held with respect to the value or reliability of the test in diagnosing diabetes mellitus. However to put these concerns into context, when diagnostic fasting or random glucose values are not available, it is the best test currently available for this purpose. Currently the use of glycated haemoglobin for the diagnosis of diabetes is not accepted in Australia and indeed the use of HbA1c for this purpose is not approved under Medicare.

### The adverse effects of the test can be divided into several categories:

1. Effects of the pre-test fast
2. Effects of the glucose load itself
3. Effects of additives used in the commercially available glucose loads.

#### Effects of the Pre-test Fast

The period of fasting prior to the test (typically for approximately 12 hours) may be associated with symptoms such as dizziness, tiredness etc, which are often ascribed to hypoglycaemia, although the initial plasma glucose performed as part of the test assures us that true hypoglycaemia is exceptionally rare. Note – If a 'true' fasting hypoglycaemia is noted (a value of below 2.0 mmol/L), it may be useful to contact the laboratory to ask for insulin to be performed on the sample on hand. Although insulinoma is rare, we come across occasional cases in this fashion.

#### Effects of the Glucose Load

Although it is always undesirable to give a 75 g glucose load to a patient with diabetes mellitus, the incidence of adverse effects arising from this is exceptionally low. Indeed, in the almost 30 years for which I have been supervising glucose tolerance tests, I am not aware of any medically significant adverse outcome arising from the test being performed inadvertently on an unrecognised diabetic.

If you have an asymptomatic patient with a fasting glucose of 7.0 mmol/L or higher or a non-fasting glucose of 11.1 mmol/L or higher (that is within the so-called 'diabetic range'), it is recommended that you review recent serum glucose tests. If there is another value within the diabetic range, then diabetes mellitus is confirmed. If not, rather than ordering glucose tolerance testing, it is recommended that you simply repeat the fasting or a two-hour post-prandial glucose test. A second value within the diabetic range confirms diabetes mellitus without the need to proceed to glucose loading. Of course in the presence of diabetic symptoms, a single diagnostic value is all that is required.

The interpretative criteria are tabulated (Table 1).

Note – These differ from the criteria for diagnosis of gestational diabetes (Table 2).

A more frequent concern is the development of mild overshoot hypoglycaemia in patients two to three hours after the glucose load. This is not a rare occurrence in normal individuals and may be associated with vague symptoms of dizziness, confusion, etc. It is exceptionally rare for severe symptoms of hypoglycaemia such as sweating, pallor, loss of consciousness etc to occur. However, if there is a concern about possible post-prandial hypoglycaemic episodes in the past, it may be appropriate to advise the patient to consider arranging for a companion or for public transport to convey them home from the test.

Clearly, should an unusually low glucose value come to light in the report, it is appropriate to contact the laboratory and ask for insulin to be assayed on the samples. Again, insulinoma is a rare finding but early recognition and treatment is beneficial to the patient.

Our collection staff are instructed that should a patient develop symptoms suggestive of hypoglycaemia during the test, they are to collect an additional sample at the time of the symptoms for glucose and potentially insulin assay.

#### Effects of Additives

Rarely we receive enquiries about possible adverse effects from other constituents in the glucose load which we routinely supply. The material used by QML Pathology contains carbonated water, glucose (75 g), food acid 330 (citric acid), preservatives 211 and 202 (benzoate and sorbate), and a colouring agent, fast green 143. It contains no gluten and is suitable for use in patients with gluten intolerance.

We use this to minimise the possibility of nausea and vomiting after taking the load - developments which frequently invalidate the test. The procedure itself - that is taking nothing other than water for up to 12 hours and then taking 2 cups of 25% glucose solution - is unnatural to put it mildly! If this is taken as a simple sugar solution, nausea and vomiting occurs in up to 40% of cases.

The commercial preparation described above approximates commercial soft drinks (although with a higher glucose content) and is much more palatable. However, even with this, the incidence of nausea and vomiting of sufficient severity to lead to cancellation of the test is not negligible. We have to cancel and rebook several tests each day for this reason. This effect is seen most frequently in pregnant women.

# QML Pathology updates Aug 07

## The Glucose Tolerance Test in the Non-Pregnant State for the Investigation of Hyperglycaemia: Interpretation:

	Fasting glucose mmol/L		2 hour glucose mmol/L
Normal	below 6.1	and	below 7.8
Impaired fasting glycaemia	6.1– 6.9	and	below 7.8
Impaired glucose tolerance	below 7.0	and	7.8 – 11.0
Confirmed diabetes mellitus	7.0 or higher	and	11.1 or higher
Possible diabetes mellitus	7.0 or higher	<b>or</b>	11.1 or higher
Possible diabetes mellitus is confirmed if:			
<ul style="list-style-type: none"><li>• The patient has symptoms</li><li>• The patient has a fasting or a non-fasting glucose within the 'diabetic range' on a different day.</li></ul>			

Table 1

## The Glucose Tolerance Test in the Pregnant State for the Investigation of Suspected Gestational Diabetes: Interpretation:

	Fasting glucose mmol/L		2 hour glucose mmol/L
Normal	below 5.5	and	below 8.0
Gestational diabetes	5.5 or higher	or	8.0 or higher

Table 2

If your patient is known to have or suspected to have an adverse response to any of the additives listed above, please note this on the request form and ask the patient to contact the collection centre in advance. In the same way that we send out dry sugar preparations for breath hydrogen tests, we can arrange to send out 75 g of pure glucose for this test, with instructions to make a concentrated solution for the patient to drink on site. However the potential difficulty with nausea and vomiting remains high with this approach.

In summary, the oral glucose tolerance test is the most frequently performed dynamic test currently in use. It is regarded as safe and relatively convenient for the patient but on occasions, mild side effects may be experienced. However if concerns exist, a quick discussion with your local Pathologist may assist in laying concerns to rest.

### Ordering Anti-D Immunoglobulin

The Blood Bank is introducing a form for quick, easy and efficient ordering and delivery of anti-D immunoglobulin. The form can be downloaded from the home page of the QML Pathology website [www.qml.com.au](http://www.qml.com.au) and then completed and faxed to the Blood Bank. Alternatively, a pad of request forms can be ordered through QML Pathology Blood Bank on (07) 3876 8371. The completed form can be faxed to the Blood Bank on (07) 3371 9029.

Requests will be processed twice a day and anti-D immunoglobulin will be delivered to the requesting clinic with the next routine courier run. Please note that the next courier delivery may be the following day if the order is faxed in the afternoon.

## Doctor's Noticeboard

- Dr Michele Calvird, General Adult Psychiatrist, has recommenced practice following maternity leave. Her practice is located at the Toowong Specialist Centre, 496 Milton Road, Toowong. Dr Calvird has a special interest in women's mental health. For appointments please telephone (07) 3721 8011.
- VERY large consulting room available for sessional use. Located inside QML Pathology's Robina Collection Centre the room is available any Monday, Wednesday or Thursday. The room is currently used by an ENT and Audiologist. Please contact Dylan on 0411 35 37 31 for further information.

## New Collection Centres

### Manly West

148 Radford Road  
Manly QLD 4179  
Phone: (07) 3348 4021  
Hours:  
Monday to Friday:  
8.00am – 12.30pm and 1.00pm – 4.00pm.

## Relocated Collection Centres

### Kingaroy

15 Glendon Street  
Kingaroy QLD 4610  
Phone: (07) 4162 1499  
Hours: 7.30am – 4.00pm (Monday to Friday)