

# QML PATHOLOGY

newsletter May 09

## >>Haemolytic Disease of the Fetus and the Newborn (Erythroblastosis Fetalis): Antenatal Testing and Management Guidelines.

**Dr David De Leacy, Consultant Haematologist**

The identification of the underlying pathology and the development of treatment protocols, followed by the implementation of a successful preventative/suppressive strategy to combat Haemolytic Disease of the Newborn (HDN) stands as a classical example of extraordinary scientific endeavour over the last century.

## >> Haemolytic Disease of the Fetus and the Newborn (Erythroblastosis Fetalis): Antenatal Testing and Management Guidelines.

Dr David De Leacy, Consultant Haematologist

It is now well-known that the Rh(D) antigen is the most immunogenic of all the red cell antigens, hence the probability of immunisation by the presence of Rh(D) positive (D+) red cells in the circulation in susceptible Rh negative (D-) persons is much higher than for all of the other red cell antigens. It was the demonstration of the interaction of this antigen with its associated antibody (Anti-D) as the major (but not sole) cause of Haemolytic Disease of the Newborn (HDN) that allowed for rational strategies to be developed to combat this devastating disease.

In Caucasian countries prior to **1945** (where approximately 15% of the population are Rh(D-)), the perinatal mortality rate stood at around 4000 per 100,000 births and HDN accounted for around 10% of all of those deaths. At that time, half of all the fetuses with HDN died of either kernicterus or hydrops fetalis. (Note: Only 5% of the African, 0.5% of the Chinese and 7% of the Indian population are Rh(D-).

From the 1940s onwards the successful transfer of basic scientific research into medical practice has seen a major decline in infant mortality and morbidity rates from HDN in all the countries of the world where large populations of Rh(D-) people live.

### History

It was in **1609** that a French midwife first reported in the popular press of the day on the fate of newborn twins where one was oedematous at birth and died immediately (hydropic), and the other developed neurologic symptoms and died three days later (kernicterus). It was not until **1932** that the two conditions were again combined when Diamond and Blackfan proposed that they were in fact two aspects of the one disease.

In **1938**, Dr Ruth Darrow, a pathologist, suggested that maternal immunisation by transplacental transfer of fetal red cells, followed by the reverse transfer of that antibody to the fetus, was the causative mechanism of the disorder. She wrongly surmised that fetal haemoglobin was the antigen involved.

The **1940** discovery of the Rh blood group system by Landesteiner and Weiner allowed Levine in **1941** to demonstrate that the Rh antigen D on Rh positive fetal red cells circulating in the maternal blood stream was the target molecule that elicited an IgG Anti-D response in Rh(D-) mothers. He also showed that the maternal alloantibody then crossed the placenta and attached to red cells of the fetus that were then removed from its circulation and destroyed.

In **1956** Levine went on to show that Rh(D-) mothers who carry an Rh(D+) fetus and lack A and B iso-agglutinins

are more likely to develop Rh antibodies than those that have them, i.e. if the Rh(D+) fetus was ABO incompatible with its Rh(D-) mother (and 20% are), then the risk of HDN was only 2% as opposed to the 16% risk where ABO compatibility between mother and fetus existed. The combined overall risk of HDN for all Rh(D-) mothers was shown to be 13.2%. Levine showed that the rapid clearance of the ABO/Rh+ incompatible fetal red cells by the maternal Anti-A or Anti-B somehow mitigated against the active immunisation that lead to maternal Anti-D production.

Interestingly, insight into the role that presence of passive antibody plays in the prevention of immunisation was first described over half a century earlier by Von Dungern in **1900** in studies using ox red cell infusions into rabbits. The significance of his unique observations had to wait 60 years until the explosion of knowledge about transfusion practice and immuno-haematology occurred in the 1950s and 1960s for translation into clinical application.

It was not until **1961** that Finn showed that administration of passive Anti-D accelerated the clearance of Rh(D+) red cells administered to Rh(D-) male volunteers. In **1963** Freda produced the first specific Anti-D immunoglobulin preparation and in the same year Schneider was able to show similar results in non-pregnant Rh(D-) female volunteers who were given passive Anti-D to protect against immunisation after infusion of Rh(D+) cells.

It was finally in **1965** that Clarke and Freda separately reported the successful prevention of Rh(D) sensitisation by the post-partum administration of Anti-D to Rh(D-) mothers with Rh(D+) babies.

### HDN Treatment

With the discovery of the main cause of HDN in the 1940s, Wallerstein in **1946** introduced the practice of neonatal exchange transfusion as a treatment modality for kernicterus. This led to a dramatic fall (75%) in HDN associated perinatal mortality from 10% to around 2 to 3% by the early 1960s.

The evolution of expert obstetric units undertaking the practices of planned premature delivery, amniotic fluid spectrophotometry, intraperitoneal fetal transfusion, fetal blood sampling, and finally intravascular fetal transfusion, all helped incrementally improve birth outcomes in HDN.

### HDN Prophylaxis

It was realised in the 1960s and 1970s that the only way left to reduce perinatal mortality further was by the prevention of the disease in the first place using passive Anti-D injections.

Two caveats were quickly established:

1. that the dose must be sufficient; and
2. that it must be given before the Rh immunisation process has begun.

Chown in **1969** reported the first successful results of a major trial in Canada, where Anti-D given to Rh(D)- mothers within 72 hours of birth prevented primary immunisation. Subsequently it has been shown that Anti-D given post-partum even up to 13 days (and maybe as long as 28 days), provides possible partial protection for a subset of patients.

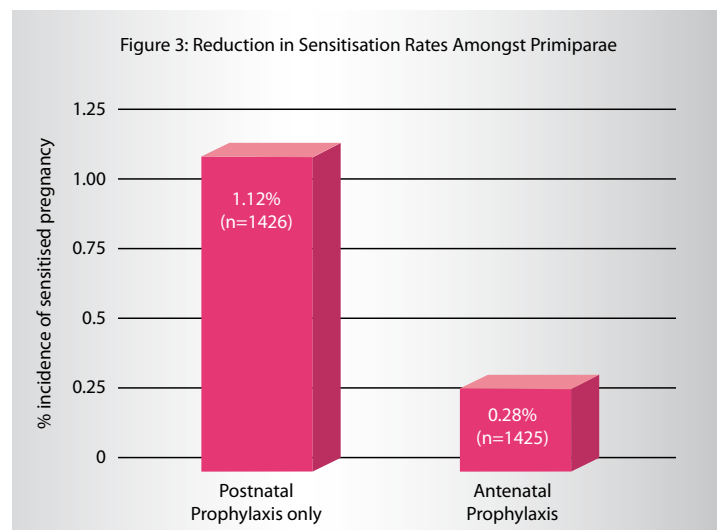
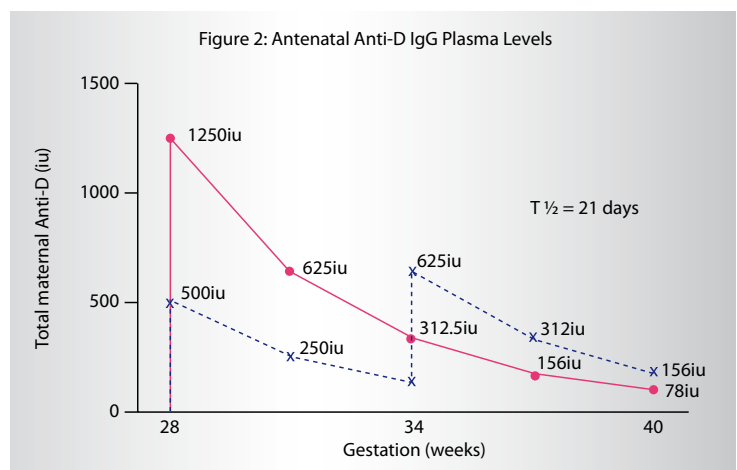
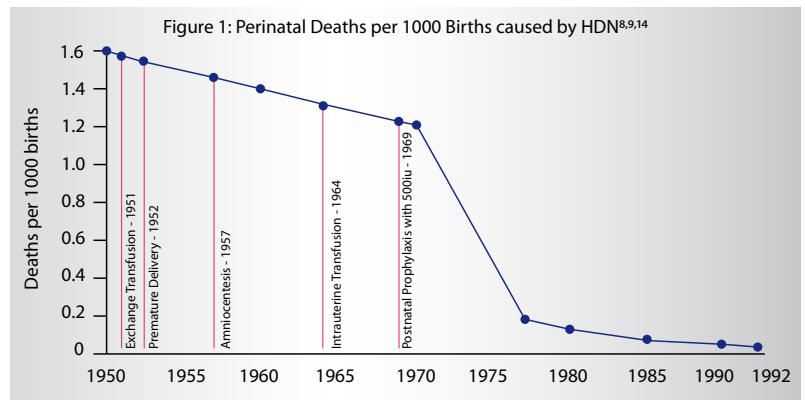
In **1971** a major overview of 10 countries (29,620 pregnancies), showed that the passive use of Anti-D post-delivery provided 90% protection against the development of a maternal allo-Anti-D. The success generated in the United Kingdom by an early post-natal (within 72hrs) Anti-D program for all Rh(D)- mothers with Rh(D+) babies that commenced in **1969** was astounding. When reviewed in **1991** deaths attributed to Rh(D) allo-immunisation had fallen from 46/100,000 to 1.6/100,000.

## Fetomaternal Haemorrhage (FMH)

Up to 99.3% of all women have a FMH of less than 4 ml at birth. 50% of the larger FMHs also occur after normal deliveries with 0.3% having a FMH of greater than 15 ml (some up to 30 ml). It became clear that an accurate estimate of FMH was of critical importance in determining the correct dose of Anti-D. It was subsequently shown that approximately 125IUs of Anti-D was required to suppress 1ml of Rh positive red cells in patients.

Kleihauer in **1957** developed a staining technique for detecting fetal red cells in adult blood smears that allowed for a reasonably accurate quantitation in experienced hands. More recently, flow cytometry techniques have replaced the Kleihauer staining technique in larger centres. This newer modality is both more accurate and more precise.

A sensitising FMH can occur at any time during pregnancy (usually after 8 weeks) with around 8% occurring before 28 weeks, 16% from 28 to 34 weeks, and 76% from 34 weeks to 3 days post partum. Protocols have now been developed that provide for Anti-D injections at 28 and 34 weeks, as well as at delivery or whenever any clinical event with an associated risk of FMH occurs during pregnancy (e.g. abortions, traumatic deliveries or Caesarean sections, twins, stillbirths and amniocentesis). These added interventions have further lowered the sensitisation rate from 1.2% to 0.28%, so that maternal Anti-D immunisation is now at similar low rates to the other known causes of HDN (e.g. Anti-C and Anti-K).



## The Australian Experience

After a major review in 2003, the NH&MRC recommended similar combined antenatal and postnatal Anti-D dosing guidelines.

The ongoing work of the ARCBS in providing CSL Australia with adequate supplies of Anti-D immunoglobulin from very generous actively Rh(D) immunised male donors has allowed Australia to be self-sufficient in Anti-D stocks. This needs to be fully acknowledged and supported.



## 2003 NH&MRC Guidelines for the use of Rh(D) Immunoglobulin (Anti-D) in pregnant women with Rh(D-) blood group and no pre-existing Anti-D antibodies

- Rh(D) Immunoglobulin should be administered as soon as possible after the sensitising event, but always within 72 hrs for successful immunoprophylaxis.
- If Rh(D) Immunoglobulin has not been administered within 72 hrs, a dose offered within up to 9-10 days may provide protection.
- Rh(D) Immunoglobulin should not be given to women with preformed Anti-D antibodies, except where the preformed Anti-D is due to the antenatal administration of Rh(D) Immunoglobulin.
- Rh(D) Immunoglobulin 100 IU is sufficient to protect against a Fetomaternal Haemorrhage (FMH) of 1.0 ml of fetal red cells (2.0 ml whole blood).
- Quantify the magnitude of the FMH following a sensitising event (including delivery) to ensure an adequate dose of Rh(D) Immunoglobulin is offered, as more than one dose may be required.
- Tests to assess the volume of FMH include but are not limited to the Kleihauer-Betke acid elution test and flow cytometry.
- The majority of fetal bleeds are less than 5 ml of red blood cells.
  - In about 50% of cases, FMH is less than 0.05 ml
  - In about 5% of cases, FMH is greater than 0.5 ml
  - In about 3% of cases, FMH is greater than 1 ml
  - In up to 0.6% of cases, FMH is 30 ml or greater.

### Sensitising Events

For each sensitising event administer Rh(D) Immunoglobulin as indicated in Table 1 below. <sup>(1)</sup>

#### Sensitising events include: <sup>(2)</sup>

- Normal delivery
- Ectopic pregnancy
- Miscarriage

- Termination of pregnancy
- Invasive prenatal diagnostic procedures (including chorionic villus sampling, amniocentesis and cordocentesis)
- Abdominal trauma considered sufficient to cause fetomaternal haemorrhage
- External cephalic version
- Antepartum haemorrhage.

The batch number of every vial of human immunoglobulin administered must be recorded in the patient's medical history and in accordance with other legal statutory requirements.

**\*\***In some circumstances, access to an intravenous preparation may be warranted. A quantity of intravenous Rh(D) immunoglobulin (WinRho SDFTM) will be available for this purpose. Contact ARCBS in your capital city for more information.

Approximately 17% of pregnant Caucasian women will be Rh(D-), and their babies (if Rh(D+)) may be at risk of developing Haemolytic Disease of the Newborn (HDN) due to Rh(D) incompatibility.<sup>(2)</sup>

Antibody formation occurs during pregnancy in 1.5% of Rh(D-) women carrying a Rh(D+) infant, despite use of postnatal prophylaxis.<sup>(2)</sup>

The rate of antibody formation can be reduced to 0.2% or less by the administration of Rh(D) Immunoglobulin during pregnancy, at 28 weeks and 34 weeks (antenatal prophylaxis), as well as after delivery.<sup>(2)</sup>



**Table 1**

ctd>

| Week 1 to Week 12 (first trimester)         |   | Beyond Week 12 (second & third trimester)   | Administer at Week 28 & Week 34  | Postpartum   |
|---|---|---|--|--|
| Rh(D) Immunoglobulin (Single pregnancy)     | Rh(D) Immunoglobulin (Multiple pregnancy)   | Rh(D) Immunoglobulin                        | Rh(D) Immunoglobulin   | Rh(D) Immunoglobulin** (WinRho SDFTM no longer required) |
| 250 IU Solution for intramuscular injection | 625 IU Solution for intramuscular injection | 625 IU Solution for intramuscular injection | 625 IU<br>The doses at 28 & 34 weeks are given in ADDITION to any doses given for sensitising events | 625 IU Solution for intramuscular injection              |

## Presentation

Single vials containing 625IU or 250IU Anti-D antibody.

## Contraindications

Should not be given to:

- An Rh(D+) or D<sup>u</sup> positive individual
- An Rh(D-) or Rh(D<sup>u</sup>) positive individual previously sensitised to Rh(D) antigen.

**Note:** *Although there is no benefit in administering Rh(D) immunoglobulin to a woman who is already sensitised to Rh factor, there is no more risk than when it is given to a woman who is not sensitised*

- Individuals with IgA deficiency – unless they have no circulating anti-IgA antibodies
- Individuals with severe thrombocytopenia or coagulation disorder that would contraindicate IM injections.

## Precautions

- MUST NOT be administered intravenously.
- Give with caution to patients with a history of prior systemic allergic reactions to human immunoglobulin preparations.
- Must not be given to D+ postpartum infants. Babies born of women given Rh(D) Immunoglobulin antepartum may have a weakly positive Coombs' test at birth.

## Availability

Requests to ARCBS. Some hospitals may hold stocks.

## Dosage and Administration

Dosage as outlined above per NHMRC Best Practice Guidelines.

Administer by slow intramuscular injection using large gauge needle (20g). If a large dose (more than 5 ml) is required, it is advisable to administer it in divided doses at different sites. Suitable local anaesthetic may be added if desired.

The product does not contain antimicrobial preservative; it must be used immediately after opening the vial.

**Note:** *Supplies of suitable plasma for Rh(D) Immunoglobulin production are scarce. Individuals who have Rh(D) antibodies should be urged to enrol as voluntary blood donors.*

## References

- (1) Communication from Professor Richard Smallwood, Chief Medical Officer, Commonwealth of Australia (November 2005)
- (2) NHMRC Guidelines on the prophylactic use of Rh(D) Immunoglobulin (Anti-D) in obstetrics 2003; [www.nba.gov.au/pubs/pdf/glines-anti-d.pdf](http://www.nba.gov.au/pubs/pdf/glines-anti-d.pdf)

## The ANZSBT and the RANZCOG produced updated Guidelines for Blood Grouping and Antibody Screening in the Antenatal and Perinatal Setting in March 2007:

### Antibodies in Pregnancy - Alloimmunisation

Whilst the Rh(D) antigen is most immunogenic of the red cell antigens, it is not alone in causing HDN. To cross the placenta an antibody must be of an IgG type (subclass 1 or subclass 3) with an Fc receptor. Many factors influence the avidity of antibody attachment to the placenta and subsequent transfer into the fetal circulation.

The ANZSBT Guidelines divide the clinically significant red cell alloantibodies into three groupings.

#### Group 1

**Anti-D, -c, -E, -e, -C, -K, -k, -Fy<sup>a</sup>.**

*These antibodies are commonly associated with clinical HDN. Those most often associated with moderate to severe HDN are Anti-D, Anti-C and Anti-K. Others less frequently encountered antibodies may also cause clinical HDN.*

*Once the antibody has been assessed as having the potential to cause clinical HDN, the titre/quantitation of antibody should be determined by a standardised technique. Antibody investigation and titre/quantitation should be repeated every 4 weeks until 32 weeks gestation then every two weeks until delivery. When a clinically significant rise in titre/quantitation occurs, the results of*

*antibody monitoring aid the clinician in determining when to initiate fetal monitoring such as ultrasound, amniocentesis or cordocentesis.*

**(Note: Anti-K is unique in causing significant HDN without a detectable rise in titre being present during testing hence close attention needs to be paid to mothers with this alloantibody.)**

#### Group 2

**Anti, -C<sup>w</sup>, -Fy<sup>b</sup>, -Jk<sup>a</sup>, -Jk<sup>b</sup>, -Jk3, -S, -s, -M, -Ge<sup>a</sup>.**

*These antibodies may cause a positive direct antiglobulin test (DAT) but therapy, if necessary, is likely to be limited to phototherapy.*

#### Group 3

**Anti-P1, -N, -H, -Le<sup>a</sup>, -Le<sup>b</sup>, -Le<sup>a+b</sup>, -Lu<sup>a</sup>, -Lu<sup>b</sup>, -Sd<sup>a</sup>, -HLA.**

*These antibodies are not documented to cause clinical HDN.*

### Titration of Non-Rh antibodies

*These titrations should be undertaken only after discussion with the obstetrician as to the significance of the results and how data obtained will affect patient management. There is little data available concerning critical titres for non-Rh antibodies encountered in pregnancy.*

In closing it should be remembered that HDN may be caused by maternal antibodies other than those derived directly by fetal red cell stimulation.

The transfusion of Rh or K incompatible blood to antigen negative women of child bearing age should be avoided if at all possible to prevent the risk red cell allo-immunisation prior to pregnancy.

Some maternal autoantibodies may cross the placenta. Hence, any positive direct antiglobulin test (DAT) result recorded in young women should be fully investigated. Most autoantibodies are pan-reacting against all the red cells in a panel, however, very occasionally autoantibodies may display an Rh tropism.

Drugs (e.g. Aldomet) can induce a strong DAT in some people and in a small percentage cause a florid haemolytic anaemia. Aldomet is an old drug but is still widely used in pregnancy.

Any concerns with Antenatal testing or blood grouping should be referred to a Haematologist or the ARCBS Reference Laboratory.

## References:

*"Thirty-five Years of Rh Prophylaxis"*. John Bowman. Transfusion 2003; 43: 1661-1666.

*"The History of Rhesus Prophylaxis with Anti-D"*. A Wegmann et al. Eur J Paediatrics 1996; 155: 835-838.

*"Probability of Anti-D Development in D - patients receiving D+ RBCs"*. Frohn et al. Transfusion 2003; 43: 893-898.

*"The Use of Anti-D Immunoglobulin for Rh Prophylaxis"*. Royal College of Obstetrician Guidelines, UK. 1999.

*"Anti-D Haemolytic Disease of the Newborn. Past Present and Future"*. Bio Products Laboratory UK Publication. April 1999.

*"Guidelines on Prophylactic use of Rh(D) Immunoglobulin (Anti-D) in Obstetrics"*. NHMRC Guidelines/NBA. 2003.

*"Consensus Conference on Anti-D Prophylaxis"*. Royal College of Physicians of Edinburgh and the Royal College of Obstetricians and Gynaecologists". 1997.

*"Guidelines for Blood Grouping and Antibody Screening in the Antenatal and Perinatal Period"*. ANZSBT and The Royal Australian and New Zealand College of Obstetricians and Gynaecologists". March 2007.



**Dr David De Leacy**  
FRCPA. Bsc.

Consultant Haematologist

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A graduate of the University of Queensland Medical School in 1974 (MBBS), Dr De Leacy undertook his residency at the Royal Brisbane Hospital. Upon completion he worked in a number of roles including rural medicine, general practice and a year as a Medical Officer in Antarctica for ANARE (1987) before finishing his training in pathology at the Royal Brisbane Hospital, having earlier also trained at the Princess Alexandra Hospital.

After gaining his fellowship in 1988 Dr De Leacy worked as a Consultant Haematologist at the Royal Canberra Hospital followed by a position as Assistant Director at the Red Cross Blood Transfusion Service (Qld).

He spent 10 years overseas working as Head of Laboratories/ Director of Medical Services at Wadi al Dawasir Military Hospital, Saudi Arabia, as a Consultant Haematologist at the National Blood Service, UK and for the NZ Blood Service/ Canterbury Health Service Laboratories, and also for the WHO for one year in Vietnam.

In 2003 Dr De Leacy returned to Australia to work as a Consultant Haematologist at the St John of God Pathology/ Pathcare Laboratories Victoria before joining QML Pathology in 2008.

The Infectious Diseases Report - Geographic Distribution - March 2009 can be found on the back page of the May updates section.

This newsletter has been prepared and published by QML Pathology for the information of referring doctors. Although every effort has been made to ensure that the newsletter is free from error or omission, readers are advised that the newsletter is not a substitute for detailed professional advice.

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# QML Pathology updates May 09

## >> Important Changes to Prostate Specific Antigen Ordering

**As of 1 May 2009, the Medicare Benefits Schedule for PSA item numbers has been amended to reflect PSTC recommendations. QML Pathology has taken this opportunity to enhance its reporting algorithms to more accurately reflect clinical practice, and more individually tailor the testing based on the age of the individual patient.**

### PSA Testing from 1 May 2009

If you order a PSA:

- A Total PSA (PSA) will be analysed and reported
- If the PSA was ordered for general screening, then the PSA:
  - will be Medicare claimable if the patient has not had a PSA being performed over the prior 12 months, OR
  - will not be Medicare claimable (and will be privately charged) if the patient has already had one or more PSAs ordered in the past 12 months.

### Free PSA Testing from 1 May 2009

The MBS changes as of 1 May, have had effect to enabling Medicare claimable fractions to be performed at lower levels of PSA, but reduced the number of fractions claimable at the upper levels of the old 'grey zone'. The lower limit is now adjusted for the age of the patient

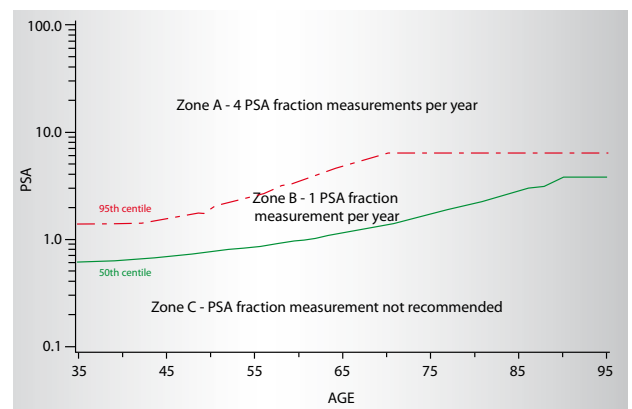
The MBS changes have meant that the PSA Fraction Ranges (the old grey zone):

- a) are now defined for Medicare purposes as two sub-segments, the lower segment being PSA results lying between the Age Related Median and the Upper Limit of Normal, and the upper segment being results lying between the Upper Limit of Normal and

the Upper Equivocal Limit

- b) one PSA Fraction analysis is Medicare claimable every 12 months in the lower segment
- c) four PSA Fraction analyses are Medicare claimable every 12 months in the upper segment.

### Fraction Testing Ranges now Age Dependent:



From 1 May 2009, if you order PSA and free PSA:

- A PSA, and free PSA will be analysed and reported
- The free PSA will be Medicare claimable if the current PSA or a recent prior PSA result for this patient:
  - is between the age related median and the upper limit of normal, and there is no record of a prior fraction result in the past 12 months
  - is between the upper limit of normal and 10ug/L, and there is no more than 3 previous free PSA results recorded over the past 12 months.
- If the free PSA do not meet either of the above criteria, then the patient will be privately charged.

## Sample Identification

As per accreditation requirements, the minimum requirements for labelling samples are two identifiers that are linked to the patient; usually the patient's full name, and either a date of birth or medical record number. As a reminder, can you please ensure all samples are fully and correctly labelled (in particular, Pap smears) with at least two identifiers.

Unlabelled or incorrectly labelled samples are the largest issue when it comes to identification problems. To maintain good pathology practice, and ensure efficient and accurate handling of patient samples, your cooperation with this process is sought.



## >> Doctor's Noticeboard

**Dr Lydia Pitcher, Paediatric Haematologist**, has commenced private clinical practice at HOCA's new clinic in Chermerside and is available for consultation regarding blood disorders in children and young adults (including cytopenias, haemostasis and thrombosis, thalassaemia and late effects of chemotherapy).

Dr Pitcher has dual fellowships in Paediatrics and Haematology (Pathology), and extensive experience in clinical Paediatric Haematology/Oncology at consultant level, having worked and trained locally and overseas.

For all appointments:

Phone: (07) 3737 4834

Fax: (07) 3737 4801

Email: [lpitcher@hoca.com.au](mailto:lpitcher@hoca.com.au)

Haematology and Oncology Clinics of Australasia (HOCA)  
Level 1, Chermerside Medical Centre  
956 Gympie Road, Chermerside.

### Allergy Practice – Dr Merv Garrett

Mermaid Central Medical Practice Building  
2431 Gold Coast Hwy (cnr William St) Mermaid Beach.  
Appointments by phone (07) 5575 2444.

### Commercial suite for lease - Murwillumbah, NSW

60 square metres. Lovely treed location shared by South Coast Radiology and Department of Ageing, Disability and Homecare. Excellent disability access and facilities. \$13,800 p.a. + outgoings + GST.

Contact: Richard Anderson (02) 6674 1100  
or Bob Meehan 0418 155 540.

**Dr Andrea Riha, Vascular Physician**, is pleased to announce that after a period of absence from practice she has now returned to consulting. Dr Andrea Riha is happy to assist with any referrals.

Contact Dr Riha at:

Wesley Vascular Centre, Suite 65, 2nd Floor, Sandford Jackson Building, Wesley Private Hospital. Wesley Private Hospital appointments can be made on (07) 3377 5900.

**Dr Scott Sommerville, Orthopaedic Surgeon**, has commenced a regular consulting session at The Greenslopes Private Hospital. Dr Sommerville also has a regular consulting session at The Sunnybank Private Hospital. His main rooms are located in The Wesley Medical Centre.

Dr Sommerville has a special interest in surgery of the Hip and Knee, Primary and Revision hip and knee joint replacement, and musculoskeletal tumour surgery. For more information or to arrange an appointment, please call (07) 3720 8333.

**Respiratory and Sleep Physician, Dr Roo Killick**, has recently joined the Respiratory and Sleep Specialists team on the Gold Coast, treating outpatients at John Flynn Hospital, Tugun.

In addition to the arrival of a new Consultant, March marked the opening of the Gold Coast Sleep Health Centre at John Flynn. Dr Killick and the team at the Gold Coast Sleep Health Centre look forward to meeting you and continuing the excellent tradition of service and treatment provided by this established local team.

Gold Coast Sleep Health Centre  
John Flynn Hospital  
Suite 1C, John Flynn Medical Centre  
42 Inland Drive, Tugun

Phone: (07) 5598 0765 or 1800 155 225.

Fax: (07) 5598 0700

Email: [goldcoastshc@sleepspecialists.com.au](mailto:goldcoastshc@sleepspecialists.com.au)

Web: [www.sleepspecialists.com.au](http://www.sleepspecialists.com.au)

**Dr Michael Keogh, Endocrinologist**, has relocated his practice rooms to:

Suite 11, Level 9, Evan Thomson Building  
24 Chasely St, Auchenflower.

Phone: (07) 3871 0050 (All Appointments)

Fax: (07) 3871 2031.

He will also continue to consult weekly at the Aspley Specialist Centre  
(now located at 601 Robinson Rd, Aspley).

**Dr Basit Mirza, Dermatologist**, with special interests in acute dermatoses, paediatric dermatology, skin cancer surgery and photodynamic therapy, has new rooms at:

Suite 3B, Level 3, Lantos Place,  
80 Stamford Road (Cnr Station Road), Indooroopilly.

Phone: (07) 3878 7275

Fax: (07) 3378 2435

Email: [info@drmirza.com.au](mailto:info@drmirza.com.au)

Dr Mirza no longer practices from Wickham Terrace or Indooroopilly Shoppingtown.

### Dr Mirza has Sessional Rooms for Lease

Adjacent to Indooroopilly Shoppingtown. Specialist rooms with amazing views to the city. Newly refurbished one or two consulting rooms, each with examination couch, desk and wash basin. Reception, meeting and greeting and invoicing available. Pathology available. Ample parking nearby. Please contact Lisa on (07) 3878 7275 or [info@drmirza.com.au](mailto:info@drmirza.com.au).

### Introducing Dr Michael Bryant at BrizBrain & Spine

Dr Michael Bryant is a neurosurgeon, specialising in conditions involving the brain and spine. He has been associated with BrizBrain & Spine since its foundation in 2005.

To contact Dr Bryant or find out more about BrizBrain & Spine, please call (07) 3833 2500 or visit [www.brizbrain.com.au](http://www.brizbrain.com.au).





# QML Pathology updates May 09

## >> Federal Government Cuts Pathology Rebates

On the 12th May the Federal Government released their budget for the 2009/10 financial year. The proposed budget has introduced a number of significant cuts to pathology funding, including reduction in collection fees and a number of testing rebate fees. Some of the core tests that will be directly affected include Full Blood Counts, Iron Studies, HDL, TSH and INR tests. The decrease in the rebate for INR testing was particularly disappointing considering the lack of funding for warfarin dosing services.

The changes that the Government has proposed will have a massive affect on the Queensland Pathology market; we are currently working through the issues and options arising from these changes. We will keep you informed as we progress through the multiple hurdles these changes represent for the industry.

### New Collection Centres

#### Stafford

Unit B, 267 Stafford Road

Phone: (07) 3357 5794

Opening Hours:

7.30am - 12.00pm, 12.30pm - 3.30pm (Mon-Fri)

#### Rosewood

Shop 1, Plaza Complex

Royal George Lane

Phone: (07) 5464 1940

Opening Hours:

7.00am - 12.00pm (Mon-Fri)

### Relocated Collection Centres

#### Auchenflower

Suite 90, Fifth Floor, Sandford Jackson Building

Wesley Hospital, 30 Chasely St

Phone: (07) 3371 3592

Opening Hours:

8.00am - 6.00pm (Mon-Fri)

#### Ingham

Hinchinbrook Central Shopping Centre

Tenancy 25, 86-92 Herbert St

Phone: (07) 4776 0999

Opening Hours:

7.30am - 12.30pm, 1.00pm - 4.00pm (Mon-Fri)

8.00am - 11.00am (Sat)

#### Logan Central (previously Woodridge)

Shop 16, Logan City Centre

Cnr Kingston and Wembley Rds

Phone: (07) 3808 1464

Opening Hours:

7.00am - 5.00pm (Mon-Fri)

8.00am - 12.00pm (Sat)

#### Surfers Paradise

Suite T5B, Ground Floor, "Circle on Cavill"

Cnr Ferny and Cavill Aves

Phone: (07) 5504 6412

Opening Hours:

8.00am - 1.00pm, 2.00pm - 4.30pm (Mon-Fri)

## >> Donate Unwanted Medical Textbooks

Awaken Mozambique, a non-profit charity based in Brisbane and Mozambique, is collecting unwanted or unused textbooks to donate to the Medical School at Beira, Mozambique, which has just started graduating new doctors and is highly unresourced. They welcome the donation of any textbooks and journals from 2007 onwards.

Donations can be made between 1-12 June. Please contact your local QML Pathology laboratory for details.

For further information please contact;  
Dr Alexandra Evans (0408 833 021) or  
Dr Jennifer Lockwood (0414 711 877).

### Warfarin Dosing Service

QML Pathology is introducing a new procedure to give your stable patients easier access to their results. This procedure is to commence from 29/06/09. Those patients who are stable, have a 'continue same dose' instruction, and a next test date of more than 14 days will be sent their INR result, dosage confirmation, and next test date in a letter by post. They will not receive a phone call. Nursing institution/care facility residents and those patients who are receiving 'Webster pack' pharmacy support will be excluded from this process. Any patients with dosage changes, short testing intervals, those new to the monitoring program, or those with unstable results will continue to be contacted by phone as a matter of priority.

## Infectious Diseases Report - Geographic Distribution - March 2009

| ORGANISM                                | Regions (as per key below) |            |            |           |           |          |            |          |            |           |            |            |           |            |           | Total       |             |             |             |
|---|----------------------------|------------|------------|-----------|-----------|----------|------------|----------|------------|-----------|------------|------------|-----------|------------|-----------|-------------|-------------|-------------|-------------|
|   | 1                          | 2          | 3          | 4         | 5         | 6        | 7          | 8        | 9          | 10        | 11         | 12         | 13        | 14         | 15        | Mar         | Feb         | Jan         | Dec         |
| Adenovirus (not typed)                  |                            | 1          |            |           |           |          | 4          |          | 2          |           |            |            |           |            |           | 7           | 12          | 27          | 30          |
| Adenovirus (typing pending)             |                            | 12         | 3          |           |           |          | 7          |          | 12         | 13        | 9          | 2          | 1         | 1          |           | 60          | 34          | 39          | 37          |
| Barmah Forest virus                     | 4                          | 2          | 1          |           |           |          | 2          |          | 3          | 4         | 4          | 6          | 1         | 4          | 1         | 32          | 32          | 37          | 29          |
| Bordetella pertussis                    | 8                          | 16         | 8          | 3         |           |          | 18         |          | 25         | 2         | 18         | 9          | 8         | 3          | 1         | 119         | 89          | 160         | 146         |
| Brucella species                        |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 3           | 2           | 3           |
| Campylobacter jejuni                    |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 1           |
| Chlamydia pneumoniae                    |                            |            |            |           |           |          |            |          |            |           |            | 1          |           |            |           | 1           | 0           | 0           | 0           |
| Chlamydia trachomatis, not typed        | 58                         | 88         | 18         | 25        | 4         |          | 86         |          | 56         | 13        | 126        | 43         | 16        | 21         | 16        | 570         | 451         | 579         | 484         |
| Coxiella burnetii                       | 4                          |            |            |           |           | 1        |            |          |            |           |            | 1          | 1         |            |           | 7           | 8           | 4           | 9           |
| Cryptococcus species                    | 1                          |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 1           | 3           | 0           | 2           |
| Cytomegalovirus (CMV)                   | 2                          | 4          | 3          | 2         |           |          | 7          |          | 9          | 1         | 12         | 8          | 3         | 3          | 2         | 56          | 53          | 87          | 67          |
| Entamoeba histolytica                   |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| Enterovirus - not typed                 |                            | 1          |            |           |           |          |            |          |            |           |            |            |           |            |           | 1           | 4           | 1           | 3           |
| Epstein-Barr virus (EBV)                | 5                          | 13         | 8          | 2         |           |          | 13         |          | 9          | 4         | 22         | 12         | 3         | 7          | 2         | 100         | 85          | 130         | 123         |
| Flavivirus unspecified                  | 44                         |            |            |           |           |          | 1          |          | 2          |           | 1          |            | 1         | 4          | 3         | 56          | 52          | 47          | 26          |
| Hepatitis A virus                       |                            | 1          |            |           |           |          |            |          |            |           |            |            |           |            |           | 1           | 2           | 3           | 1           |
| Hepatitis B virus                       | 4                          | 6          | 2          | 2         | 1         |          | 9          |          | 5          | 1         | 49         |            |           |            |           | 79          | 48          | 84          | 51          |
| Hepatitis C virus                       | 14                         | 37         | 20         | 6         | 2         |          | 30         |          | 20         | 5         | 60         | 20         | 3         | 6          | 5         | 228         | 224         | 267         | 184         |
| Hepatitis D virus                       |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| Hepatitis E virus                       |                            |            |            |           |           |          | 1          |          |            |           |            |            |           |            |           | 1           | 0           | 1           | 0           |
| Herpes simplex Type 1                   | 21                         | 32         | 11         | 8         | 2         |          | 35         |          | 14         | 8         | 51         | 25         | 4         | 12         | 5         | 228         | 157         | 250         | 256         |
| Herpes simplex Type 2                   | 11                         | 31         | 14         | 3         |           |          | 20         |          | 16         | 4         | 30         | 23         | 4         | 5          | 1         | 162         | 131         | 180         | 164         |
| Herpes simplex virus - not typed        | 3                          | 14         | 3          | 6         |           |          | 4          |          | 7          | 2         | 8          | 2          | 1         | 1          | 3         | 54          | 40          | 57          | 69          |
| HIV-1                                   | 1                          | 1          |            |           |           |          | 1          |          |            | 1         | 1          |            |           |            |           | 5           | 5           | 4           | 3           |
| HTLV-1                                  |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| Influenza A virus                       |                            |            |            |           |           |          | 2          |          | 1          |           |            | 2          |           |            |           | 5           | 7           | 32          | 31          |
| Influenza B virus                       |                            |            |            |           |           |          | 1          |          |            |           | 1          |            |           |            |           | 2           | 4           | 6           | 3           |
| Legionella pneumophila (all serogroups) |                            | 1          | 1          |           |           |          |            |          |            | 2         |            |            |           |            |           | 4           | 1           | 0           | 0           |
| Legionella species                      |                            |            |            | 1         |           |          |            |          |            |           |            | 2          |           |            |           | 3           | 1           | 1           | 1           |
| Leptospira species                      | 2                          |            |            |           |           |          |            |          |            |           |            | 1          | 1         | 2          |           | 6           | 7           | 7           | 3           |
| Measles virus                           |                            |            |            |           |           |          | 3          |          | 1          |           |            | 2          |           |            |           | 6           | 3           | 1           | 0           |
| Mumps virus                             |                            |            |            |           |           |          |            | 1        |            |           |            |            |           |            |           | 1           | 0           | 1           | 0           |
| Mycoplasma pneumoniae                   |                            | 8          | 3          |           |           |          | 4          |          | 5          | 4         | 12         | 4          | 2         | 3          | 3         | 48          | 36          | 50          | 53          |
| Neisseria gonorrhoeae                   | 7                          | 2          |            |           | 1         |          | 7          |          | 2          | 2         | 7          |            |           | 1          |           | 29          | 23          | 23          | 28          |
| Parainfluenza virus Type 1              |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| Parainfluenza virus Type 2              |                            |            | 1          |           |           |          | 4          |          |            |           |            |            | 1         |            |           | 6           | 2           | 0           | 0           |
| Parainfluenza virus Type 3              |                            | 1          |            |           |           |          |            |          |            |           |            |            |           |            |           | 1           | 0           | 1           | 18          |
| Parvovirus                              |                            | 1          |            |           |           |          | 1          |          | 3          |           | 1          | 1          | 1         |            |           | 8           | 12          | 31          | 18          |
| Pneumocystis carinii                    |                            | 1          |            |           |           |          |            |          |            |           |            |            |           |            |           | 1           | 1           | 1           | 1           |
| Respiratory Syncytial virus             | 4                          | 6          | 2          | 1         |           |          | 12         |          | 6          | 4         | 10         | 2          |           | 6          |           | 53          | 20          | 20          | 16          |
| Rickettsia - Spotted Fever Group        | 8                          | 2          |            |           |           |          | 1          |          |            |           |            |            |           |            |           | 11          | 6           | 3           | 5           |
| Ross River virus                        | 20                         | 7          | 2          | 8         | 4         |          | 5          |          | 8          | 14        | 9          | 12         | 3         | 14         | 18        | 124         | 81          | 92          | 52          |
| Rubella virus                           |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 1           | 1           | 1           |
| Salmonella paratyphi A                  |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| Salmonella paratyphi B                  |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| Salmonella typhi                        |                            |            |            |           |           |          |            |          | 1          |           |            |            |           |            |           | 1           | 2           | 1           | 0           |
| Shigella dysenteriae                    |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| Shigella flexneri                       |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| Streptococcus Group A                   | 10                         | 6          | 2          |           |           |          | 7          |          | 11         | 3         | 14         | 9          | 2         | 7          | 2         | 73          | 59          | 82          | 80          |
| Toxoplasma gondii                       |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 4           | 0           |
| Treponema pallidum                      | 26                         | 7          | 3          |           | 3         |          | 15         |          | 7          | 4         | 24         | 4          | 2         | 15         | 1         | 111         | 76          | 99          | 108         |
| Trichomonas vaginalis                   | 8                          |            |            |           | 1         |          |            |          | 1          |           | 1          |            |           |            |           | 11          | 7           | 11          | 17          |
| Varicella Zoster virus                  | 17                         | 20         | 11         | 4         |           |          | 30         |          | 21         | 6         | 32         | 15         | 3         | 7          | 6         | 172         | 143         | 191         | 158         |
| Yersinia enterocolitica                 |                            |            |            |           |           |          |            |          |            |           |            |            |           |            |           | 0           | 0           | 0           | 0           |
| <b>TOTAL</b>                            | <b>282</b>                 | <b>321</b> | <b>116</b> | <b>71</b> | <b>18</b> | <b>1</b> | <b>330</b> | <b>1</b> | <b>247</b> | <b>97</b> | <b>502</b> | <b>206</b> | <b>61</b> | <b>122</b> | <b>69</b> | <b>2444</b> | <b>1925</b> | <b>2617</b> | <b>2281</b> |

### REGIONS

|                              |                          |                           |                     |
|------------------------------|--------------------------|---------------------------|---------------------|
| 1 Cairns                     | 4 Mackay                 | 8 Northern Territory      | 12 Sunshine Coast   |
| 2 Gold Coast/Northern Rivers | 5 Mount Isa              | 9 Redcliffe               | 13 Toowoomba        |
| 3 Ipswich                    | 6 New England            | 10 Rockhampton            | 14 Townsville       |
|                              | 7 North Brisbane Suburbs | 11 South Brisbane Suburbs | 15 Wide Bay/Burnett |

February 2009 and further historical clinical data can be obtained by contacting your local Medical Liaison Officer