



# The Weekly Probe

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**Pathology labelling** – Over the last couple of weeks there have been several incidents where pathology tubes and samples have been incorrectly labelled and/or sent to pathology with another patient's request form.

It is each individual's responsibility to ensure that the correct patient, correct label and correct form are identified and sent. Try labelling, and checking of labels and request forms at patient's bedside to avoid the mixing of labels.

Please be vigilant – a couple of seconds of checking will avoid the minutes answering the phone call from pathology and the 5 minutes to retake, reorder and resend the sample.

## THIS WEEK

<b>Burns Blisters</b>
<b>Tinea and Cellulitis</b>
<b>Acute Coronary syndromes – 2016 NHF guideline</b>
<b>Joke / Quote of the Week</b>
<b>The Week Ahead</b>

## Burns blisters

The ACI have produced a consensus statement based on the opinion of burns experts at Westmead, Concord and RNSH re management of burns blisters. There is a link to this on the Intranet under Trauma with the other burns information.

### Recommendation:

The ACI Statewide Burn Injury Service (SBIS) recommends that:

- Appropriate analgesia must be administered prior to procedure (note that the blister itself is insensate yet pulling on the attached wound edge may produce some pain)
- Burn blisters **≤5mm** can be left intact
- Burn blisters **>5mm** should be
  - 'de-roofed' - use the disposable sterile scissors to trim the blister as close as possible to the surrounding edges
  - dressed appropriately with a non or low-adherent dressing

### Rationale:

'De-roofing' (removal of skin and fluid) burn blisters

- Allows assessment of burn wound bed
- Removes non-viable tissue
- Prevents uncontrolled rupture of blister
- Avoids risk of blister infection
- Relieves pain in tense blisters
- Reduces restriction of movement of joints

### However consideration should be given to:

- The risk/ benefit of 'de-roofing' small, non-tense blisters
- The risk/ benefit of removing blister skin when infection may occur (i.e. in remote area)
- The risk/benefit of 'de-roofing' blisters on the palmar surface of the hand and the plantar aspect of the foot
- Patient compliance with the procedure and on-going care when considering the management of small, non-tense blisters i.e. patients with dementia, learning difficulties, and toddlers

## Tinea + cellulitis patients

We often see patients with lower limb cellulitis who have concomitant tinea. This is often a portal through which the bacteria enter, so this should be treated simultaneously.

Andrew recently received some advice from a dermatologist who suggested Lamisil (Terbinafine) as it is a once daily treatment. They recommended clotrimazole cream for groin/more sensitive areas. They also made the suggestion (which should be passed on in the discharge letter) that the GP take skin scrapings /toenail clippings if it does not respond well to topical therapy as documentation is required before PO (griseofulvin) Rx can be commenced.

The other challenge for the GP is getting rid of the reservoir for re-infection. Viable fungal elements reside in shed skin flakes for prolonged periods, and often lead to re-infection once the initial disease is treated. Suggest the patient discards old shoes/slippers (especially if they wear them without socks), bleach showers/baths, and that other family members should also get examined/undergo therapy.

The other issues to address are vascular/venous disease, glycaemic control and leg elevation/compression and oedema control.

## Management of Acute Coronary syndromes 2016

The last time the National Heart Foundation of Australia and the Cardiac Society of Australia and NZ updated their ACS guidelines was back in 2011. Since then there has been a number of technological developments ( esp with high sensitivity troponins), research into various Accelerated Diagnostic Protocols (ADPs) using these hs-troponins and some changes with pharmacological agents.

Subsequently they have published their latest recommendations / guidelines for 2016 in the MJA, and Heart, Lung and Circulation (2016; 25:895-951). This is available from the [National Heart Foundation](#) if you want to look at the full document. Note one of the authors is Liverpool Cardiology Dept, giving the guideline some local endorsement.

### Key evidence based recommendations:

- **Oxygen** – routine use of oxygen is not recommended if O2 sats are > 93%. However care when using oxygen with CO2 retainers where aim is 88-92%
- **Initial meds** – aspirin should be given as a routine with suspected ACS if there are no contraindications. Other treatments such as antiplatelets should not be given without a confirmed or probable diagnosis of ACS.
- **ACS assessment protocols** – care should be guided by a evidence based ACS assessment protocol that includes formal risk stratification.
  - **The ADAPT or modified ADAPT** (Accelerated Diagnostic Protocol to Assess Patients with Chest Pain Symptoms using Contemporary Troponins) protocol is highlighted.

This tool identifies those at *low risk* of major adverse cardiac events (MACE) at 30 days on the basis of *negative troponins* AND *no ischaemic changes* on ECGs at 0 and 2 hours with a *TIMI score of ≤1* for modified ADAPT– 6/641 had MACE (NPV 99.1%)- (TIMI score of zero for ADAPT improved the NPV). Below is a reminder of the TIMI score

TIMI score
Age ≥65
≥3 risk factors for ACS; hypertension, hyperlipidemia, smoking, diabetes, family history
Use of aspirin in last 7 days
Prior coronary stenosis ≥50%
>2 angina events in 24 hours or persisting discomfort
ST-segment deviation of ≥0.05 mV on initial ECG
Elevated cardiac biomarkers

PS – what constitutes “family history” (premenstrual FHx is not specifically mentioned), do we include all ends of the diabetic spectrum, should we exclude the 50 pack year recently stopped ex-smoker and include the < 5 per day smoker. This is where it is difficult to strictly apply inclusion criteria used in trials to our clinical decision making and eliminating the “clinical “gestalt”

- **To access the ADAPTScore-** go through the Firstnet toolbar to CIAP (otherwise it won't work on the floor) then “Tools” - “MD calc” - type in ADAPT

- Other stratification tools exist for the stratification of such as the HEART score, the EDACS score (derived from the same group as the ADAPT yet incorporates age, sex, and discriminating historical features), TIMI and GRACE scores (see comment later on re the latter 2) . Below is a table taken from the Heart Foundation link regarding the utility, particularly in defining the low risk patient.

**Table 5 Performance of various risk scores and Clinical Assessment Protocols in the management of suspected ACS\***

Tool	Sens	Spec	PLV <sup>†</sup>	PLV <sup>‡</sup>	LR	Prevalence in study group	Reference
<b>High risk Mark 1 score (Positive (50/50/50))</b>							
ADAPT High risk	98	100	3	92	26	23	1,227
TIMI 1-7	77	95.4	37	74	1.5	1-75	[7,9]
GRACE >100	60	75	36	54	1.3	28%	[8]
ADAPT score 7-10					1.3		[10,11,12]
<b>Low risk Mark 1 score (Negative (50/50/50))</b>							
ADAPT Low	89	98	98	99	12	20	1.1
ADAPT score	90-100	98-100	90-99	4-24	0.15	21	0.47
GRACE <60	100	1	100	10	0.1	1-17%	[7,9]
GRACE PCI score	90-100	95-100	100	13	0.24	34%	[8]
ADAPT rule	98		98		1.1		[8,12]
<b>Low risk Suspected ACS APs (Negative (50/50/50))</b>							
ADAPT ADP*	100	25	100	19	0.14	20%	[13]
Unmodified ADP*	99	17-49	100	76-78	0.17	16-17%	[13,14]
ADAPT Pathway**	99	100	100	100	0.24	30	20%
ADAPT**	99	100	97-99		0.11	17	11
ADAPT (age cut-off)	100	21.9	100		0	18%	[15]
TIMI ADP	99	40	100	14	0.25	40%	[20]
HEART	97	79	97	96	0.10	17%	[9]

\* In all values are rounded to nearest whole number  
 † Positive Likelihood Ratio  
 ‡ Negative Likelihood Ratio  
 \* Risk was modified from Ferrero et al. (2010) Patient With Chest Pain Have Acute Coronary Syndrome: The National Clinical Examination Study

- PS- All these tools can be accessed through the same MD calc link
  - **Timing of Troponins-** controversial topic. They suggest:
    - Single troponin for :
      - Patients in whose pain and symptoms resolved 12 hours prior to testing
      - Patients in whose symptom onset was > 3 hrs AND the troponin was less than the level of detection (ie < 5ng/L)
    - Two troponins – depends on the validated algorithm that you are using
      - If using ADAPT - 0 & 2 hrs
      - If using HEART - 0 & 3 hrs
      - Note that there a number of articles which point out a negative troponin rules out / rules in **AMI** at 0 and 1 hr – However they are NOT related to the 99<sup>th</sup> percentile eg one study used the levels of < 5 on arrival and a increase of < 2ng/L to rule out AMI
  - **Non-invasive testing-** recommended in intermediate risk patients if normal troponins, ECG and if they remain symptom free
    - **Timing** – it is suggested that for intermediate risk patients testing should be done within 7 days ideally, but can be done up to 14 days post presentation (previous recommendation for 72 hrs)
    - **Low risk** – patients in whom no further objective testing is required are those at low risk as defined by a validated ACS assessment protocol – age < 40yo, symptoms

atypical for angina, in the absence of known coronary artery disease, with normal tropon and ECGs who remain pain free (weak evidence – level IIIb)

- **Other risk stratification tools**

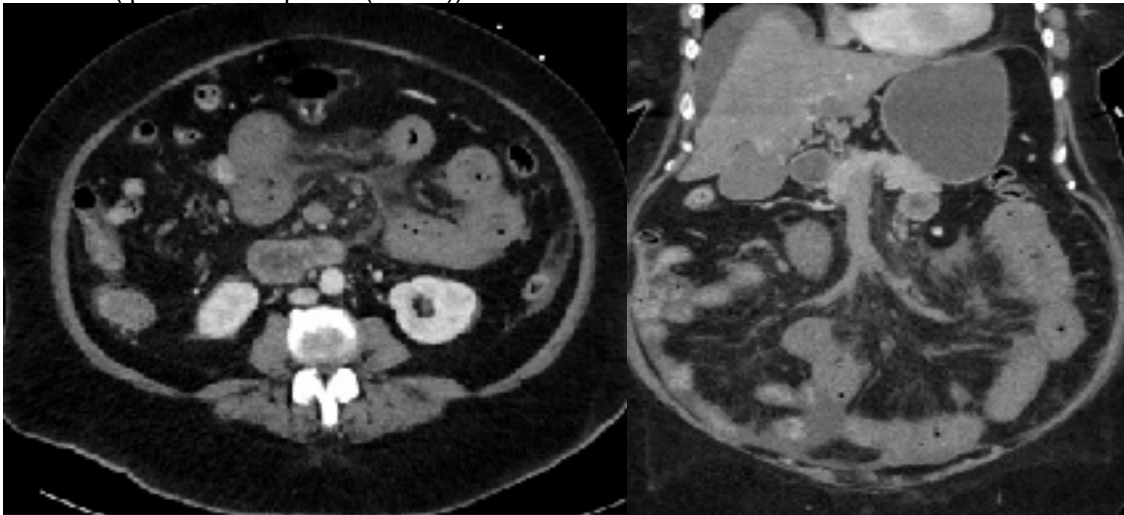
- They mention that “ the routine use of validated risk stratification tools for ischaemic or bleeding events (eg GRACE for ischaemic risk and CRUSADE score for bleeding risk ) may assist in patient-centric clinical decision making in regards to ACS care”
  - However remember that GRACE looks at mortality risk as an inpatient, 3 months and 6 months for those diagnosed as ACS rather than the deciding which of the undifferentiated ED patients has ACS. Try this [link](#)
  - Likewise the TIMI score is derived from a study conducted from 1994-1998 (pre-troponin days) and stratifies the 14 day risk of death, MI, and urgent revascularization in those defined as unstable angina or NSTEMI ie not our undifferentiated mix of low- intermediate – high risk patients.
- Therefore they may guide treatment and the urgency of investigations once the diagnosis has been made – ie the patient has been referred to cardiology for followup / workup

- **AMI Management-** most of the treatments are determined by the inpatient cardiology staff. However couple of points

- Primary PCI preferred if it can be done within 90 minutes.
- P2Y12 inhibitors –choice will depend on preference yet due to superior efficacy ticagrelor and prasugrel are preferred first line agents (over clopidogrel). For more info check out the article which looks at the pros and cons of the different agents as agent choice will depend on age, patient weight , Hx of TIA / CVAs, , asthma / CAL, previous clopidogrel use , if taking oral anticoagulants- in particular withhold if any concern re need for CABGs (extensive ECG changes, haemodynamic instability, ongoing ischaemia)
- Other information is included on heparin / enoxaparin, glycoprotein 11b/ 12a inhibitors (eg tirofiban)

## NEXT WEEK'S CASE

68yo diabetic lady presents with 5 days of upper abdominal pain and distension on a background of HT'n, hyperchol and DM  
Obs NAD- hydration N  
Abdo soft yet tender upper abdo with no peritonism- ECG – sinus – lactate 10.1 – WCC 33  
CT abdo-( portovenous phase ( a hint)) below



What is going on?



Please forward any funny and litigious quotes you may hear on the floor (happy to publish names if you want)

**THE WEEK AHEAD**

*Tuesdays - 14:30 – 15:30 Intern & JMO teaching -Thomas & Rachel Moore*

*Wednesday- 0800-0900 Critical Care Journal Club. ICU Conf Room / 14:30 – 15:30 Intern & JMO teaching -Thomas & Rachel Moore*

*Thursday 0730-0800 Trauma Audit. Education Centre / 0800-0830 MET Review Education centre / 1300-1400 Medical Grand Rounds. Auditorium.*