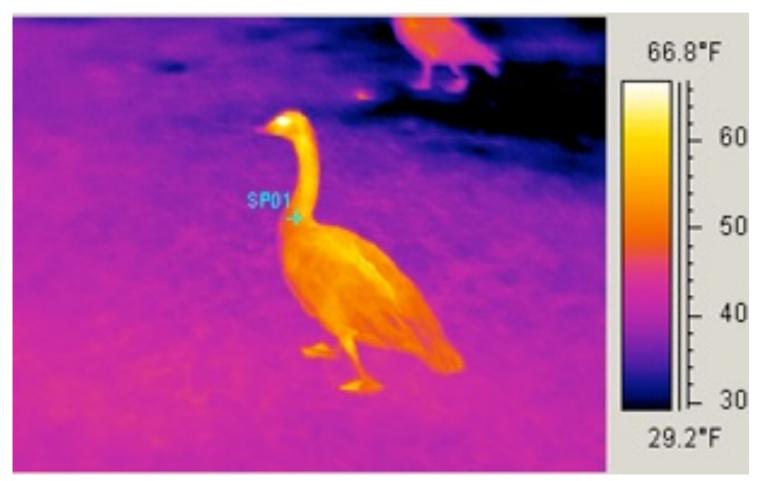
RGEM BLOGS BOOK 2

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TYMPANIC THERMOMETERS WORK BY INFRARED

OVERVIEW

It's probably fair to say that few of us give any great thought on how a temperature is measured. I admit its always amusing when the new start student nurse puts the temperature in the respiratory rate box but rarely do we consider the accuracy of the number presented to us.

A fever catches our eye in Emergency Medicine, it often radically changes the diagnostic pathway, treatment and disposition of our patients. An 80 year old with a temp of 39 is a different kettle of fish from an 80 year old with a temp of 37.

A reasonable number of us could cite a sensitivity and specificity of a CT at 6 hrs for ruling out SAH or the sensitivity of the newest high sensitivity troponin T but I doubt any of us could cite a sensitivity or specificity for one of the most commonly used devices in our ED's.

She has also been given haloperidol which is contra-indicated in Parkinson's.

This patient urgently needs her PD medications. These will now have to be given via nasogastric (NG) tube since the patient is unable to swallow. This must be done as a priority. A transdermal patch (of rotigotine) is available if the NG is not possible, but the first line option is to give her usual medications urgently via NG.

It is also important to check the patients CK. Not only has this patient had a long lie, but people with Parkinson's can develop a Neuroleptic Malignant Syndrome like disorder if their medications are suddenly stopped or if they are given a dopamine antagonist such as haloperidol, as in this case.

So what is the sensitivity and specificity then?

Lets just say at this stage of the blog that the diagnostic characteristics of the tympanic thermometer used in your ED is somewhat poor. Ah.. such impatience..First a little, needless, background.

To many of us in the sepsis-sphere, the English word fever probably stems from the latin root hypoTazocinaemia and its use in the modern ED has likely triggered more sepsis alerts for 19 year olds with tonsillitis than any other vital sign in the known world.

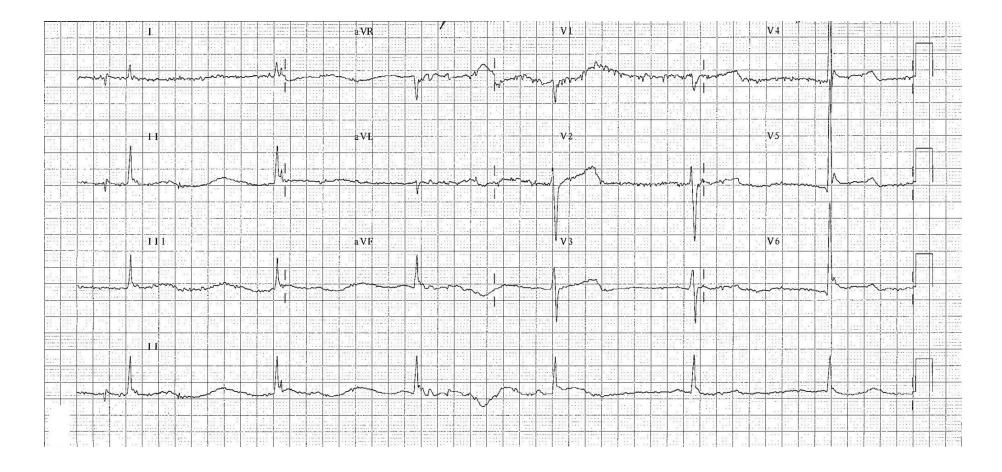
Temp Taking and How to Take It

So how can we measure peoples temperature?

Probably the best way to do so is via some kind of intravascular catheter or some other catheter lying deep within some body cavity unspeakable in polite company.

In trials looking at this subject, reference standards include pulmonary artery catheters, oesophageal catheters and urinary catheters. The most commonly used central temperature Ive seen used in the ED is the rectal thermometer.

And think back to the last time you saw a rectal temp recorded in your ED – was it that 80 year old on New Years day who'd been lying on the kitchen floor for 9 hours with a broken hip and an ECG like this?



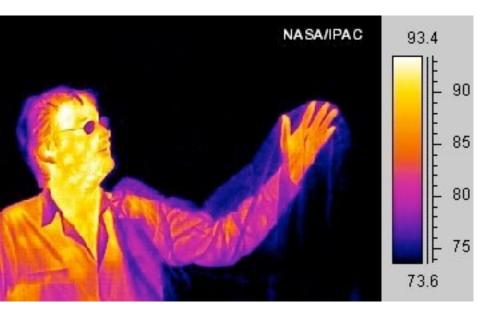
LITFL ECG library

This seems to be a fairly accepted and commonly practised indication for abandoning the humble tympanic thermometer and searching the cupboards for the rectal thermometer that gets somewhat awkwardly placed in a bit of frozen poo in the rectum.

How do thermometers work?

Honestly I would have said "pixie dust" if asked this in an exam but it turns out it's more scientific than that.

Tympanic thermometers work by infrared.



They measure the infra-red spectrum of light emitted by the tympanic membrane. As the tympanic membrane gets warmer it emits a different frequency of infra-red light. The machine correlates the infra red light with a temperature and Robert's your Mother's Brother you have a temperature reading.

Rectal thermometers, and I think most central thermometers, work by a "thermocouple" (Not two hot people on a date...) and utilise the <u>Seebeck</u> effect.

The problem isn't actually the tympanic method, it's probably because the ear is too peripheral to accurately reflect the central temperature. In the same way that many patients with severe sepsis and high central temperatures have cool peripheries so many patients with high central temperatures have relatively cold tympanic membranes.

Think the opposite applies for those with otitis media getting their temp measured tympanically? Think again, apparently, as studies have shown that having a red, bulging tympanic membrane does not give you a different tympanic temp than if it was taken via the axilliary route.

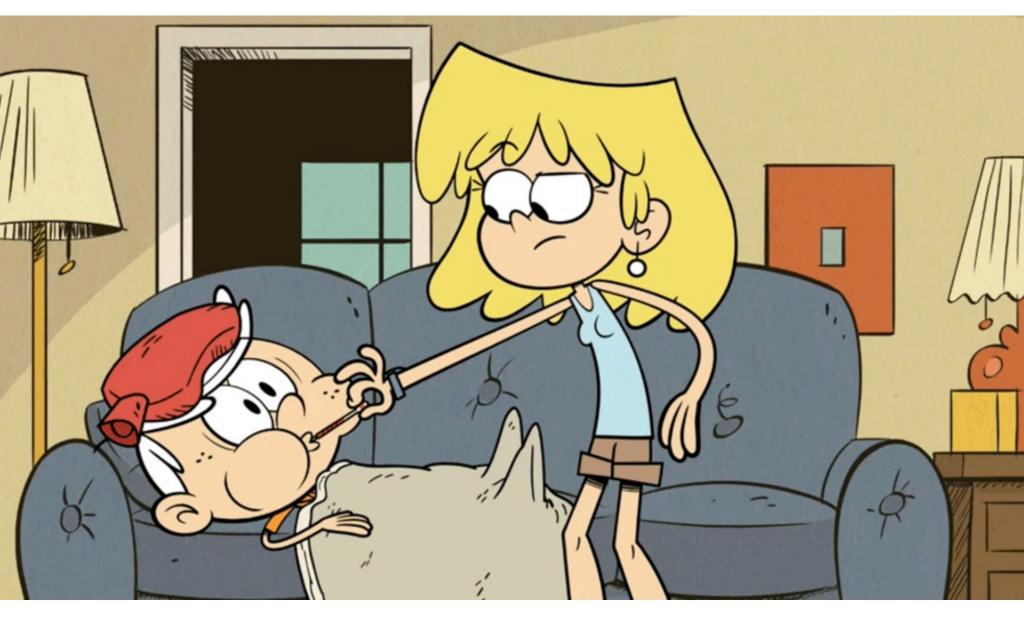
However, be cautious with tympanic thermometers in the very young. The rule is simple: if it doesn't fit in their ear canal, then it's not likely to be accurate.

And if parents tell you they own a disposable forehead thermometer - the kind which have colour codes for different temperature ranges- please do encourage them to invest in a more accurate thermometer while they have a child (unless they want to risk a PUO - pyrexia of unknown origin- screen).

So what are the diagnostic characteristics of the non-invasive thermometers then?

Well, according to one of the <u>most recent systematic</u> <u>reviews and meta analyses</u> on the subject the sensitivity is 64% and the specificity is 96%.

The reported range of inaccuracy was plus or minus 1.5 degrees.



"Hmm..That's 37.5 degrees more or less 1.5 degrees, so you may or may not have a temp.. useful!"

This was actually a fairly nicely done study, with a well defined search strategy and appropriate inclusion and exclusion criteria (prospective studies only, all had to have a clear "gold standard" comparator, ie rectal, oesophageal, endovascular temp etc...) and it's by no means the first paper to question the use of non invasive techniques for measuring temperatures.

What does that mean for my practice then?

If your tympanic thermometer reads 39 degrees then it's probably true. It's a very specific test.

However if your tympanic thermometer reads 37.5 degrees then it doesn't mean a great deal. The patient may well have a temperature and you've

just missed it with your infra-red duck/goose detecting machine.

So in short, a peripheral temperature is good as a rule-in test for fever/hypothermia, but not so great as a rule-out.

Perhaps the best comparison would be the FAST examination in trauma. All of us who have done our level 1 ultrasound competencies will be able to recognise that a positive FAST examination is probably reflective of reality - the patient probably does have free fluid in the abdomen - it's a fairly specific test. However we're all savvy enough to know that the FAST exam in trauma will often show no free fluid even when in reality there is free fluid there- in other words it's not a very sensitive test.

Temp Taking and How to Take It

In the situation of trauma, if we have a reasonably high pre-test probability that the patient has a serious injury we don't just trust the poorly sensitive FAST exam, we go on and arrange a more definitive test like a CT.

So maybe, when we have an 80 year old who we're sending to the medical team with the oh so unsatisfactory label of "collapse query cause" or "acute confusion" when we were suspicious of an infectious cause we should really consider getting the definitive test and getting a rectal temperature.

P.S. A non-scientific word of warning here for those planning on changing practice in their work up of patients: If you suddenly decide to request rectal temperatures on every punter who rolls through the door with a "normal" temperature on your tympanic be prepared for some very poor multi source feedback for your eportfolio and perhaps you better invest in a new Nespresso machine for the tea room and learn from Cliff Reid's tips on how to make things happen.

References:

- Accuracy of peripheral thermometers for estimating temperature: a systematic review and meta-analysis.
- International Journal of Paediatric Otorhinolaryngology: Infrared transtympanic temperature measurement and otitis media with effusion
- LITFL Hypothermia ECG
- Cliff Reid, Making things Happen, SMACC 2013
- Don't Forget the Bubbles

UPDATE:

I just found this paper published in print in the EMJ just now. It's an observational trial looking at temp measurement and it found just what we all expected it to find. "None of the non-invasive methods met benchmarks for diagnostic accuracy using the criterion of 38°C to detect rectal temperature of 38°C"





DAWN TRIAL

Peer review: Richard Pullicino Clinical Fellow in Interventional Neuroradiology

Patients who wake up with a stroke may still be eligible for endovascular thrombectomy, according to preliminary results from the DAWN trial.

Presented in the European Stroke Organisation Conference (ESOC17) last month, the principal investigators for the trial said they found an impressive 2-point difference in the 90-day weighted modified Rankin Scale (mRS) score of 48.6% in the thrombectomy group, compared to 13.1% in the control group who received medical treatment alone. This translated in a number needed to treat of 2.8.

However, study thrombectomies were performed using the Trevo device, a product of Stryker Medical who funded the trial, which critics may analyse as a likely conflict of interest.

The aim of the study was to demonstrate superior functional outcomes in patients with a large vessel (specifically, the intracranial ICA and M1 segment of MCA) acute ischaemic stroke at 90 days with thrombectomy and medical management compared to medical management alone, in a select subgroup of patients treated 6-24 hours since they were last seen well.

A new DAWN for Stroke Patients

To understand what the trial was about, here is a PICO:

DAWN Trial

Population

Patients with acute ischemic stroke due to large vessel occlusion presenting 6-24hrs since they were last seen well, who had clinical imaging mismatch (defined by age, core and NIHSS)

Intervention

Mechanical embolectomy plus medical management

Control

Medical management only

Outcomes

Modified Rankin Scale score at 90 days & stroke-related mortality at 90 days

The international trial was stopped prematurely after a planned interim review of 206 recruited patients had shown that the trial's aims were reached successfully. In their <u>youtube interview</u> at the ESOC17, Tudor Jovin and Raul Nogueira, the principal investigators of DAWN, explained that due to lack of previous data for this population group, they had difficulty with a sample size calculation and therefore had planned interim looks into the data. You can see them present the results from this prospective, randomized, multicenter, Bayesian adaptive-enrichment, open label trial with blinded endpoint assessment here:

Without actually having access to the trial results which are pending publication, it is difficult to be able to critically appraise them, but they do seem promising. A 73% relative reduction of dependency in activities of daily living and a number needed to treat for any lower disability of 2.0 for those in the thrombectomy group will mean that the intervention is going to have a massive benefit for those people who present late with a large vessel stroke and challenges conceptions that reperfusion outcomes are inversely related to time.

"Time is still obviously very important and the earlier the treatment is given the better the results, but we have shown in this trial that it shouldn't be the only factor that determines whether we consider endovascular therapy," Tudor Jovin said.

In their later presentation at LINNC17 this month, Raul Nogueira explained that a physiologic rather than timebased only approach may be the reason behind the study's results as patients with a Clinical Core Mismatch e.g. significant clinical deficits but limited infarct size may have compensatory mechanisms for ischaemia, like collateral flow.

With regards to safety outcomes, the results remain impressive, with a 4.8% symptomatic haemorrhage rate which the investigators claim is non-significant compared to the control groups and data from previous trials. Stroke related deaths, they claim, is lower in the treatment group too.

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A new DAWN for Stroke Patients

What does this mean for me?

At the moment, as an emergency physician not very much. In the near future, for certain stroke patients and the physicians that treat them, it is likely to be life-changing.

It may mean that many stroke patients who present within 24 hours with a large-vessel occlusion will need to be screened for treatment with thrombectomy with CT perfusion or MRI-diffusion-weighted imaging, with the infarct core volume measured by automated software. This will see hospitals having to offer a 24 hour perfusion-imaging service together with a round-the-clock endovascular team.

Experts have estimated that if implemented, this may mean a 2-5% increase in patients eligible for thrombectomy, but how this translates in terms of service provision can be higher or lower depending what this is like in your neck of the woods. However, with evidence pointing towards a 73% relative reduction of dependency in activities of daily living (ADLs) and a 35% absolute increase in the number of patients achieving functional independence (mRS score, 0 to 2) in the thrombectomy group, it will be difficult to argue against implementing the new practise.

Speaking to Medscape after the conference Professor of neurology, Andrew Demchuk, said, "Stroke is a very heterogeneous disease and so in order to be successful in treatment we have to classify patients into the correct subtypes who may benefit the most and they did a masterful job of doing that in this trial."

"Patients with a poor clinical score have had large strokes, but if they have a small core infarct size they tend to have a long time window - - the stroke expands very slowly. By selecting patients this way, they have achieved a large effect size with thrombectomy," he pointed out.

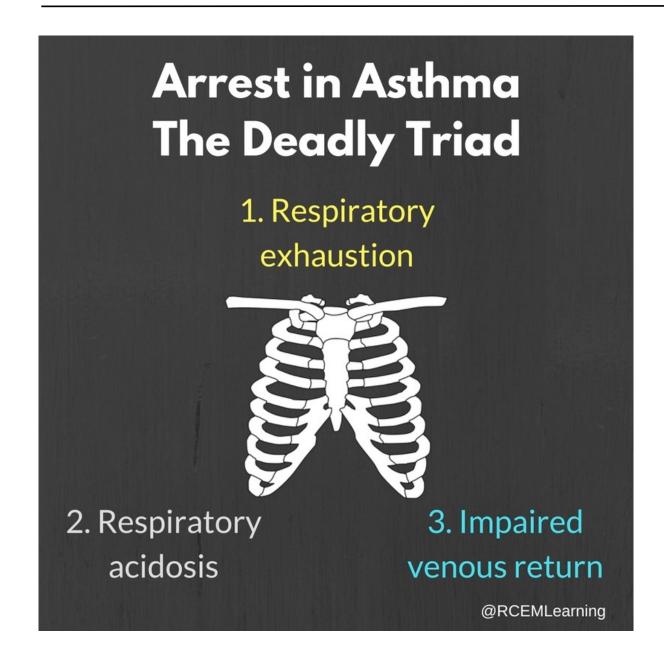
Further reading/resources:

- Clinical trials.gov: DAWN Trial
- Endovascular today: DAWN Trial Results
 Presented for Stryker's Trevo Retriever
- Youtube: Tudor Jovin and Raul Nogueira DAWN
 Trial at ESOC 2017
- Medscape: DAWN: Thrombectomy Effective Up to 24 Hours After Stroke



ARREST ASTRIBUTES TO A STRING THE STRING THE

Author: James Yates / Codes: CC17, CC23, CC24, CC25 / Published: 16/10/2017



In the UK, there are 3 asthma-related deaths per day. In 2015, this translated to 1468 deaths in that year.

Many of these patients will have their cardiac arrest in the pre-hospital environment, whilst others will present to the emergency department with a deteriorating acute attack.

It is therefore crucial that we get the treatment right for the 138 patients per day that visit our UK emergency departments with an acute exacerbation of asthma - regardless of the severity.

A peri-arrest asthma exacerbation can be a nightmare scenario to manage if you aren't prepared. There is an, often young, otherwise healthy patient dying in front of you, so knowing the best approach to take is critical.

Initial Treatment

As a Specialist Paramedic, I think it is important to highlight that prehospital practice mirrors the BTS/SIGN guidelines almost exactly and the severity of the attack will have been classified using the same criteria. As such, a patient suffering from a life-threatening asthma attack will be receiving back-to-back nebulisers of salbutamol and a single dose of 500mcg ipratropium will have been added to one of these. An intravenous dose of 100mg hydrocortisone should also have been given.

No surprises so far

The IM Adrenaline & Nebuliser via T-Piece Controversy

However, the ambulance crew may have given the patient 0.5mg IM adrenaline, which does not feature in the BTS guidelines. This treatment is included in the ambulance guidelines, in part, due to the chance of misdiagnosing anaphylaxis and therefore the chance of inappropriately withholding adrenaline from this cohort. It also represents a relatively safe final treatment option for those patients with failing ventilation, in whom inhaled bronchodilators may be ineffective but who still urgently require bronchodilation. Interestingly, the UK Resuscitation Council suggest that IM adrenaline can be used as a first line drug in asthmatic cardiac arrest if IV access cannot be rapidly gained, stating that the benefit is uncertain but it is unlikely to be harmful.

In those patients who have deteriorated further, into a respiratory or cardiac arrest, the ambulance crew may be utilising in-line nebulisation with a T-piece attached to an airway or directly to the resuscitation bag. However, the practice of using in-line nebulisation with an endotracheal tube was recently removed from the ambulance guidelines due to the theoretically increased risk of a tension pneumothorax (it is postulated that the 6-8L/min of oxygen driving the nebuliser may create a high degree of PEEP which would lead to increased gas trapping in the alveoli, worsening the efficacy of ventilation and reducing venous return through the increased intrathoracic pressure.

Add this trapped volume to the positive pressure tidal volume from a BVM and this also increases the chance of barotrauma and subsequent tension pneumothorax).

Resus Room Management

So, the patient is wheeled into your resus bay with a life-threatening asthma attack despite the best efforts of the prehospital team.

Where do you go from here?

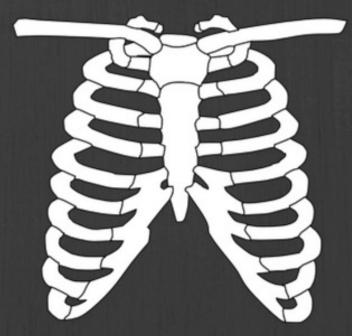
The BTS guidelines advocate a single bolus of IV magnesium 1.2-2g over 20mins for all asthma patients who present with a peak expiratory flow of <50% and who have not had a good initial response to nebulised bronchodilators. Our life-threatening case definitely falls into this category. Magnesium is pretty safe to give, with hypotension being the main side effect. You don't even need to know what the magnesium level is before giving the first dose! Nebulised B2 agonists can be continued, but these are unlikely to be delivered successfully to the target tissues, and therefore intravenous salbutamol should be considered. In some trusts this next step may be replaced with aminophylline. The adult bolus dose of salbutamol is 250mcg IV which can be repeated if necessary, although for a prolonged attack these bolus doses can be replaced with an infusion. If you"re using high doses of intravenous salbutamol it's worth considering monitoring the serum lactate and potassium levels. Dilulting salbutamol can be tricky - there's a great checklist from Brighton here.

Impending Doom

Some patients will respond well to these additional interventions, but for others a continued deterioration into cardiac arrest may be the unfortunate trajectory.



1. Respiratory exhaustion



2. Respiratory acidosis

3. Impaired venous return

@RCEMLearning

Cardiac arrest is ultimately the result of prolonged respiratory exhaustion, respiratory acidosis and impaired venous return due to the increased intrathoracic pressure.

As soon as cardiac arrest is identified Advanced Life Support should be initiated as normal but, as the resuscitation attempt gets established, there a few adaptions that may be required.

1. Intubate Early

Firstly, due to the high inflation pressures, this is one group of patients that should definitely be managed with a tracheal tube. Depending on the supraglottic device used these begin to leak at pressures around 20-30cmH2O and in an asthmatic patient. pressures exceeding this are not uncommon. Using a supraglottic device may therefore result in hypoventilation of a patient who is already critically hypoxic and hypercapnic. These increased inflation pressures are also higher than that of the lower oesophageal sphincter, increasing the chance of gastric inflation, regurgitation and aspiration, unless the airway is secured with an ETT.

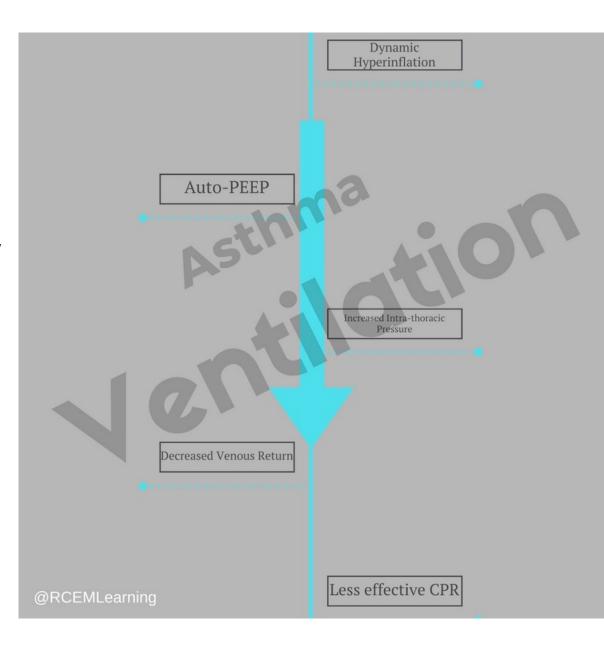
2. Ventilate with Caution

With the airway secured attention should turn to an appropriate ventilation strategy. The respiratory rate advocated by the European Resuscitation Council is only slightly lower than normal, at 8-10 breaths per minute, with a tidal volume just enough to cause normal chest rise.

The reason for this is due to the potential for dynamic hyperinflation. This occurs when the expiratory time is not sufficient to allow complete exhalation. This results in a gradual increase in the residual volume in the alveoli, to the point at which the inspiratory reserve volume becomes minimal and further ventilation almost impossible.

This auto-PEEP, as well as affecting ventilation, also causes a significant rise in intrathoracic pressure, impeding venous return and reducing the efficacy of chest compressions.

Dynamic hyperinflation may be identified through an increasing resistance to ventilation, poor excursions of the chest wall and potentially a hyper-inflated appearance to the chest.



3. Manual Chest Deflation

Correction of the condition can be achieved by disconnecting the ETT and applying manual pressure to the patients' chest. Air being forced out of the lungs may well be heard escaping from the ETT. If dynamic hyperinflation was the cause of difficult ventilation, an immediate improvement will be felt on reconnecting the BVM. This manual deflation technique may have to be repeated throughout the resuscitation attempt if the condition redevelops.

4. Think about Tension

An alternative cause of difficult ventilation is tension pneumothorax. If manual deflation of the lungs does not result in a reduction in the airway pressures, then tension pneumothorax should immediately be considered. A tension pneumothorax can be very difficult to identify in cardiac arrest but in asthmatic

Arrest Asthma

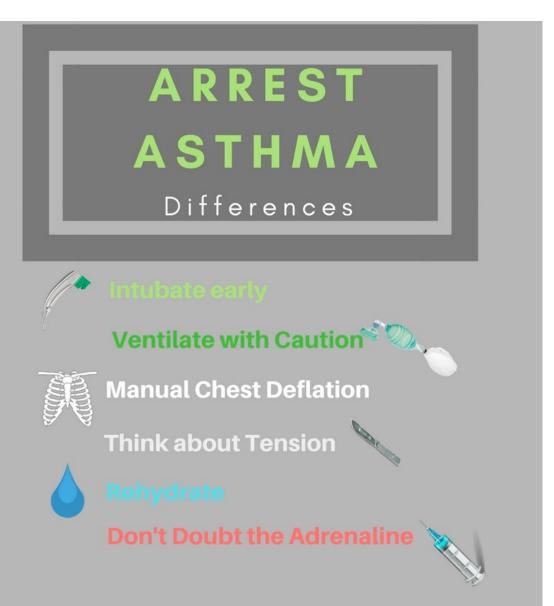
patients a low index of suspicion should be maintained, particularly if signs such as subcutaneous emphysema are noted. Decompression should be completed with a thoracostomy rather than needle decompression as multiple studies have highlighted concerns that a standard 14G cannula will not have an adequate length to penetrate the thoracic wall, as well as having a number of other limitations. It should be remembered that decompression may be required bilaterally, but a quick review of the patient should be carried out after the first thoracostomy to ensure a second invasive procedure is not carried out unnecessarily.

5. Rehydrate

Dehydration can often be a finding in these patients' due to the increased insensible losses associated with prolonged tachypnoea and mouth breathing. In the cardiac arrest setting this dehydration can have a couple of detrimental effects. Firstly, any degree of dehydration and reduction in intravascular volume is going to compromise effective chest compressions, particularly when found in conjunction with dynamic hyperinflation. Second, it is hypothesised that the mucus being secreted into the airways becomes thicker and therefore more likely to plug the respiratory bronchioles and small airways. This can worsen the, already significant, V:Q mismatch as well as contributing to dynamic hyperinflation. As a result, IV fluid during an asthmatic arrest may provide some benefit to these patients.

6. Don't Doubt the Adrenaline

Finally, with the current equipoise surrounding the use of adrenaline during cardiac arrest, it's worth highlighting that this is one group of patients in which it may have positive effects. A regular and large dose of a non-selective, B2 agonist definitely has sound, theoretical, physiological benefits.



So, in summary, take a breath! Secure the airway with an endotracheal tube and watch for changes in the compliance of the BVM. Manually deflate the chest if there is resistance to ventilation and if there's no improvement after, then decompress the chest. Get the IV fluid running and push the adrenaline early and often.

James Yates is a Specialist Paramedic (Critical Care) with the Great Western Air Ambulance based in Bristol. In his prehospital career to date, James has worked nationally and internationally in a range of clinical and educational roles. He has an interest in education and simulation.

Further Reading

- BTS/SIGN Asthma guidelines
- The Resus Room: Roadside to Resus series: Asthma