**Cardiac Arrest – Journal Summaries**

9/7/7

**POST-CARDIAC ARREST CARE**

Bernard SA, *et al*. *“Treatment of comatose surivors of out-of-hospital cardiac arrest with induced hypothermia.”* N Engl J Med 2002; 346:557-563

- RCT

- n = 77

- patients who remained unconscious after resuscitation from OOHCA due to VF.

- hypothermia = cooled to 33 C within 2 hours and maintained there for 12 hrs

-> significant improvement in neurological outcome in cooled group.

Hypothermia after Cardiac Arrest Study Group. N Engl J Med 2002: 346:549-556

- multicentre study

- n = 136

- coolled to 32-34 C for 24hrs within 4 hrs

-> same results as above

Hope, J. *et al* (2010) “*Association Between Arterial Hyperoxia Following Resuscitation From Cardiac Arrest and In-Hospital Mortality*” JAMA 303(21): 2165-2171

- hypothesis = that post-resuscitation hyperoxia is associated with increased mortality.

- too little oxygen -> hypoxic brain injury

- too much oxygen -> O2 radical production triggering cell injury and apoptosis

- multicenter co-hort study

- 120 hospitals

- n = 6326 patients

- inclusion criteria: adults, nontraumatic cardiac arrest, CPR within 24 hours prior to ICU arrival, ABG analysis performed within 24 hours following ICU arrival

- patients divided into 3 groups based of PaO2 – hyperoxia (>300mmHg), hypoxia (<60mmHg), or noroxia (between)

-> significantly higher in-hospital mortality (OR = 1.8)

- not cooled

Bernard, S *et al* (2010) “*Induction of Theraputic Hypothermia by Paramedics After Resuscitation from Out-of-Hospital Ventricular Fibrillation Cardiac Arrest – A Randomized Controlled Trial*” Circulation, April 2010

RICH Investigators – Rapid infusion with Ice Cold Hartmans

- n = 234

- 2L ice cold Hartmans pre hospital (with midazolam and pancuronium) then a further 2L in ED vs standard cooling to 33 C for 24 hours

-> favourable outcome 47% in treatment group vs 53% in hospital cooling group

-> RR of favourable vs non-favourable outcome = 0.89 (p 0.433)

-> patient were 0.5 C colder who were treated in the field but there was no difference between groups @ 30 min.

**PROGNOSTICATION POST CARDIAC ARREST**

Zandbergen, E.G., *et al* (1998) “*Systematic review of early prediction of poor outcome in anoxic-ischaemic coma*” Lancet 352:1808-1812

- 33 studies

- 14 prognostic variables looked at

- 3 variable had a specificity of 100% for poor outcome:

1. absent pupillary reflexes @ day 3

2. absent motor response to pain (worse than withdrawal) on day 3

3. bilateral absence of early cortical SSEP within the first week

- other poor prognostic factors = an isoelectric EEG, burst suppression, myoclonus on stimulation.

Fugate, J. E. *et al* (2010) “*Predictors of Neurological Outcome in Hypothermia after Cardiac Arrest*” Ann Neurol 68:907-914

- the predictive value of neurological prognostic indicators for patients treated with hypothermia after surviving cardiac arrest is unknown.

- prospective co-hort study

- single center study (US)

- n = 192 (103 hypothermia, 89 nonhypothermia)

- June 2006 to October 2009

- information gathered at 72 hours:

- clinical examination (pupillary light reflex, corneals, extensor or absent motor response)

- SSEP’s

- EEG

- outcome measure: in-hospital mortality

-> clinical examination (brainstem reflexes, motor response, myoclonus) remained an accurate predictor after therapeutic hypothermia

-> myoclonic status invariably associated with death

-> malignant EEG patterns (burst-suppression, generalized suppression, status epilepticus and non-reactivity) associated with death.

-> serum neuron specific enolase (NSE) > 33ng measured at 1-3 days after cardiac arrest are associated with a poor outcome but has a high false-positive rate.

-> CT showing global cerebral oedema associated with death.

-> sedative medication and liver/renal dysfunction can alter examination accuracy

*Strengths*

*Weaknesses*

- single center

- non-hypothermia group (non-VF or inhospital)

- residual sedatives may have compounded examination findings

**MET TEAMS**

Hillman K, et al. MERIT study investigators. Introduction of the medical emergency team (MET) system: a cluster-randomised controlled trial. Lancet 2005: 365:2091-2097

- multi-centre

- cluster randomised control trial

- 23 Australian Hospitals (12 MET trained and implemented, 11 did not)

- training (2 months), implementation (4 months), study period (6 months)

- n = 741,744

- primary outcomes: incidence of cardiac arrests, unplanned admissions to intensive care units and death.

-> significant increases in emergency team calls

-> no significant change in cardiac arrest, unplanned ICU admissions or deaths

- problems:

-> observations were not frequent enough

-> MET teams were not called when poor observations were measured

-> there was a substantial decreased in unexpected deaths in both groups during the study period (contamination) -> 30% reduction!

-> under powered as base line event rate was assumed to by 30 per 1000 when it was actually 7 per 1000.

Post-hoc Analysis of the MERIT Study (Critical Care Med. 2009 Jan;37(1):349-50):

- examination of the relationship between early emergency teams calls and incidence of serious adverse events (not associated with cardiac arrest or death)

-> as the proportion of early emergency teams calls increases -> the rate of cardiac arrest and unexpected death decreases.

**PHARMACOTHERAPY IN ARREST**

Wenzel V, et al. A comparison of vasopressin and epinephrine for out-of-hospital cardiopulmonary resuscitation. N Engl J Med 2004; 350:105-113.

- triple blinded multi-centre randomised trial

- n = 1219

- initial vasopressin (40IU) vs adr (1mg) then increments of adr

-> rates of admission unchanged

-> higher survival to hospital admission for patients resuscitated with vasopressin from asystole

Olasveengen, T. M., *et al* (2009) “*Intravenous Drug Administration During Out-of-Hospital Cardiac Arrest: A Randomized Trial*” JAMA 302 (20):2222-2229

- RCT

- Norwegian

- 2003-2008

- n = 851

- ACLS with IV drug administration vs ACLS and no drug administration

- primary outcome = survival to hospital discharge

- secondary outcomes = 1 year survival, survival with favourable neurological outcome, hospital admission with ROSC, quality of CPR (chest compression rate, pauses, ventilation rate)

- inclusion criteria: > 18 years, non-traumatic, OHCA

- exclusion criteria: cardiac arrest witnessed by ambulance crew, resuscitation initiated by physicians, cardiac arrest induced by anaphylaxis or asthma

-> increased short term survival in IV drug group

-> no difference to survival to hospital discharge, quality of CPR or long term survival

*Weaknesses*

- 3 min of CPR prior to defibrillation in VF

- 10% of no drugs group received drugs during resuscitation

- not powered correctly

**COMPRESSION ONLY CPR**

30/1/10

Rea, T.D. *et al* (2010) “*CPR with Chest Compression Alone or with Rescue Breathing*” NEJM, 363:423-33

- MRCT (Washington and London x 2 sites)

- chest compressions only vs chest compression + rescue breathing

- primary outcome: survival to hospital discharge

- secondary outcomes: favourable neurological outcome, ROSC

- inclusion criteria: >18 years, OHCA, dispatcher initiated CPR instruction to bystanders

- exclusion criteria: all ready having CPR, trauma, drowning, asphyxiation, less than 18 yrs, DNR orders

- n = 1941

-> no difference in survival

-> no difference in favourable neurological outcome

-> trend towards increased survival in chest compression alone group in those who had a cardiac arrest and a shockable rhythm

-> bystanders more likely to perform CPR than rescue breathing

*Criticisms*

- one site unable to provide neurological status at discharge!

- despite having nearly 2000 patient it required 4200 to obtain 80% power

Svensson, L. *et al* (2010) “*Compression-only CPR or Standard CPR in Out-of-Hospital Cardiac Arrest*” NEJM 363:434-42

- RCT

- n = 1276

- swedish

- compression only CPR only vs Standard CPR

- primary end point = 30 day survival

- secondary end points = 1 day survival, first detected cardiac rhythm, survival to discharge from hospital

- inclusion criteria: witnessed, unconsciousness, abnormal or no breathing

- exclusion criteria: arrest caused by: trauma, airway obstruction, drowning, intoxification, age < 8, difficulty communicating, no CPR started, knowledge of how to perform CPR

-> no difference between the groups in all aspects

*Criticisms*

- some differences between baseline characteristics of groups – standard CPR group (younger)

- 113 patients assigned to compressions only got ventilation

- needed 1000 patient in each arm -> only got 600

SOS-KANTO Group (2007) - Bystander CPR – Lancet, 361:2298

- outcome measures @ 30 days (good outcomes)

-> 2.2% no CPR

-> 3.1% standard CPR

-> 6.2% external cardiac massage only

-> external cardiac massage may be better than standard CPR

**AED’S**

Bardy (2008) “HAT trial – Home AED Trial” NEJM

- n = 7001 patients

- mean age 62 years

-> AED group produced 4 survivors (out of 3500 patients)

-> need to arrest at home and be witnessed for an AED to be beneficial

Chan, P. S., *et al* (2010) “*Automated External Defibrillators and Survival after In-hospital Cardiac Arrest*” JAMA, 304(19) 2129-2136

- AED’s improve survival in out-of-hospital cardiac arrest.

- data on effectiveness in hospitalized patients are limited.

- cohort study

- n = 11,695

- 204 US hospitals following the introduction of AEDs to general hospital wards

- 82.2% had a nonshockable rhythm (asystole and PEA)

- 17.8% had a shockable rhythm (VF and pulseless VT)

- AED’s used in 38.6% of time

- 18% survival to hospital discharge

-> overall AED use associated with a lower rate of survival

-> AED use in nonshockable cardiac arrest associated with a significantly lower survival

-> AED use in shockable cardiac arrest not associated with survival!