Journal Meeting

Reporter: R1 許哲彰
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2010/10/09

Therapeutic hypothermia

- Unconscious adult patients with return of spontaneous circulation after out-of-hospital cardiac arrest should be cooled to 32-34ºC for 12-24 hours when initial rhythm was ventricular fibrillation (VF) (class IIa).
- Similar therapy may be beneficial for patients with non-VF arrest out-of-hospital or with in-hospital arrest (class IIb).
- Hemodynamically stable patients with spontaneous mild hypothermia (>33ºC) after resuscitation from cardiac arrest should not be actively rewarmed.

Treatment goal

- The patient is actively cooled by using an induced hypothermia protocol for 24 hours to a goal temperature of 32-34ºC.
- The goal is to achieve the target temperature as quickly as possible.
- In most cases, this can be achieved within 3-4 hours of initiating cooling.
- Rewarming is begun 24 hours after the time of initiation of cooling

- Sedation , paralysis
- MAP goal of more than 80 mm Hg is preferred from a cerebral perfusion standpoint.
- Placing the head of the bed at 30º

- Monitor the patient for arrhythmia (most commonly bradycardia) associated with hypothermia.
- An ECG Osbourne or camel wave may be present when cooling.
- Heart rate less than 40 is frequent and is not a cause for concern in the absence of other evidence of hemodynamic instability.

- Hypothermia commonly causes hypokalemia, which may be exacerbated by insulin administration.
- When patients are rewarmed, potassium exits cells, and hyperkalemia may occur.

Hypokalemia during the cooling phase of therapeutic hypothermia and its impact on arrhythmogenesis

- Resuscitation 2010
- Sultan A.Mirzoyev, ChristopherJ.McLeod , T.JaredBunch , MalcolmR.Bell , RogerD.White

Introduction

- Therapeutic hypothermia (TH):
  to therapy in patients with VF as the cause of arrest ➔ American Heart Association Class IIa
- Malignant ventricular arrhythmias continue to be a major cause of in-hospital death after OHCA
- Review our experience with the arrhythmogenic milieu during TH and to propose optimal management strategies.

Methods-1

- Observational study
- OHCA between 2005 12月~ 2009 8月
- Cooling could be commenced within 4h of arrest
- In Public service area (PSA) ➔ ambulance
  out of PSA ➔ helicopter (17 p'ts)

Include
- 87 p'ts in VF
- 7 p'ts in PEA or asystolic responded to therapy yet remained comatose
- 2 p'ts sustained VF in hospital
- TH was terminated in 7 cases

Methods-2

- In 32 p'ts by helicopter with 2L iced N/S infusuion & icepacks on the groin, in the axillae, and around the neck
- Core temperature of 33 ◦C for 24h.
- Rewarming not exceeding 0.5 ◦C/h.

- Over all performance category (OPC) score
  1.goodrecovery
  2.moderate disability
  3.Severe disability
  4.A vegetative state
  5.death

Methods-3

- Blood glucose checked every 2h, keep 120–140mg/dl
- K+ checked every 2h, keep > 3.0 mmol/l
- amiodarone use in hemodynamic unstable arrhythmia
- EKG daily
- Arterial catheters: keep MAP 70–80mmHg
- Mg2+ keep > 1.8 mg/dl

Result-1

- 58(62%) survived to discharge with a median OPC score of 1
- Of the 87 patients presenting with VF, 59(68%) survived to discharge with a median OPC score of 1

Result-2

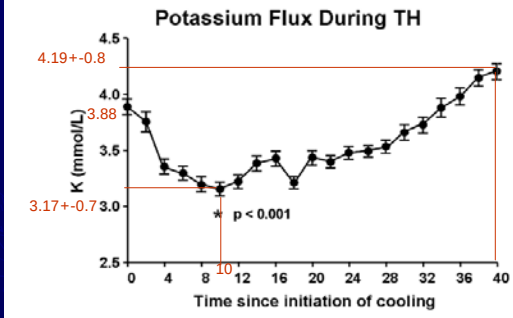
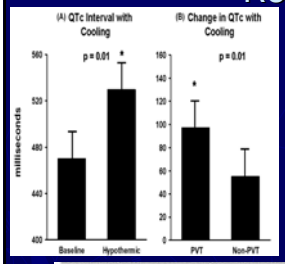


Fig. 1. Serum potassium values at 33 °C compared with baseline were significantly lower, (*$p < 0.001$). Peak serum potassium was not found to be significantly greater than control measurements.

Result-3

- PVT in 11 p'ts (11.7%)
  8: during cooling (34.7±1.0 ◦C)
  1: during rewarming 1: normothermic
  1: at target temp. 33 ◦C
- K+ in the 11 p'ts with PVT: 2.4±0.5mmol/l
  V.S. p'ts without PVT: 3.0±0.6mmol/l
- Severe hypokalemia (K < 3.0mmol/l) was a predictor of PVT (p = 0.01)
  but hyperkalemia (K > 5.5mmol/l) was not.

Result-4



- The change in QTc with cool-ing was found to be strongly associated with PVT (p = 0.01)

Serum potassium and QTc variability during hypothermia

	PVT	No PVT	*p*-Value
Serum K on admission (mmol l^{-1})	3.65 ± 0.7	3.9 ± 0.7	0.2
Core temp at onset of TdP, (°C)	34.7 ± 1.0	n/a	
Lowest serum K (mmol l^{-1})	2.44 ± 0.5	3.0 ± 0.5	0.002
Peak serum K (mmol l^{-1})	4.26 ± 0.9	4.28 ± 0.7	0.9
Baseline QTc (ms)	466 ± 73	472 ± 49	0.7
QTc at 33 °C (ms)	563 ± 74	527 ± 79	0.13
QTc change with cooling (ms)	97 ± 68	55 ± 47	0.01*
QRS duration at baseline (ms)	117 ± 44	129 ± 48	0.4
QRS duration at 33 °C (ms)	122 ± 83	120 ± 129	0.9
QTc at onset of PVT (ms)	580 ± 120	n/a	

Result-5

- Amiodarone, hypomagnesemic, vasoactive or inotropic drugs
  ➔ not associated with PVT

Discussion-1

- Cooling phase ➔ K↓ associated with :
  1. QT prolong 2. PVT
- 72% of PVT occurred during cooling.
  ➔ electrolyte moniter
- rebound hyper-K is not associated with PVT.
- Hypo-K or hypothermia ➔ QT change ➔ predictor of PVT

Discussion-2

- Mild hypothermia has been shown to have as actuary effect on ventricular tachyarrhythmias in animal models. Not only does hypothermia confer resistance to the initiation or maintenance of VT/VF, but also appears to improve defibrillation efficacy

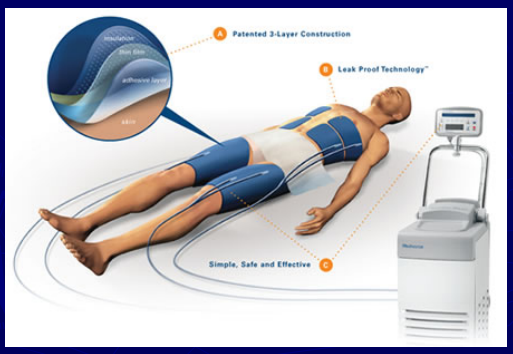
Limitation

- Sample size (ex. vasoactive or inotropic drugs).
- Lack of a control group (normothermic or no K supplement).
- Can' t control for size of infarct or changes in reperfusion therapy–both of which are independent risk factors of PVT.
- anoxic-intracranial injury was not studied in our analysis.

Conclusion

- TH is associated with a K ↓ especially during cooling.
- Maintenance K+ levels > 3.0mmol/l during this potentially vulnerable period may be critical in avoiding the development of PVT.

Tanks for your attention!



Arctic Sun thermoregulation system