Journal Reading
2011/07/19

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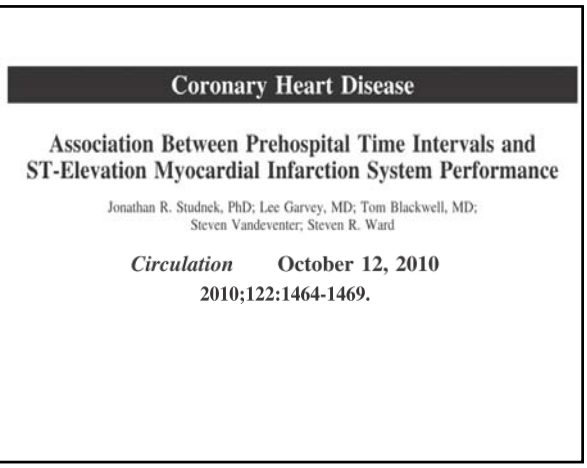

Coronary Heart Disease

Association Between Prehospital Time Intervals and ST-Elevation Myocardial Infarction System Performance

Jonathan R. Studnek, PhD; Lee Garvey, MD; Tom Blackwell, MD; Steven Vandeventer; Steven R. Ward

Circulation October 12, 2010
2010;122:1464-1469.

Introduction

- Current guidelines (from ACC and AHA) recommend that the interval from first medical contact to percutaneous coronary intervention (PCI) be ≤ 90 minutes among STEMI patients.
- Aim to reduce time to PCI by integrating the prehospital and in-hospital care of STEMI patients.

Introduction

- Reduce time to PCI :
  - early acquisition of prehospital 12-lead ECGs
  - subsequent hospital notification in patients with symptoms of an acute coronary syndrome
- Few guidelines on how EMS should optimize there time before arriving the hospital.

Introduction

This retrospective study
- to describe prehospital system time intervals from first medical contact to reperfusion by PCI among STEMI patients,
- to assess associations between time intervals and achieving PCI in ≤ 90 minutes,
- to derive theoretical benchmarks for system time intervals,
- to estimate the probability of achieving PCI in 90 minutes based on proposed timing benchmarks.

Methods - Design and Setting

- Prehospital patients presenting with an acute STEMI as diagnosed by prehospital ECG in Mecklenburg County, North Carolina.
- The Mecklenburg EMS Agency
  - a registry of all prehospital STEMI activations within the county.
- Outcomes data were provided through a cooperative agreement with the 3 hospitals that perform PCI in the county.
- Each STEMI-receiving hospital is an accredited chest pain center with PCI

Methods - Design and Setting

- First responders are all basic life support providers possessing automatic external defibrillator capability.
- Prehospital triage, treatment, and transport protocols are uniform within both the county and city limits.

Methods - Study Population

Inclusion criteria

- Age > 18,
- met the current protocol guidelines for prehospital cardiac catheterization laboratory (CCL) activation,
- had a PCI performed between May 2007 and March 2009.

Methods - Study Population

- A "code STEMI" protocol allowing the prehospital diagnosis of STEMI, CCL activation, and bypass of hospitals not PCI capable.
- signs and symptoms consistent with cardiac ischemia
- the prehospital 12-lead ECG must indicate acute MI
- paramedic overread must confirm this interpretation as
  - ≥ 1-mm ST-segment elevation in ≥ 2 contiguous limb leads
  - or ≥ 2 mm in ≥ 2 contiguous precordial leads.

Methods - Study Population

- Paramedics communicated their findings and ECG information via radio to emergency physicians at the PCI centers. ECG images were not transmitted.
- Emergency physicians activated the PCI center and CCL and notified the interventional cardiologists of incoming patients via a paging system.
- 16% false positive because paramedics incorrectly applied the above algorithm.

Method - Variable Description

- STEMI system performance - first medical contact to reperfusion = the time of 9-1-1 call receipt to first device deployment
- A dichotomous variable – if STEMI system performance ≤ 90mins or NOT

Method - Variable Description

The system time intervals :

- **Response time** = 9-1-1 call receipt to ambulance on scene
- **ECG time** = ambulance on scene to 12-lead ECG acquisition
- **On-scene time** = Ambulance on scene to ambulance departure
- **Notification time** = prehospital ECG acquisition to STEMI team notification
- **Table time** = Scene departure to patient on CCL table
- Age, Gender, and Race

Result

- There were 181 patients during the study period who had prehospital CCL activation and received PCI.
- 165 patients with complete data, most miss the notification time
- Patients had an average age of 60.3 years, 121 (73.3%) were white, and 119 (72.1%) were male.
- 110 received PCI ≤ 90 mins

Table 1. Descriptive Analysis of Prehospital Time Intervals for the Entire Study Population and by Performance Outcome

Variable Name	Mean, min	Median (90th Percentile)	PCI in ≤90 min (95% CI) (n=110)	PCI in >90 min (95% CI) (n=55)
Response time, min	8.0	7.5 (12.5)	7.4 (6.8–7.9)*	9.3 (8.3–10.3)
ECG time, min	6.6	5.1 (12.0)	5.6 (5.0–6.1)*	8.8 (6.9–10.7)
Scene time, min	14.8	14.5 (20.3)	13.5 (12.5–14.4)*	17.4 (16.1–18.8)
Notification time, min	12.8	11.0 (24.2)	10.8 (9.8–11.9)*	16.6 (13.3–19.9)
Table time, min	42.6	39.3 (62.9)	34.8 (32.8–36.7)*	58.3 (51.3–65.4)

PCI indicates percutaneous coronary intervention; ECG; electrocardiogram.
*$P<0.05$.

Table 2. Frequencies, Unadjusted ORs, and 95% CIs for the Occurrence of PCI in <90 Minutes by Each Benchmark Time Interval

Variable	PCI in ≤90 min, n (%)	PCI in >90 min, n (%)	Unadjusted OR (95% CI)	Multivariable OR* (95% CI)
Response time, min				
>11	14 (12.7)	15 (27.3)	1.0	1.0
≤11	96 (87.3)	40 (72.7)	2.6 (1.1–5.8)	9.2 (2.8–29.8)
ECG time, min				
>8	18 (16.4)	26 (47.3)	1.0	1.0
≤8	92 (83.6)	29 (52.7)	4.6 (2.2–9.5)	3.4 (1.2–9.3)
Scene time, min				
>15	32 (29.1)	40 (72.7)	1.0	1.0
≤15	78 (70.9)	15 (27.3)	6.5 (3.2–13.4)	9.6 (3.5–26.6)
Notification time, min				
>10	57 (51.8)	37 (67.3)	1.0	1.0
≤10	53 (48.2)	18 (32.7)	1.9 (1.0–3.8)	1.5 (0.6–3.9)
Table time, min				
>30	64 (58.2)	50 (90.9)	1.0	1.0
≤30	46 (41.8)	5 (9.1)	7.2 (2.6–19.4)	11.1 (3.4–36.0)

*Adjusted for age, gender, and race.

Discussion

- To provide the best possible care to STEMI patients, process improvements must occur in both the prehospital and in-hospital settings.
- This study identified pre-hospital benchmark time intervals in a STEMI care system and estimated their association with achieving PCI in ≤ 90 minutes.

Discussion

- Time to CCL table, scene time, and response time were most significant.
- The ability to rapidly transport patients off scene and arrive on the CCL table in ≤ 30 minutes was the variable most strongly associated with achieving PCI in ≤ 90 minutes.

Discussion

- It has also been shown that delay to the CCL table can be reduced if prehospital STEMI triage protocols incorporate bypassing non-PCI facilities and emergency departments within PCI facilities.

Discussion

- Prehospital scene times of ≤15 minutes were associated with a higher probability of achieving PCI in ≤ 90 minutes.
- Current research on prehospital scene times has focused primarily on the treatment and transport of trauma patients.

Discussion

- Prolonged response times resulted in a decreased probability of achieving rapid PCI.
- Several studies have evaluated the effect of response time on outcomes and demonstrated that after the first 4 minutes, patient outcome was not associated with further delays in prehospital response time.

Discussion

- Results from this study indicated a significant association between achieving PCI in ≤ 90 minutes and obtaining a 12-lead ECG in ≤ 8 minutes.
- But notification time was not associated with.
- These benchmarks may serve as plausible starting points for EMS systems process improvement in STEMI care.

Limitations

- Limitations included threats to generalizability - multiple EMS agencies delivering patients to STEMI-receiving centers operating with various treatment algorithms
- This study also used an uncommon definition for first medical contact, the time of 9-1-1 call receipt.

Limitations

- The benchmark table time includes EMS transportation time from the scene to the PCI center, any time spent in the emergency department, and time required for transportation from the emergency department to the CCL.
- The potential for unrecognized confounding and nondifferential misclassification - not include extensive patient demographics and prior medical history.

Limitations

- In addition, the impact of first responders was not considered in this evaluation.
- The process of transforming and analyzing continuous data as dichotomous may lead to misclassification bias.

Conclusion

- Five theoretical benchmarks were derived from these time intervals that enabled the estimation of the probability of achieving PCI in ≤ 90 minutes.
- Although meeting all 5 benchmarks may be an ideal goal, this model may be more useful for identifying areas for system improvement that will have the greatest clinical impact.

Review

Bubble trouble: a review of diving physiology and disease

D Z H Levett,[1] I L Millar[2]

Postgrad Med J 2008;**84**:571–578. doi:10.1136/pgmj.2008.068320

Introduction

This review will address:

- the types of diving practised today
- the physical and physiological effects of the underwater environment
- the breathing gases used by divers and their toxic effects
- the etiology and pathophysiology of diving diseases
- the management principles of diving diseases

可刪?

Physiological risks as a result of the hyperbaric underwater environment including:

- the toxic effects of hyperbaric gases,
- the respiratory effects of increased gas density,
- drowning,
- hypothermia
- bubble related pathophysiology.

TYPES OF DIVING

- Breath-hold diving (apnea diving)
- Diving with breathing apparatus
  - self contained underwater breathing apparatus (SCUBA)
  - surface supplied breathing apparatus, (SSBA)
  - closed circuit rebreather (CCR)
- Saturation diving.

PHYSICAL AND PHYSIOLOGICAL EFFECTS

- cold water triggers vasoconstriction
  - ADH ↓ → diuresis → hypovolemia on surfacing
- Breathing dry cylinder gas exacerbates any fluid deficit
  - high risk of dehydration

PHYSICAL AND PHYSIOLOGICAL EFFECTS

- Each 10 m of sea water → increase 1 atm (deepest: 5340 m = 54.4 atm)
- Dalton's law – 混和氣體總壓=各氣體分壓和
- Henry's law – 氣體溶解度與分壓成正比
  → profound physiological effects including seizures in the case of oxygen toxicity or impaired cerebral function with nitrogen narcosis.
- Super-saturation of tissue with gases → bubble formation when on surfacing

PHYSICAL AND PHYSIOLOGICAL EFFECTS

- Boyle's law – 密閉容器裡, 氣體體積與壓力成反比
  - Distortable tissue – bowel, lung
  - Non-distortable tissue – middle ear, sinus
  ➔ barotrauma
- Reduced pulmonary compliance,
  Increased airways resistance,
  Increased VQ mismatch,
  Increase in the work of breathing

DIVING GASES: CHARACTERISTICS AND TOXIC EFFECTS

Table 1 Properties of commonly used diving gases

Gas	Advantages	Disadvantages	Use
Compressed air	Cheap and readily available	Nitrogen narcosis below 30 msw Density increases work of breathing below 50 msw	Most common breathing mixture for recreational diving
100% oxygen	Minimal narcotic potency	CNS oxygen toxicity above 2 bar	Military and experimental divers Decompression gas in technical divers Limited to depths of 6–8 msw
Nitrox (nitrogen–oxygen, nitrogen <80%)	Increased dive time Reduced decompression time	Potential for oxygen toxicity if inappropriate mixture used at depth	Technical recreational diving Decompression gas for technical divers
Heliox (helium–oxygen)	Reduced narcosis Reduced density	High thermal conductivity Speech distortion High pressure neurological syndrome beneath 200 msw Cost Taste loss	Commercial diving >50 msw Military diving Technical recreational diving
Trimix (helium–nitrogen–oxygen)	Reduced narcosis Reduced density Reduced high pressure neurological syndrome risk on deep diving Avoid hyperoxia	Cost Complicated mixing and risk of error	Deep commercial diving Deep technical recreational diving

CNS, central nervous system; msw, metres of sea water.

DIVING GASES

- Compressed air / 100% O2
- He-O (Heliox) mixture usually replaces air for diving over 50m → less dense and viscosity
- H-O as the least dense but more neurologic effects
- Nitrox (N-O) / Trimix (He-N-O)

- High work rates or failure of absorbent in rebreathing circuits can lead to CO2 retention
  → deaths or near misses

- Oxygen toxicity -

Box 1: Clinical features of oxygen toxicity

Acute oxygen toxicity symptoms

- V: vertigo, visual disturbance
- E: ears, excitability, euphoria
- N: nausea, numbness
- T: tinnitus, twitching, tremor
- I: irritability, irrational behaviour
- D: dizziness, disorientation, depression
- C: convulsions

- Nitrogen narcosis -

Table 2 Typical clinical features of nitrogen narcosis

Nitrogen partial pressure (bar)	Symptoms and signs
2–4	Mild impairment of performance of unpractised tasks Mild euphoria
4	Impaired reasoning and immediate memory Delayed response to visual and auditory stimuli Increased reaction time
4–6	Overconfidence and fixed thinking Calculation errors
6	Impaired judgement Hallucinations
6–8	Laughter approaching hysteria Talkative, occasional dizziness
8	Severely impaired intellectual performance Mental confusion, impaired concentration
10	Stupefaction
>10	Hallucinations, unconsciousness, death

High pressure neurological syndrome and helium

- Helium is used for deep diving → absence of narcotic effects.
- But, below 120 m divers may develop ‘‘high pressure neurological syndrome’’ (HPNS)

  ➔ hyper-excitatory symptoms such as tremor, myoclonic jerks and irritability thought to be due to membrane and neurotransmitter mediated effects of pressure.

  ➔ reversible on return to normal ambient pressure but limit the performance of divers at extreme depths.

Contaminant gas toxicity

- Carbon monoxide poisoning → death
- Low molecular weight volatile hydrocarbon → underwater incapacitation and post-dive malaise.

DIVING DISEASES:
ETIOLOGY AND PATHOPHYSIOLOGY

Barotrauma

- affect any non-vented gas containing space
- Middle ear barotrauma is the most common but usually minor and self limiting.
- inner ear barotraumas with rupture of the round or oval window and perilymph fistula are particularly serious.

  → transient alternobaric vertigo
- Apnea divers - pulmonary barotrauma

  → as a 20-fold compression of the lung volume would probably result in fatal intrapulmonary hemorrhage

Pulmonary barotrauma of ascent and arterial gas embolism

- The divers using breathing apparatus during ascent, the compressed gas in their lungs expands with the falling ambient pressure → If intrapulmonary gas is prevented from escaping as a result of a closed glottis, bronchospasm→ barotrauma, focal shearing → gas escape

Box 2: Types of barotrauma

Ear, nose and throat
- ► Mask squeeze
- ► Sinus barotrauma
- ► Tooth barotrauma
- ► Middle ear barotrauma
- ► Inner ear barotrauma

Gastrointestinal tract
- ► Hollow viscus perforation

Pulmonary barotrauma
- ► Surgical emphysema
- ► Pneumomediastinum
- ► Pneumothorax ± tension

Arterial gas embolism
- ► Coronary artery gas embolism
- ► Cerebral artery gas embolism

Decompression sickness: ‘‘The bends’’

- As divers ascending, the partial pressure of inert gas in the blood and tissues exceeds ambient pressure, bubbles form in the tissues and blood vessels, which may result in the clinical syndrome of decompression sickness.
- Immediately to 48h.
- The presence of bubbles is not always associated with overt clinical symptoms and many, if not most, dives result in ‘‘silent’’ bubbles.

Table 3 Clinical features and incidence in decompression sickness: data from 1170 cases recorded in the Institute of Naval Medicine, UK between 1990 and 1999 (personal communication)

Manifestation	Prevalence (%)
Neurological symptoms (including auditory)	77
Limb pain	48
Constitutional	29
Skin	9.7
Respiratory	3.7
Girdle/back pain	3.2
Lymphatic	0.9

Long term effects of diving

- The Evaluation of Long Term Health Impacts of Diving (ELTHI diving study) has compared the long term health of offshore oil industry divers with other offshore workers and found some increase in self reported complaints.
- forgetfulness, loss of concentration, hearing loss and musculoskeletal symptoms
- however, overall health quality of life was not significantly different from other workers and the overall findings did not exceed the threshold of ‘‘clinical significance’’

PREVENTION AND TREATMENT OF DIVING DISEASE

- Factors associated with an increased risk of decompression sickness include obesity, fatigue, dehydration, and inter-current illness.
- Avoiding decompression sickness

  → limits on dive duration for any given depth and prescribe maximum ascent rates and decompression stops for various combinations of depth, dive time and breathing gas mixture.

Treatment for decompression sickness and gas embolism

- In addition to normal first aid procedures, oxygen administration is a priority.

  - enhances the rate of elimination of inert gas and the resolution of bubbles.
- Keep a supine position to minimise the risk of further cerebral artery embolism.
- Intravenous fluid resuscitation is recommended to counteract the intravascular depletion and compromised microcirculation
- Oral hydration if no IVF

Definitive treatment

- The definitive treatment is rapid recompression in a hyperbaric chamber.
  - rapid elimination of inert gas,
  - maximal oxygenation of ischaemic tissues,
  - reduction of edema,
  - inhibition of secondary inflammatory and reperfusion injury,
- Then slow and staged decompression

Other treatment

- NSAID for reduce recompression sessions required
- Steroids, aspirin, heparin
- Lignocaine has been shown to reduce brain dysfunction after air embolism in animal models.
  - anti-leucocyte effect and the deceleration of ischaemic transmembrane ion shifts.

SUMMARY

- Diving is associated with multiple risks, some potentially fatal including
  - drowning,
  - hypothermia,
  - inert gas narcosis,
  - oxygen toxicity,
  - arterial gas embolism,
  - decompression sickness,
  - high pressure neurological syndrome,
  - chronic joint dysfunction
  - neurocognitive impairment.

SUMMARY

- Diving is getting popular
- Diving related pathology in their acute medical practice and an understanding of diving pathophysiology is increasingly important.

Thanks for your attention.

Journal Reading
2011/07/19

Speaker: Int 黃莉婷

Supervisor : 許瓔文醫師

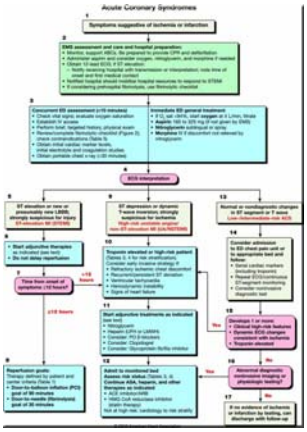
Original article

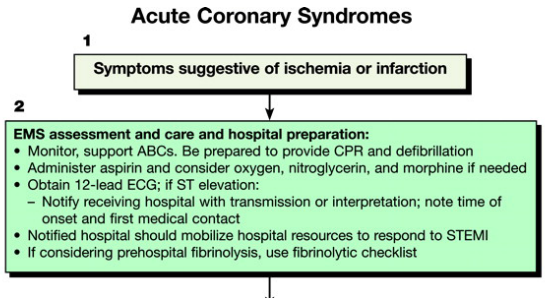
Impact of prehospital diagnosis in the management of ST elevation myocardial infarction in the era of primary percutaneous coronary intervention: reduction of treatment delay and mortality

Roberto Zanini, Marco Aroldi, Silvia Bonatti, Francesca Buffoli, Antonio Izzo, Corrado Lettieri, Michele Romano, Luca Tomasi and Maria Rosa Ferrari

Introduction

- Early and complete myocardial reperfusion: morbidity and mortality in STEMI
- Primary PCI vs fibrinolysis
  - higher infarcted-artery patency
  - lower rates of reinfarction, stroke, and death
- Current guidelines for STEMI
  - fibrinolytic therapy: a door-toneedle time < 30 min
  - primary PCI: door-to-balloon time < 90 min
- In the real world
  - delays in interhospital or intrahospital transfer for primary PCI ⟶ reduce the advantage of PCI



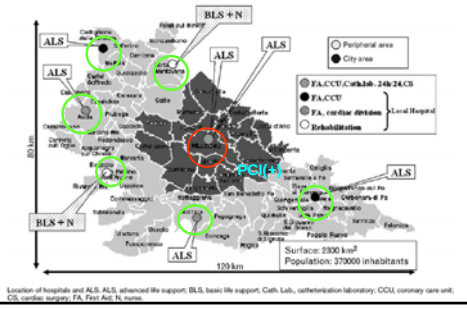


The aim of this study

- 2 groups
  - First Aid admission then transfer to PCI room
  - Ambulance direct transfer to PCI room
- Comparing
  - In-hospital mortality
  - PCI mortality
  - the outcome

Methods

- Mantova sanitary district (north of Italy)
  - 1 acute care hospital with primary PCI
  - 6 local hospitals without primary PCI

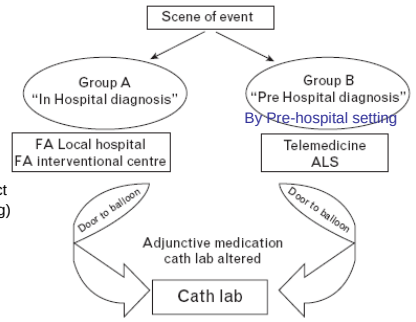


Location of hospitals and ALS. ALS, advanced life support; BLS, basic life support; Cath. Lab., catheterization laboratory; CCU, coronary care unit; CS, cardiac surgery; FA, First Aid; N, nurse.

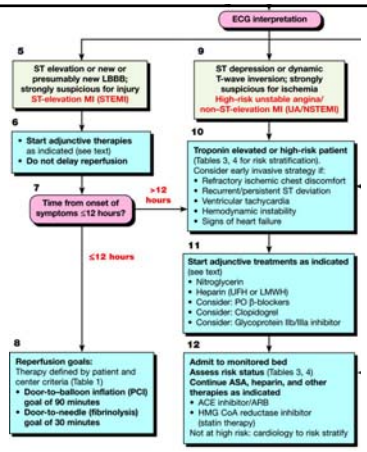
Methods

Fig. 2

- June 2003 ~ June 2005
- Excluded:
  - Symptom duration>12hr
  - Symptom resolution after medication
- First medical contact (ALS or First Aid setting)
  - ASA
  - Abciximab
  - Heparin
  - Addictive nitrates
  - Beta-blocker



Scene of event. ALS, advanced life support; Cath Lab, catheterization laboratory; FA, first aid.



Methods

- Rocord:
  - Baseline of patients
  - Medical therapy in First Aid or ALS
  - Angigraphic data
  - Treatment delay
    - Onset-to-door time: the time of symptom onset~the time of first medical contact
    - Door-to balloon time: the time of first medical contact ~ the time of the first balloon inflation
    - Onset-to-balloon time: the time of symptom's onset ~ the first balloon inflation
- The primary endpoints: the total in-hopital mortality and PCI mortality

Statistics

- Comparions of categorical variables: $x2$ test
- Continuous variables: mean values±SD
- Levels of significance: $P \leqq 0.05$
- SPSS10.0

Results

- 399 patients with STEMI and symptom duration < 12 h
- 34%: by ALS ambulence
  - 31%: from the peripheral area (>20km from the catheterization laboratory)
  - remaining 69%: from an area closer to the interventional centre (<20 km)

Results

Table 1 Clinical and angiographic features

	Group A (FA) 263 patients	Group B (ALS) 136 patients	*P*
Age	64 ± 12.4	63 ± 12.7	NS
Sex (men)	195/263 (74)	101/136 (74)	0.99
Diabetes	60/263 (23)	20/136 (15)	0.11
Anterior AMI	105/263 (40)	42/136 (31)	0.22
Prior AMI, CABG, PCI	58/263 (22)	37/136 (27)	0.37
Killip classes III–IV	13/263 (5)	7/136 (5)	0.93
Multivessel disease	134/263 (51)	75/136 (55)	0.66
Left main	12/263 (4.5)	9/136 (7)	0.41
Pre-PCI TIMI flow 2–3	26/263 (10)	24/136 (18)	0.052

Percentage values are shown in parentheses. ALS, ambulance with advanced life support; AMI, acute myocardial infarction; CABG, coronary artery bypass graft; FA, First Aid; PCI, percutaneous coronary intervention.

The patients baseline characteristics has no significant difference.

Results

Table 2 Therapy in First Aid or in ALS

	Group A (FA) 263 patients	Group B (ALS) 136 patients	*P*
ASA	216/263 (82)	116/136 (85)	0.80
Heparin	208/263 (79)	103/136 (76)	0.78
Abciximab	118/263 (45)	69/136 (51)	0.51
Beta-blockers	68/263 (26)	27/136 (20)	0.29
Nitrates	147/263 (56)	60/136 (44.5)	0.20

Percentage values are shown in parentheses. ALS, ambulance with advanced life support; ASA, aspirin; FA, First Aid.

No difference was found between the groups in relation to pharmacological therapy performed before PCI

Results

Table 3 Primary PCI

	Group A (FA) 263 patients	Group B (ALS) 136 patients	*P*
Primary PCI	223/263 (85)	122/136 (90)	0.72
Procedural success	212/223 (95)	117/122 (96)	0.95
TIMI-3 flow	204/223 (91)	112/122 (92)	0.98
No reflow	15/223 (7)	7/122 (6)	0.73
ST ↓	156/223 (70)	93/122 (76)	0.62

Percentage values are shown in parentheses. ALS, ambulance with advanced life support; FA, First Aid; PCI, percutaneous coronary intervention; ST ↓, ST resolution greater than 50% for anterior acute myocardial infarction and greater than 70% for inferior acute myocardial infarction.

- Among all patients, two had urgent coronary bypass, one from each group.
- Procedural success was distributed equally between both groups

Results

Table 4 Treatment delay

	Group A (FA) 263 patients	Group B (ALS) 136 patients	*P*
Onset to door	174' ± 85	99' ± 67	<0.001
Door to balloon	88' ± 29	49' ± 18	<0.001
Onset to balloon	262' ± 112	148' ± 81	<0.001

ALS, ambulance with advanced life support; FA, First Aid.

- The First Aid of the referral centre: the mean door-to-balloon time: 73±18
- First Aid of local hospitals to interventional centre:
  - -the mean inherent delay time: 47 min
  - -the mean door-to-balloon time was 117±59 min.

Results

Table 5 Mortality

	Group A (FA) 263 patients	Group B (ALS) 136 patients	*P*
Total mortality	23/263 (8.7%)	4/136 (3%)	0.039
PCI mortality	16/223 (7.2%)	4/122 (3.3%)	0.16
Killip class			
I–II	7/210 (3.3%)	3/115 (2.6%)	0.72
III–IV	9/13 (69%)	1/7 (14%)	0.14

ALS, ambulance with advanced life support; FA, First Aid; PCI, percutaneous coronary intervention.

Positive trend noted in B group, esp. in III–IV killip class
Though no significant difference

Killip class

- **Killip class I** includes individuals with no clinical signs of heart failure.
- **Killip class II** includes individuals with rales or crackles in the lungs, an S3, and elevated jugular venous pressure.
- **Killip class III** describes individuals with frank acute pulmonary edema.
- **Killip class IV** describes individuals in cardigenic shock or hypotension (measured as systolic blood pressure lower than 90 mmHg), and evidence of peripheral vasoconstriction (oliguria, cyanosis or sweating).

Results

Table 6 Predictors of mortality

	RC (CI 95%)	*P*
Age (>65 years)	12.1 (5.75–20.22)	0.001
Different pathway to catheterization laboratory (ALS vs. FA)	−7.35 (−15.0–0.31)	0.05
Onset-to-balloon time (<2 h)	−7.19 (−14.44–0.064)	0.05
Killip classes (III–IV)	29.7 (12.9–46.3)	0.001
TIMI pre-PCI	−7.56 (−14.2–0.9)	0.02

ALS, ambulance with advanced life support; FA, First Aid; PCI, percutaneous coronary intervention; RC, regression coefficient. $P < 0.05$.

Diabetes, P<0.01
Predictors with no significance in mortality
1. Gender
2. flowPrevious AMI
3. previous CABG
4. chronic renal failure
5. post-PCI TIMI
6. multi-vessel disease

Discussion

Table 4 Treatment delay

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ALS, ambulance with advanced life support; FA, First Aid.

- The key element in treatment delay
  - the delay in first aid, both remote or of referral centre
- The inherent delay at the local hospital before transferral
  - A major delay of local hospital before transferring to PCI
  - 50 and 73 min in DANAMI-2 and Air PAMI
  - Compared with 47 min in this study

Discussion

Table 4 Treatment delay

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Onset to balloon	262' ± 112	148' ± 81	<0.001

ALS, ambulance with advanced life support; FA, First Aid.

- The importance of patient decision to call the medical emergency system.

Discussion

Table 4 Treatment delay

	Group A (FA) 263 patients	Group B (ALS) 136 patients	*P*
Onset to door	174' ± 85	99' ± 67	<0.001
Door to balloon	88' ± 29	49' ± 18	<0.001
Onset to balloon	262' ± 112	148' ± 81	<0.001

ALS, ambulance with advanced life support; FA, First Aid.

- The importance of pre-hospital diagnosis with telemedicone.

Discussion

- Medical treatment
  - Similar medical treatment in both the groups
- Total mortality
  - Group B(3%) lower significantly than group A(8.7%), P<0.05
- Higher Killip classes (III~IV)
  - Group B: lower mortality trend

Discussion

- Independent predictors of mortality
  - In this study
    - Diabetes
    - Age
    - Killip class

    Already well known factors
    - Pre-PCI TIMI
    - Onset-to-balloon time
    - Different pathway to the cath. room
  - In other studies
    - Onset-to-balloon time
      - Only in high risk patients or <2 hrs. of symptoms onset
      - Not a predictor
  - Different pathway to the cath. Room
    - Comparable with previous study
    - A prehospital ECG by ALS/emergency medical system (EMS): an effective method of reducing time to reperfusion and mortality

Study limitation

- Retrospective study
- Optimal: randomized design to pre-hospital diagnosis system
  - Ethical problem
- 34% of the patients by ALS
  - Low percentage: inadequate number

Conclusion

- Call to ALS ambulances
- Telematic transmission of ECG and clinical parameters
  - a quick diagnosis of STEMI
  - direct referral to interventional centre
- A more extensive use of ALS ambulances equipped with telemedicine
  - Reduce mortality for STEMI

Thank you!

Journal Reading
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Speaker: Int 黃莉婷

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Brief Report

Swimming-induced pulmonary edema in triathletes☆,☆☆

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Introduction

- Pulmonary edema of immersion
  - select populations such as submersion divers and combat swimmers
  - only sporadically in the community setting
- Recently, reports of SIPE (swimming induced pulmonary edema)
  - Triathletes (the unusually large number of swim-related deaths that occurred in triathlons in the 2008 North American season)

Swimming-induced pulmonary edema

- Occurs when fluids from the blood leak abnormally from the small vessels of the lung (pulmonary capillaries) into the airspaces (alveoli)
- SIPE usually occurs during heavy exertion in conditions of water immersion

Signs and symptoms

- Shortness of breath
- Crackles
- Cough, usually distressing and productive of copious pink, frothy or blood-tinged sputum

Proposed mechanisms

- Multiple factors
- Hydrostatic pressure from water immersion squeezes the extremities, and forces blood from the peripheral circulation (arms, legs) to the central circulation (heart, lungs, great vessels of the chest
- Cold water may cause peripheral vasoconstriction
- Wetsuits may add additional extrinsic compression to the extremities

Introduction

- A survey of symptom history and potentially associated risk factors
- Assist in generation of hypotheses for future research
- In this article
  - the findings of a prevalence survey
  - a follow up case-control study
- The way: via the internet
- People: North American triathletes
- Organization: USA triathlon organization (USAT)

Methods

- No validated, community-based pulmonary edema questionnaire
- Limited: “cough productive of pink frothy or blood-tinged secretions”
  - pathognomonic for pulmonary edema
  - difficult to miss
- Also: detailed personal, family medical history, training, hydration, wetsuit wearing habits, swim conditions on the day of first episode

Methods-Avoid Bias

- Servey title: “swim-related breathing problems survey”
  - avoid thinking of pulmonary edema when completing the survey
- Pilot testing
  - 3 small groups of triathletes
  - identify ambiguities in questions
  - evaluate completeness of responses.
- Variable responses
  - “pink frothy or blood-tinged secretions”

  D/D from bleeding due to injury (a crowded triathlon swim can involve a lot of contact between athletes)

Methods-phase 1

- Cooperation: USA Triathlon organization (USAT)
  - All 104,887 triathletes
- The online survey
  - Each monthly e-mail newsletter
  - 3 consecutive months between August and October 2008
  - accept only one response per IP address to avoid duplicate responses

Method-phase 2

- The same online survey
  - A group of cases identified themselves through an online triathlon discussion forum (slowtwitch.com)
- The goal of 2 phases
  - a population based estimate of the prevalence of SIPE-compatible symptoms

Methods

- The sample population
  - $\geqq$20 years old
- Prevalence: the ratio of cases with SIPE-symptoms to the total number of respondents
- The case-control study
  - Study group: all known SIPE-symptoms cases
  - Control group: all non-cases
- Analysis program: SAS 9.1.3

Results

- Returned mails: 1423 respondents over three cycles of distribution (1.3%)
  - 17 junior
  - 6 imcomplete responses
  - Left: 1400 surveys

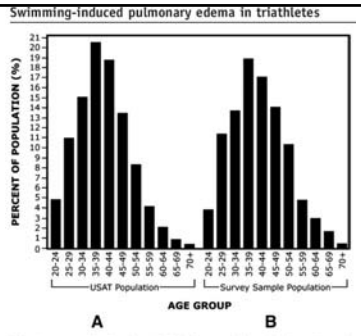

Fig. 1 Age distribution of USAT population (left panel) and survey sample population (right panel). Chi square for difference $P = .21$.

- Fig. 1A: the age distribution of the overall USA Triathlon population, excluding juniors.
- Fig. 1B: the age distribution among the survey respondents.
- Age group relative frequencies between the population and the sample were not different by statistical comparison.

Results

- 20/1400 patients (1.4%)
  - experienced cough productive of pink, frothy or bloody secretions during a swim

Table 1 Case-control study SIPE risk factors

Variable	No. patients (%)	No. Cases (%)	Odds Ratio	95% CI **	P***
Overall	1411 (100.0)	31 (2.2)			
Age					
20 – 29	216 (15.3)	3 (1.4)	1		
30 – 39	461 (32.7)	7 (1.5)	1.09	0.28 – 4.27	1.0
40 – 49	440 (31.2)	11 (2.5)	1.82	0.50 – 6.59	.41
50 – 59	222 (15.7)	10 (4.5)	3.35	0.91 – 12.34	.09
60 – 69	64 (4.6)	0 (0.0)	x		
70 – 79	8 (0.6)	0 (0.0)	x		
Female	662 (46.9)	20 (3.0)	2.08	1.0 – 4.38	.07
Male	749 (53.1)	11 (1.5)	1		
Hypertension	85 (6.0)	7 (8.2)	4.87	2.03 – 11.65	.002
Normal BP	1326 (94.0)	24 (1.8)	1		
Diabetes	7 (0.5)	1 (14.3)	7.63	0.89 – 65.38	.14
No Diabetes	1404 (99.5)	30 (2.1)	1		
Multivitamin	750 (51.0)	18 (2.5)	1.34	0.65 – 2.75	.47
No Multivitamin	691 (49.0)	13 (1.9)	1		
Vitamin C	224 (15.9)	9 (4.0)	2.22	1.00 – 4.88	.07
No Vitamin C	1187 (84.1)	22 (1.9)	1		
Vitamin E	127 (9.0)	4 (3.2)	1.51	0.52 – 4.40	.52
No Vitamin E	1284 (91.0)	27 (2.1)	1		
Fish Oil	304 (21.6)	14 (4.6)	3.10	1.51 – 6.35	.003
No Fish Oil	1107 (78.5)	17 (1.5)	1		
Flax Oil	128 (9.1)	5 (3.9)	1.97	0.74 – 5.21	.19
No Flax Oil	1283 (90.9)	26 (2.0)	1		
Swimming Skill					
Strong	364 (25.8)	8 (2.2)	1.0	0.44 – 2.25	1.0
Not strong	1047 (74.2)	23 (2.2)	1		
Pre-Swim					
Warm Up	402 (28.5)	5 (1.2)	0.48	0.18 – 1.25	.16
No Warm Up	1009 (71.5)	26 (2.6)	1		
Pre-Swim Hydration					
>1L	227 (16.1)	9 (3.9)	2.18	0.99 – 4.80	.08
<1L	1184 (83.9)	22 (1.9)	1		
Open Water	843 (59.7)	23 (2.6)	1.66	0.74 – 3.63	.22

Wetsuit	482 (34.2)	18 (3.7)	2.73	1.33 – 5.63	.007
No Wetsuit	929 (65.8)	13 (1.4)	1		
Long Course	183 (13.0)	10 (5.5)	3.32	1.54 – 7.17	.004
Short Course	1228 (87.0)	21 (1.7)	1		
Climate Trained In					
Hot	659 (46.7)	12 (1.8)	0.72	0.35 – 1.49	.47
Not Hot	752 (53.3)	19 (2.5)	1		

Table 2 Multiple logistic regression risk factors for SIPE

Variable	Parameter Estimate	Adjusted Odds Ratio	95% C.I.	P
Intercept	-5.1506			
Hypertension	1.6821	5.38	2.15-13.48	.0003
Female Gender	1.0114	2.75	1.26-6.02	.02
Long Course	1.1938	3.30	1.50-7.27	.003
Fish Oil	0.9792	2.66	1.28-5.54	.009

Discussion

- Prevelance of SIPE symptoms: 1.4 %
- Independent risk factors:
  - hypertension, fish oil use, longcourse event distance
  - women
- Wetsuit: not independent after adjustments for gender, fish oil, H/T, course length

Discussion

- Immersion in water → preload↑ → cardiac output↑
- Immersion → pulmonary artery pressure*2, right atrial pressure*>2
- In hypertensive athletes
  - Diastolic dysfunction + the increased PA pressure and LV end-diastolic volume → capillary breach → pulmonary edema.

Discussion

- Fish oil
  - antiplatelet
  - vasodilation
    - Increased pulmonary artery pressure pulmonary → capillary leak
- Massive pre-swim hydration (>5 L consumed 2 hours prior to swimming)
  - Risk factors in other reports
  - This study: some increase in the odds of SIPE (>1L)
  - Not statistically significant

Pre-Swim Hydration					
>1L	227 (16.1)	9 (3.9)	2.18	0.99 – 4.80	.08
<1L	1184 (83.9)	22 (1.9)	1		

Limitations

- Self-reported symptoms and risk factors
- Lack of an independently validated measurement tool for SIPE
- Self-report hypertension
  - Underestimate true prevalence
- Unknown water temperature
- Not differentiate between salt and fresh water swims

Conclusions

- 1.4% of community triathletes: an unusual but not rare event
- Risk factors
  - Hypertension
  - fish oil use
  - long course
  - female gender