Journal Meeting

Defibrillator charging before rhythm analysis significantly reduces hands-off time during resuscitation: a simulation study.

American Journal of Emergency Medicine (2013) 31, 395–400 Lars Koch Hansen MD^{a,*}, Lars Folkestad MD^b, Mikkel Brabrand MD^c

> 報告者: PGY莊梓昱 指導者: 李尚醫師 2013.3.12

Introduction

- Two pivotal importance of treating a patient in cardiac arrest.
 - High-Quality Chest compression
 - Rapid defibrillation
- Studies have shown that ANY INCREASE in hands-off time leads to significantly increased mortality.

Yu T et al. Adverse outcomes of interrupted precordial compression during automate defibrillation. Circulation 2002;106:368-72



Guideline told us ...

 2010 International guidelines for resuscitation suggest interruptions in chest compression should be less than

 $5 \text{ sec.} \rightarrow \text{It's Hard } !!!$

Koster RW et al. Part 5: adult basic life support: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. Resuscitation 2010;81(Suppl. 1):e48-70

- 2005 European Resuscitation Council (ERC) guideline:
 - -- ALS provider keep hands off while charging defibrillator.
- 2010 ERC guideline :
 - -- Keep hands off while rhythm check, keep compression while charging defibrillator, and remove for shock delivery.

Author's hypothesis

- Hands-off time in the context of defibrillation can be reduced even further with simple means.
- This study <u>using an alternative sequence</u> for defibrillation of cardiac arrests could be reduced compared to both 2005 and 2010 ALS guideline.

Methods

- Equipment : Resusci Anne HLR-D mannequin and Lifepak 12 and 20 defibrillators.
 - $\mathbin{\hspace{1pt}\text{--}\hspace{1pt}}$ unable to record data on the quality of chest compression.
- Participants
 - -- On call junior physicians at regional Danish hospital
 - -- All volunteers.

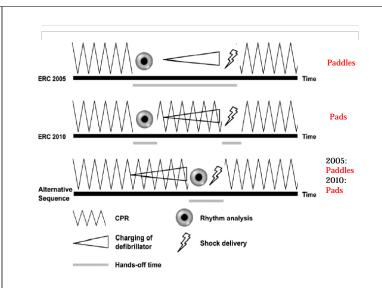


Experiment

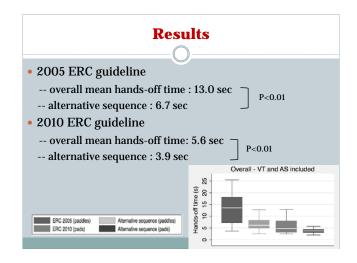
- Participant randomly assigned to preplanned scenarios by a 6-sided dice roll.
- All were confronted with both pulseless VT and asystole.
- Each experiment lasted until the participants had treated both pVT and asystole using either <u>2005</u> or <u>2010 guideline</u> or <u>alternative sequence</u> according to randomization.

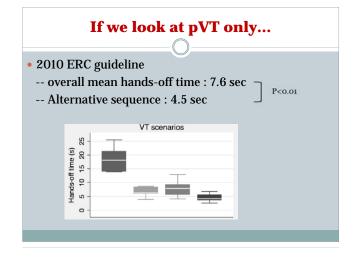


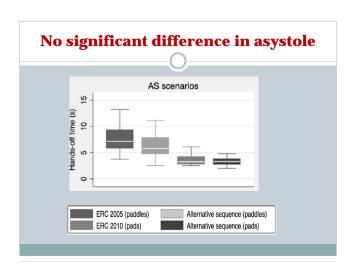
- Participant was set to be team leader and handle defibrillation.
- Start and stop of chest compressions and ventilation were handled on the order of team leader, and the stand-ins had no decisions.
- Screen was cover until the order to halt the chest compression by team leader.
- Hands-off time was recorded using a manual timer.



Information about participants Table Demographics/information about participants ERC 2010 vs alternative sequence 1 Medicine Residency Participant Level of No. of CPRs Years since 2 Cardiology specialty training graduation Specialist 100 24 ERC 2005 vs alternative sequence: 3 Medicine Residency 10 16 1 Medicine Residency 4 Medicine Residency 20 2 Medicine Intemship 5 Medicine Intemship 3 Medicine Residency 6 Medicine Residency 4 Medicine Residency 7 Medicine Intemship 5 Medicine Intemship 8 Medicine Residency 6 Medicine Residency 15 9 Medicine Intemship 0 7 Medicine 10 Medicine 20 Residency 8 Medicine Intemship 25 11 Medicine 50 9 Anaesthesia Specialist Residency 10 Cardiology Residency 25 12 Medicine Residency







Discussions

- Only a small alteration could make a big change, we were able to reach hands-off time < 5 sec.
- Study showed even a brief pause in chest compression
 - → perfusion pressure of the coronary and carotid arteries fell extremely.
- Our alternative sequence compared to

-- 2005 ERC : 6.3 sec reduction -- 2010 ERC : 1.7 sec reduction

Data from defibrillators showed us

- Most of the time, chest compression are often not resumed while charging.
- Rescuers often thought (1)unnecessary to resume chest compression for such a brief interval (2)afraid of being shocked accidentally.
 - → Major objection to the alternative sequence.

Potential increased risk of shocking ALS providers?

- Limited experienced junior physicians with only brief introduction to alternative sequence.
 - -- However, 88 rhythm checks in our study show no examples of potential dangerous defibrillations.
- Edelson et al analyzed 562 defibrillations only 1 shock was delivered while chest compression was ongoing.
 - -- compression still continued at same rate, suggesting no harm was done.
- Recent studies indicates that chest compression can safely be continued during defibrillations.

Paddle v.s. Pads

- Only when using pads and alternative sequence, we were able to have hands-off time <5 sec. (3.9sec)
- In the updated 2010, use of pads is encouraged, and it came close to 5 sec. (5.6 sec)
- And our alternative sequence have only 1 intermission compared to 2010 with 2 intermissions.





Limitations

- A simulation study
- -- participants may be less cautious
- -- no normal chaos, noise, confusion
- Only focus on defibrillation itself
- -- not a full-scale simulation
- Old Anne
 - -- unable to record the quality of chest compression and ventilation (limited findings)

Limitations

- Manual timer
 - -- inferior method compared to automatic registration by novel resuscitation Anne.
 - -- potential measurement error in all sequences.
- We measured the interval from the participants asked for chest compression to be halted until they were asked to resume.
 - -- potentially lead to significant effect of individual participants.

Take home massage

- Charging of the defibrillator before rhythm analysis significantly reduced hands-off time compared with ERC 2005 and ERC 2010 guidelines.
- Pads is recommended for current defibrillation strategy.
- It's not that risky to have chest compression while defibrillation.

Journal Meeting

Red cell distribution width as a prognostic marker in patients with community-acquired pneumonia.

American Journal of Emergency Medicine (2013) 31, 72–79 Jae Hyuk Lee MD, PhD, Hea Jin Chung MD, Kyuseok Kim MD, PhD*, You Hwan Jo MD, PhD, Joong Eui Rhee MD, PhD, Yu Jin Kim MD, PhD, Kyeong Won Kang MD

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Introduction

- Community-acquired pneumonia (CAP)
 - -- leading infectious cause of death
 - -- prognostication is important with management.
- Current prognostic scales
 - -- Pneumonia Severity Index (PSI)

Age, gender, comorbid disease, PE, Lab

-- CURB-65

Confusion, Urea, RR, BP, Age≥65

Prognostic scales

- These scales are used to predict probability of mortality/morbidity among 30-day survival.
- PS
 - -- Useful for identifying low-risk patient.
 - -- Could be safety managed as outpatient.
- CURB-65
 - -- Useful for identifying high-risk patient.
 - -- risk of death.
- Thus, we need add some more biomarkers to improve the prognostic performance.

Red cell distribution width

- RDW a coefficient of variation of circulating red cells.
 - -- reflect the heterogenicity of red cell volume
 - -- a component of CBC
 - -- limited to differential diagnosis of anemia



- Recently, reports associated elevated RDW with outcomes in :
 - > Cardiovascular disease; Rheumatoid arthritis
 - > Colon cancer ; Metabolic syndrome
- We hypothesized that RDW would be associated with 30-day mortality and prognosis factor with CAP.

Methods

- Study design
 - -- Retrospective analysis of a prospective registry database
 - -- 950-bed tertiary academic hospital
 - -- Annual ED census of 67,000
- Participants
 - -- visit ED, and hospitalized for CAP by attending physician of ED based on PSI scale and other medical conditions.
 - -- between April 2008 and March 2011.
 - -- All are older than 18yr

Methods - Selection of participants

- CAP was defined as pulmonary infiltrate on CXR, and symptoms consistent with pneumonia, which were not acquired in hospital or nursing home.
- Also, CXR (X) initial, abnormal lung sound, f/u CXR (O).
- Exclusion criterias
 - -- transfer, discharge from hospital within 10days, experience pneumonia within past 30days.
 - -- active TB, HIV(+),
 - -- chronic immunosuppressed (organ transplant, postsplenectomy, >10mg/d prednisolone < 30 day, neutropenia)

Methods - Data collection

- Initial lab data at ED
 - -- leukocyte count, Hb, Hct, MCV, MCH, RDW, plt
 - -- Glu, crea, BUN, albumin, T-chol, PT, aPTT, CRP.
 - -- PSI and CURB-65 while ED visit.
- Determine the status of patient 1 month after initial ED visit, ascertain death by medical record or by telephone after discharge.
 - -- 744 pt hospitalized, 702 pt identified by chart.
- -- 42 pt telephone contact, 10 pt were transferred to other facility after acute management for CAP.

Methods - Outcome measure

Primary end point

- -- 30 day mortality after the ED visit.
- -- survival time was also investigated.

Secondary end point

- -- hospital length of stay
- -- use of vasopressors
- -- ICU admission
- -- mechanical ventilator require

Methods - Primary data analysis

- Impact of RDW on outcome of CAP patients
 - -- categorized into quartiles

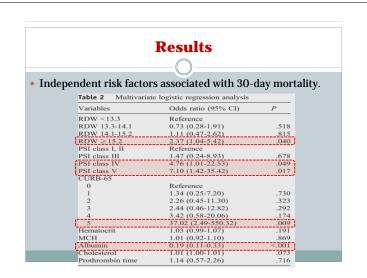
RDW <13.3% | 13.3~14.1% | 14.1~15.2% | >15.2%

 Test of the added ability of RDW to predict 30-day mortality.

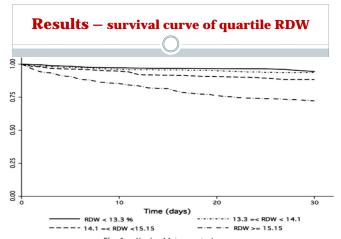
Results – Characterisitcs

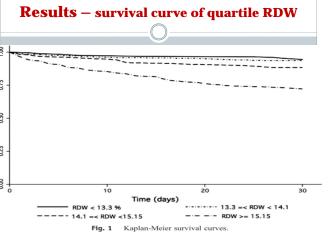
Variables	$\frac{\text{Total patients}}{n = 744}$	Quartile (RDW)				
		<13.3 (n = 196)	13.3-14.1 (n = 172)	14.1-15.2 (n = 190)	≥15.2 (n = 186)	
Age (mean ± SD)	70.1 ± 15.0	65.2 ± 17.5	70.6 ± 14.3	73.1 ± 13.8	71.5 ± 12.7	<.001
Male sex (%)	238 (32.0)	62 (33.2)	65 (37.8)	58 (30.5)	50 (26.9)	.159
Comorbidities, n (%)						
Heart failure	18 (2.4)	2 (1.0)	2 (1.2)	8 (4.2)	6 (3.2)	.120
Renal failure	83 (11.2)	16 (8.2)	15 (8.7)	24 (12.6)	28 (15.1)	.113
Liver disease	44 (5.9)	7 (3.6)	11 (6.4)	15 (7.9)	11 (5.9)	.322
COPD	148 (19.9)	38 (19.4)	39 (22.7)	38 (20.0)	33 (17.7)	.704
Neoplasm	195 (26.2)	35 (17.9)	26 (15.1)	55 (29.0)	79 (42.5)	<.001
Neurologic disease	187 (25.1)	42 (21.4)	44 (25.6)	56 (29.5)	45 (24.2)	.331
Diabetes mellitus	222 (29.8)	48 (24.5)	50 (29.1)	63 (33.2)	61 (32.8)	.207

Variables	Total patients n = 744	Quartile (RDW)				
		<13.3 (n = 196)	13.3-14.1 (n = 172)	14.1-15.2 (n = 190)	≥15.2 (n = 186)	
aboratory findings						
WBC	12.7 ± 6.8	13.0 ± 5.8	13.2 ± 7.1	12.8 ± 5.9	11.7 ± 8.1	.1
Hemoglobin level	12.1 ± 2.2	13.0 ± 1.8	12.5 ± 2.0	12.3 ± 2.0	10.8 ± 2.3	<.0
Hematocrit (%)	36.1 ± 6.4	38.0 ± 5.2	36.8 ± 6.1	36.8 ± 6.0	32.8 ± 6.7	<.(
MCV	93.4 ± 6.7	93.8 ± 4.7	93.0 ± 5.3	94.1 ± 6.4	92.5 ± 9.3	.0
MCH	31.3 ± 2.6	32.0 ± 1.9	31.4 ± 2.1	31.5 ± 2.3	30.5 ± 3.6	<.0
Platelet (×10 ^{3/} mm ³)	252.8 ± 127.0	252.1 ± 110.5	257.4 ± 110.4	256.4 ± 153.2	245.7 ± 128.8	.8
Glucose (mg/dL)	164.6 ± 111.6	160.0 ± 80.7	178.6 ± 144.3	164.7 ± 123.0	156.3 ± 90.3	.2
Albumin (g/dL)	3.4 ± 0.6	3.7 ± 0.5	3.5 ± 0.6	3.4 ± 0.5	3.2 ± 0.6	<.0
Cholesterol (g/dL)	143.0 ± 40.3	146.5 ± 34.9	144.6 ± 40.4	148.4 ± 42.6	132.3 ± 41.4	<.0
BUN (mg/dL)	24.4 ± 16.9	19.5 ± 11.6	22.8 ± 16.2	26.3 ± 18.2	29.0 ± 19.3	<.0
Creatinine (mg/dL)	1.4 ± 1.3	1.2 ± 1.0	1.3 ± 1.2	1.5 ± 1.5	1.6 ± 1.5	.0
Arterial pH	7.43 ± 0.07	7.44 ± 0.07	7.42 ± 0.07	7.42 ± 0.07	7.43 ± 0.09	.3
Pao2	68.6 ± 29.7	69.3 ± 25.1	67.2 ± 25.3	68.7 ± 35.8	69.1 ± 31.1	.5
Paco ₂	35.2 ± 10.9	35.1 ± 9.3	36.2 ± 12.0	35.7 ± 10.8	34.0 ± 11.2	.3
HCO ₃ -	22.6 ± 5.1	23.0 ± 3.7	22.8 ± 5.4	22.8 ± 5.6	21.8 ± 5.3	
Na	134.7 ± 7.6	134.7 ± 9.8	134.1 ± 6.1	135.0 ± 6.6	135.1 ± 7.0	.6
K	4.2 ± 0.7	4.1 ± 0.5	4.2 ± 0.7	4.2 ± 0.7	4.3 ± 0.7	.0
CI	99.6 ± 6.7	99.9 ± 5.3	98.5 ± 7.0	99.6 ± 7.0	100.3 ± 7.4	.0
PT (INR)	1.2 ± 0.4	1.14 ± 0.30	1.19 ± 0.47	1.21 ± 0.28	1.27 ± 0.37	((
apt (s)	43.2 ± 11.9	44.5 ± 13.6	42.1 ± 13.2	41.9 ± 8.0	44.3 ± 12.2	.6
CRP (mg/dL)	13.5 ± 9.4	12.9 ± 8.4	14.3 ± 10.2	13.2 ± 10.1	13.7 ± 8.9	3
PSI class		1817 = 014	1415 - 1012	15.2 - 10.1		<.0
I. II	132 (17.7)	66 (50.0)	35 (26.5)	15 (11.4)	16 (12.1)	
III	136 (18.3)	50 (36.8)	40 (29.4)	31 (22.8)	15 (11.0)	
IV	300 (40.3)	61 (20.3)	68 (22.7)	82 (27.3)	89 (29.7)	
V	176 (23.7)	19 (10.8)	29 (16.5)	62 (35.2)	66 (37.5)	
CURB-65	110 (8011)	17 (1010)	25 (1015)	02 (0012)	00 (0110)	<.0
0	96 (12.9)	47 (49.0)	22 (22.9)	13 (13.5)	14 (14.6)	
1	214 (28.8)	71 (33.2)	58 (27.1)	52 (24.3)	33 (15.4)	
2	253 (34.0)	48 (19.0)	55 (21.7)	71 (28.1)	79 (31.2)	
3	133 (17.9)	25 (18.8)	30 (22.6)	42 (31.6)	36 (27.1)	
4	38 (5.1)	4 (10.5)	5 (13.2)	10 (26.3)	19 (50.0)	
5	10 (1.3)	1 (10.0)	2 (20.0)	2 (20.0)	5 (50.0)	

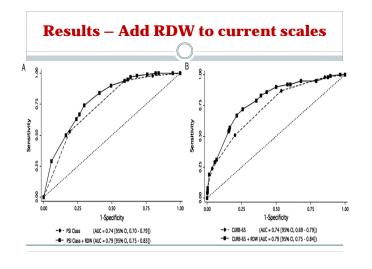


Variables	Total patients (n = 744)	Quartile (RDW)					
		Quartile 1 (RDW <13.3, n = 196)	Quartile 2 $(13.3 \le RDW < 14.1,$ n = 172)	Quartile 3 (14.1 \le RDW < 15.2, n = 190)	Quartile 4 (RDW ≥ 15.2, n = 186)		
30-d mortality, n (%)	100 (13.4)	11 (5.6)	12 (7.0)	24 (12.6)	53 (28.5)	<.00	
PSI							
I, II	2/132 (1.5%)	0/66 (0.0%)	0/35 (0.0%)	0/15 (0.0%)	2/16 (12.5%)	.02	
III	4/136 (2.9%)	1/50 (2.0%)	1/40 (2.5%)	1/31 (3.2%)	1/15 (6.7%)	.73	
IV	41/300 (13.7)	6/61 (9.8%)	4/68 (5.9%)	10/82 (12.2%)	21/89 (23.6%)	.01	
V	53/176 (30.1)	4/19 (21.1%)	7/29 (24.1%)	13/62 (21.0%)	29/66 (43.9%)	.02	
CURB-65							
0	2/96 (2.1%)	1/47 (2.1%)	0/22 (0.0%)	1/13 (7.7%)	0/14 (0.0%)	.39	
1	11/214 (5.1%)	0/71 (0.0%)	3/58 (5.2%)	3/52 (5.8%)	5/33 (15.2%)	.00	
2	36/253 (14.2%)	4/48 (8.3%)	2/55 (3.6%)	7/71 (9.9%)	23/79 (29.1%)	<.00	
3	27/133 (20.3%)	4/25 (16.0%)	3/30 (10.0%)	10/42 (23.8%)	10/36 (27.8%)	.28	
4	16/38 (42.1%)	1/4 (25.0%)	2/5 (40.0%)	2/10 (20.0%)	11/19 (57.9%)	.23	
5	8/10 (80%)	1/1 (100.0%)	2/2 (100.0%)	1/2 (50.0%)	4/5 (80.0%)	1.00	
Secondary outcomes							
HLOS, median	11 (7-18)	10 (6-15)	11 (8-17.5)	11.5 (8-21)	12 (8-20)	.00	
(IQR)							
ICU admission,	107 (14.4)	18 (9.2)	28 (16.3)	29 (15.3)	32 (17.2)	.07	
n (%)							
Vasopressor	103 (13.8)	10 (5.1)	23 (13.4)	32 (16.8)	38 (20.4)	<.00	
use, n (%)							
MV use, n (%)	102 (13.7)	17 (8.7)	25 (14.5)	28 (14.7)	32 (17.2)	.08	





Results - Cox proportional hazard regression analysis Evaluate the relation between RDW and outcomes while adjusting for age, sex, PSI and other risk factors. Table 4 Hazard ratios by categories of RDW Model Hazard ratio (95% CI) Test for trend P value RDW RDW RDW quartile 1 quartile 2 quartile 3 quartile 4 1.28 (0.59-2.74) Model 1 1.0 (reference) 0.86 (0.36-2.05) 2.31 (1.12-4.79) < 001 Model 2 1.0 (reference) 0.76 (0.32-1.81) 1.06 (0.49-2.28) 2.06 (1.01-4.20) <.001 Model 3 1.0 (reference) 0.88 (0.37-2.08) 1.37 (0.64-2.95) 2.45 (1.20-4.98) <.001 Model 1 is adjusted for age, sex, neoplastic disease, hematocrit, albumin, cholesterol, BUN, and prothrombin time. Model 2 is adjusted for PSL albumin cholesterol, and prothrombin time. Model 3 is adjusted for URB-65 albumin, cholesterol, and prothrombin time



Limitations

- Single institution and only included patient hospitalized via the ED.
- No gather data about anemia, transfusion status, or nutritional deficiency.
- ED physician knew the results of the lab values, including RDW, before enrollment.

Discussions

- This study demonstrate higher RDW associated with increased 30-day mortality in CAP patient, especially in RDW >15.2%.
- Length of hospital stay and vasopressor require were also affected by RDW ↑.
- Exact mechanism?

Role of albumin

- Among other variables, albumin was significant correlated with RDW, and was identify as an independent predictor of mortality in multi-analysis.
- Thus, low serum albumin level in high RDW patient may reflect the severity of CAP.
- In Future, while revise for the severity scale, may consider adding these biomarkers.

Currently we knew ...

- Suggest "inflammation" and "oxidative stress" affect red cell homeostasis.
- Studies showed RDW have a strong association with inflammatory biomarkers in general outpatient.
- Serum antioxidant level (selenium, carotenoids) have association with RDW in older women.
- However, CRP level were not different across the quartile change of RDW → CRP-insensitive inflammatory mechanism

Thank you for your listening!