

WORKSHEET for Evidence-Based Review of Science for Veterinary CPR

1. Basic Demographics

Worksheet author(s)

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Date Submitted for review:

2. Clinical question: PA19

In dogs and cats with ROSC after cardiac arrest, does the use of a comprehensive treatment protocol, as opposed to standard care, improve outcome (eg, survival)?

3. Conflict of interest specific to this question:

Do any of the authors listed above have conflict of interest disclosures relevant to this worksheet?
NO

4. Search strategy (including electronic databases searched):

4a. Databases

-MEDLINE via PUBMED (1950-June 2011)

1. Cardiac arrest
 2. Post cardiac resuscitation protocols
 3. Outcome
- 1,2 and 3 : 4 relevant hits out of 15

4b. Other sources

Google Scholar:

- 1) Werling 2007: Treatment and outcome in post-resuscitation care after out-of-hospital cardiac arrest when a modern therapeutic approach was introduced.
- 2) Behringer 2002: Antioxidant Tempol enhances hypothermic cerebral preservation during prolonged cardiac arrest in dogs
- 3) Ebmeyer 2000: Thiopental combination treatments for cerebral resuscitation after prolonged cardiac arrest in dogs. Exploratory outcome study.
- 4) Safar 1996: Improved cerebral resuscitation from cardiac arrest in dogs with mild hypothermia plus blood flow promotion
- 5) Sterz 1990: Hypertension With or Without hemodilution after Cardiac arrest in dogs.

Review articles assessed:

Couper 2010: Advances in postresuscitation care
Nolan 2010: Postresuscitation care: entering a new era
Cokkinos 2009: Post-resuscitation care: Current therapeutic concepts
Ewy 2009: Recent Advances in Cardiopulmonary resuscitation

Relevant articles:

Knafelj 2007: Primary percutaneous coronary intervention and mild induced hypothermia in comatose survivors of ventricular fibrillation with ST-elevation acute myocardial infarction.

Nago 2010: Early induction of hypothermia during cardiac arrest improves neurological outcomes in patients with out-of-hospital cardiac arrest who undergo emergency cardiopulmonary bypass and percutaneous coronary intervention.

Kirves 2007: Adherence to resuscitation guidelines during prehospital care of cardiac arrest patients.

4c. State inclusion and exclusion criteria for choosing studies and list number of studies excluded per criterion

Inclusion criteria

- Prospective, retrospective, experimental laboratory studies in human or whole animal successfully resuscitated from cardiac arrest (Excluded- 2: Herzog 2010, Werling 2007)
- Patient/Animal treated with two or more interventions in the post cardiac resuscitation period (Excluded 1- Rittenberger 2008)
- Monitored or follow thru for greater than 24 hours with procedures that are relevant to vet med or even possible in vet med- (Excluded -2: Knafeli 2007, Nagao 2010)

Exclusion criteria

- Review articles, abstracts only, non-peer reviewed publication, practice guidelines, systemic reviews or surveys, conference proceedings
- Studies that do not address the stated question,.
- Studies that had only one intervention in the post-resuscitation period or followed the patients/animals for less than 24 hours.
- Interventions applied during cardiopulmonary/cerebral resuscitation and not in the post-resuscitation period

4d. Number of articles/sources meeting criteria for further review: 7

5. Summary of evidence

Evidence Supporting Clinical Question

Good			Safar P1996 D			<i>Sunde k 2007 D</i>
Fair						<i>Kirves H 2007 E</i>
Poor						<i>Gaieski DF 2009 C</i>
	1	2	3	4	5	6
Level of evidence (P)						

A = Return of spontaneous circulation
 B = Survival of event

C = Survival to hospital discharge
 D = Intact neurological survival

E = Other endpoint
Italics = Non-target species studies

Evidence Neutral to Clinical question

Good						
Fair			Sterz F 1990 D Ebmeyer U 2000 D Behringer W 2002 D			
Poor						
	1	2	3	4	5	6
Level of evidence (P)						

A = Return of spontaneous circulation
B = Survival of event

C = Survival to hospital discharge
D = Intact neurological survival

E = Other endpoint
Italics = Non-target species studies

Evidence Opposing Clinical Question

Good						
Fair						
Poor						
	1	2	3	4	5	6
Level of evidence (P)						

A = Return of spontaneous circulation
B = Survival of event

C = Survival to hospital discharge
D = Intact neurological survival

E = Other endpoint
Italics = Non-target species studies

6. REVIEWER'S FINAL COMMENTS AND ASSESSMENT OF BENEFIT / RISK:

There were three articles from the human literature which described a comprehensive protocol for post-cardiac arrest resuscitation (PCAR). Two of these articles described in hospital protocols, however only one of these Gaieski et al Resuscitation 2009 had a post arrest protocol that was applicable to veterinary medicine (therapeutic hypothermic and early goal-directed hemodynamic optimization (EGDHO) simultaneously within a 6 hour window of presentation to the ER department. The other two studies Sunde et al Resuscitation 2007 and Kirves et al Eur J Emerg Med 2007, are not applicable to veterinary medicine. Sunde et al had several interventions in the PCAR phase, however a significant percentage of the clinical improvement in outcome (survival) was attributed to coronary reperfusion via percutaneous coronary intervention. It is not possible to tease out the degree of improvement due to the other interventions which were applicable to veterinary medicine versus reversal of the likely primary underlying cause of the cardiac arrest- myocardial infarction.. The underlying etiology of cardiac arrest in our populations is almost never secondary to coronary artery disease which is what this procedure is addressing and attempting to reverse in the human population. Kirves et al study was in the pre-hospital period and this is a situation which does not occur in veterinary medicine.

The four articles from the Experimental Laboratory Research side were performed by the same core group of researchers and used over the last 20 years in a reproducible and proven dog model of cardiac arrest with long follow up in an intensive care situation (96 hrs). The ICU care in all dogs consisted of IPPV for 20-24 hours post ROSC (sedation with fentanyl or morphine, neuromuscular blockade, FiO₂ of 1 (0-2 hrs) and then 0.5 (2- extubation) with a normalization of the PaCO₂, treatment of any cardiac arrhythmias, and normalization of the electrolytes, blood pH, blood glucose plus or minus HCT (hemodilution was an intervention in two of the experiments: Safar 1996 and Stertz 1990)

These studies were not assessing survivability rather they were evaluating improvement of the post ischemic encephalopathy which occurs in the post arrest period. There was a definitive improvement in overall (neurological) performance categories (1-Normal, 2-Moderate disability, 3-Severe disability, 4-Coma, 5 - Brain Death) and histological brain damage with a post-arrest protocol which included mild hypothermia for 12 hours (34 degrees Celsius), normocapnia (PaCO₂ 35-40 mm Hg) and hemodilution hypertension. Stertz F 1990 previously evaluated the use of immediate hypertensive episode and hemodilution post ROSC. They utilized a similar dog model and demonstrated a lack of significant clinical or statistical benefit with delayed hemodilution (≥ 3 minutes post ROSC). The evidence for benefit of hemodilution is not clear, Stertz et al did not demonstrate any significant functional or histological improvement in the group specifically targeted to a lower HCT (0.2-0.25 range) in the ROSC phase and the benefits seem primarily due to hypertensive reperfusion. In Safar et al the experimental group had a combination of hemodilution and hypertensive interventions, so further comment on the benefits or harm of hemodilution cannot be made at this time. The dogs did tolerate the lower HCT (0.2) without any sequelae.

Behringer W 2002 and Ebmeyers groups used a similar dog model with the addition of an antioxidant (Tempol) and poly pharmacy (thiopental, methylprednisolone and phenytoin) respectively. In each of these experiments there was statistically significant neurological functional improvement and a trend towards histological improvement.

7. Conclusion

There were no LOE 1 studies in the literature which, addressed the clinical question in our target species or in humans. The “ best” evidence we have which addressed an important aspect of the overall survivability in the ROSC situation, neurological outcome, are four LOE 3 studies (Behringer W 2002, Ebmeyer U 2000, Safar P 1996, Sterz 1990) . There was significantly better overall neurological performance and histological scores with post-ROSC interventions after a comprehensive treatment protocol (mild hypothermia for 12 hours at 34 degree Celsius, immediately post-ROSC SAP>200 mmHg from 0-5 minutes and then a sustained hypertension MAP>140 mm Hg for 4 hrs with vasopressor support as required to achieve this and normocapnia achieved with IPPV for 20-24 hours) in an experimental laboratory setting with a long follow-up period. The addition of specific pharmacologic interventions such as Tempol, or thiopental, methylprednisolone and phenytoin (in combination) demonstrated improvement in the functional neurological status but not the histological scores.

The interventions of mild hypothermia (12 hrs) and increased cerebral blood flow via immediate and sustained hypertensive episode (with the addition of vasopressors) consistently produced a significant improvement in neurological outcome, both clinically and statistically under experimental conditions. However, how well these will translate in to clinical practice will depend upon how practical, timely and economically they can be implemented in a clinical setting.

8. Acknowledgement

9. Citation list

1) Behringer W, Safar P, Kentner R, et al. (2002). “Antioxidant Tempol enhances hypothermic cerebral preservation during prolonged cardiac arrest in dogs”. J Cereb Blood Flow Metab. Jan;22(1):105-17.

Abstract

The authors are systematically exploring pharmacologic preservation for temporarily unresuscitable exsanguination cardiac arrest in dogs. They hypothesized that the antioxidant Tempol improves cerebral outcome when added to aortic saline flush at the start of cardiac arrest. In study A, no drug (n = 8), Tempol 150 mg/kg (n = 4), or Tempol 300 mg/kg (n = 4) was added to 25 mL/kg saline flush at 24 degrees C (achieving mild cerebral hypothermia) at the start of 20-minute cardiac arrest. In study B, no drug (n = 8) or Tempol 300 mg/kg (n = 7) was added to 50 mL/kg saline flush at 2 degrees C (achieving moderate cerebral hypothermia) at the start of 40-minute cardiac arrest. Cardiac arrest was reversed with cardiopulmonary bypass. Mild hypothermia lasted for 12 hours, controlled ventilation was sustained to 24 hours, and intensive care was provided for up to 72 hours. In study A, overall performance category 1 or 2 (good outcome) was achieved in all eight dogs treated with Tempol compared with three of eight dogs in the control group (P = 0.03). In study B, good outcome was achieved in all seven dogs treated with Tempol versus only two of 8 dogs in the

control group ($P = 0.007$). In both studies, neurologic deficit scores were significantly better in the Tempol group, but not total histologic damage scores. At 72 hours, electron paramagnetic resonance spectroscopy of Tempol revealed direct evidence for its presence in the brain. Single- and double-strand DNA damage, nitrotyrosine immunostaining, total antioxidant reserve, and ascorbate acid levels were similar between groups, and thiol levels were decreased after Tempol in study B. The authors conclude that when added to aortic saline flush at the start of prolonged cardiac arrest, the antioxidant Tempol can enhance mild or moderate hypothermic cerebral preservation in terms of improved functional outcome. The mechanisms involved in this beneficial effect need further clarification.

Comment: LOE 3, FAIR, NEUTRAL

Experimental laboratory study done in target species with a proven CA and post resuscitation model. Concurrent controls . All dogs that received Tempol had improved functional outcome , however this did not translate into improved total histological damage scores.

US Department of Defense and US Office of Naval Research Grants

2) Ebmeyer U, Safar P, Radovsky A, et al. (2000). "Thiopental combination treatments for cerebral resuscitation after prolonged cardiac arrest in dogs. Exploratory outcome study." *Resuscitation* 45(2):119-31

Abstract

We postulate that mitigating the multifactorial pathogenesis of postischemic encephalopathy requires multifaceted treatments. In preparation for expensive definitive studies, we are reporting here the results of small exploratory series, compared with historic controls with the same model. We hypothesized that the brain damage mitigating effect of mild hypothermia after cardiac arrest can be enhanced with thiopental loading, and even more so with the further addition of phenytoin and methylprednisolone. Twenty-four dogs (four groups of six dogs each) received VF 12.5 min no-flow, reversed with brief cardiopulmonary bypass (CPB), controlled ventilation to 20 h, and intensive care to 96 h. Group 1 with normothermia throughout and randomized group 2 with mild hypothermia (from reperfusion to 2 h) were controls. Then, group 3 received in addition, thiopental 90 mg/kg i.v. over the first 6 h. Then, group 4 received, in addition to group 2 treatment, thiopental 30 mg/kg i.v. over the first 90 min (because the larger dose had produced cardiopulmonary complications), plus phenytoin 15 mg/kg i.v. at 15 min after reperfusion, and methylprednisolone 130 mg/kg i.v. over 20 h. All dogs survived. Best overall performance categories (OPC) achieved (OPC 1 = normal, OPC 5 = brain death) were better in group 2 than group 1 (< 0.05) and numerically better in groups 3 or 4 than in groups 1 or 2. Good cerebral outcome (OPC 1 or 2) was achieved by all six dogs only in group 4 ($P < 0.05$ group 4 vs. 2). Best NDS were 44 +/- 3% in group 1; 20 +/- 14% in group 2 ($P = 0.002$); 21 +/- 15% in group 3 (NS vs. group 2); and 7 +/- 8% in group 4 ($P = 0.08$ vs. group 2). Total brain histologic damage scores (HDS) at 96 h were 156 +/- 38 in group 1; 81 +/- 12 in group 2 ($P < 0.001$ vs. group 1); 53 +/- 25 in group 3 ($P = 0.02$ vs. group 2); and 48 +/- 5 in group 4 ($P = 0.02$ vs. group

2). We conclude that after prolonged cardiac arrest, the already established brain damage mitigating effect of mild immediate postarrest hypothermia might be enhanced by thiopental, and perhaps then further enhanced by adding phenytoin and methylprednisolone

Comment:

LOE 3 FAIR NEUTRAL

This experimental model is the same as used in the previous study, it too had concurrent and historical controls. There was a definitive neurologic improvement (NDS and HDS between the normothermic group and the mild hypothermia group) and the dogs in the group with Mild hypothermia, thiopental, phenytoin and methylprednisolone all had good cerebral functional outcome (OPC of 1 or 2 in 6/6 dogs), however this did not translate in to a statistical significance in NDS or HDS scores.

A.S Laerdal Foundation and US Navy Medical research and Development Command.

3) Gaieski DF, Band RA, Abella BS, et al. (2009). "Early goal-directed hemodynamic optimization combined with therapeutic hypothermia in comatose survivors of out-of-hospital cardiac arrest." Resuscitation 80(4):418-24

Abstract

Comatose survivors of out-of-hospital cardiac arrest (OHCA) have high in-hospital mortality due to a complex pathophysiology that includes cardiovascular dysfunction, inflammation, coagulopathy, brain injury and persistence of the precipitating pathology. Therapeutic hypothermia (TH) is the only intervention that has been shown to improve outcomes in this patient population. Due to the similarities between the post-cardiac arrest state and severe sepsis, it has been postulated that early goal-directed hemodynamic optimization (EGDHO) combined with TH would improve outcome of comatose cardiac arrest survivors.

Objective: We examined the feasibility of establishing an integrated post-cardiac arrest resuscitation (PCAR) algorithm combining TH and EGDHO within 6h of emergency department (ED) presentation.

Methods: In May, 2005 we began prospectively identifying comatose (Glasgow Motor Score<6) survivors of OHCA treated with our PCAR protocol. The PCAR patients were compared to matched historic controls from a cardiac arrest database maintained at our institution.

Results: Between May, 2005 and January, 2008, 18/20 (90%) eligible patients were enrolled in the PCAR protocol. They were compared to historic controls from 2001 to 2005, during which time 18 patients met inclusion criteria for the PCAR protocol. Mean time from initiation of TH to target temperature (33 degrees C) was 2.8h (range 0.8-23.2; SD=h); 78% (14/18) had interventions based upon EGDHO parameters; 72% (13/18) of patients achieved their EGDHO goals within 6h of return of spontaneous circulation (ROSC). Mortality for historic controls who qualified for the PCAR protocol was 78% (14/18); mortality for those treated with the PCAR protocol was 50% (9/18) (p=0.15).

Conclusions: In patients with ROSC after OHCA, EGDHO and TH can be implemented simultaneously.

Comment: LOE6 In a non target species POOR, POSITIVE.

This is a study with historical controls and does not have clearly targeted outcomes stated. However, this combination of therapies- mild hypothermia and early goal directed hemodynamic optimization are feasible and the most applicable to our patient populations in veterinary medicine.

4) Kirves H, Skrifvars MB, Vähäkuopus M et al. (2007).“Adherence to resuscitation guidelines during prehospital care of cardiac arrest patients.”*Eur J Emerg Med* 14(2):75-81.

Abstract

Objective: The impact of prehospital care after the return of spontaneous circulation in out-of-hospital cardiac arrest patients is not known. This study describes adherence to the resuscitation guidelines, factors associated with poor adherence and possible impact of prehospital postresuscitation care on the outcome of out-of-hospital cardiac arrest.

Methods: One hundred and fifty-seven Finnish out-of-hospital cardiac arrest patients hospitalized during 1 year, were analyzed retrospectively. Patient and arrest characteristics, prehospital postresuscitation care and survival to hospital discharge were analyzed using multivariate logistic regression.

Results: Forty percent of the patients received care accordant with the guidelines. Male sex ($P=0.045$), witnessed arrest ($P=0.031$), initial ventricular fibrillation/ventricular tachycardia rhythm ($P=0.007$) and the presence of an emergency physician ($P=0.017$) were associated with care in line with the current guidelines. In multivariate logistic regression analysis, age over median (odds ratio=3.6, 95% confidence interval 1.5-8.6), nonventricular fibrillation/ventricular tachycardia initial rhythm (odds ratio=4.0, 95% confidence interval 1.6-9.8), administration of adrenaline (odds ratio=7.0, 95% confidence interval 2.3-21.4) and unsatisfactory prehospital postresuscitation care (odds ratio=2.5, 95% confidence interval 1.1-6.3) were associated with a failure to survive up to hospital discharge.

Conclusions: Less than 50% of out-of-hospital cardiac arrest patients received prehospital postresuscitation care compatible with the current guidelines. Markers of poor prognosis were associated with unsatisfactory care, which in turn was more frequent among the patients who did not survive to hospital discharge.

Comment: LOE 6, non target species, FAIR, POSITIVE.

This situation is not applicable to our current reality in veterinary medicine, we do not encounter out-of hospital CA that have ROSC and furthermore are then transported to us with the possibility of

care being provided. However, the study demonstrated that the better the protocol in the post-ROSC phase was adhered to the stronger the association there was to survival. This is an important point as it demonstrated that we need to adopt a multifaceted approach to the treatment of this post arrest phase and pay attention to how well we perform these protocols if there is to be survival benefits.

5) Safar P, Xiao F, Radovsky A, et al. (1996)

“Improved cerebral resuscitation from cardiac arrest in dogs with mild hypothermia plus blood flow promotion”. *Stroke*_27(1):105-13.

Abstract

Background and purpose: In past studies, cerebral outcome after normothermic cardiac arrest of 10 or 12.5 minutes in dogs was improved but not normalized by resuscitative (postarrest) treatment with either mild hypothermia or hypertension plus hemodilution. We hypothesized that a multifaceted combination treatment would achieve complete cerebral recovery.

Methods: With our established dog outcome model, normothermic ventricular fibrillation of 11 minutes (without blood flow) was followed by controlled reperfusion (with brief normothermic cardiopulmonary bypass simulating low flow and low PaO₂ of external cardiopulmonary resuscitation) and defibrillation at < 2 minutes. Controlled ventilation was provided to 20 hours and intensive care to 96 hours. Control group 1 (n = 8) was kept normothermic (37.5 degrees C), normotensive, and hypocapnic throughout. Experimental group 2 (n = 8) received mild resuscitative hypothermia (34 degrees C) from about 10 minutes to 12 hours (by external and peritoneal cooling) plus cerebral blood flow promotion with induced moderate hypertension, mild hemodilution, and normocapnia.

Results: All 16 dogs in the protocol survived. At 96 hours, all 8 dogs in control group 1 achieved overall performance categories 3 (severe disability) or 4 (coma). In group 2, 6 of 8 dogs achieved overall performance category 1 (normal); 1 dog achieved category 2 (moderate disability), and 1 dog achieved category 3 (P < .001). Final neurological deficit scores (0% [normal] to 100% [brain death]) at 96 hours were 38 +/- 10% (22% to 45%) in group 1 versus 8 +/- 9% (0% to 27%) in group 2 (P < .001). Total brain histopathologic damage scores were 138 +/- 22 (110 to 176) in group 1 versus 43 +/- 9 (32 to 56) in group 2 (P < .001). Regional scores showed similar group differences.

Conclusions: After normothermic cardiac arrest of 11 minutes in dogs, resuscitative mild hypothermia plus cerebral blood flow promotion can achieve functional recovery with the least histological brain damage yet observed with the same model and comparable insults.

Comment:

LOE 3 POSITIVE, GOOD

Concurrent and historical controls. This study was the “best “at reproducing a clinically relevant situation of cardiac arrest and resuscitation with feasible interventions which could be implemented in both human medicine and in particular veterinary medicine. The combination of mild hypothermia, hemodilution hypertension and normocapnia in combination produced the “ best” functional neurologic performance and histological improvements in the experiments this group has conducted over the last 15 years. The issue will be how easily these interventions maybe translated in to clinical practice.

AS Laerdal Foundation and National Institutes of Health.

6) Sterz F, Leonov Y, Safar P, et al. (1990). “Hypertension with or without hemodilution after cardiac arrest in dogs.” *Stroke* 21(8):1178-84.

Abstract

We studied blood flow-promoting therapies after cardiac arrest in 18 dogs. Our model consisted of ventricular fibrillation (no blood flow) lasting 12.5 minutes, controlled reperfusion with cardiopulmonary bypass and defibrillation within 5 minutes, controlled intermittent positive-pressure ventilation to 20 hours, and intensive care to 96 hours. Group I (control, n = 6) dogs were reperfused under conditions of normotension (mean arterial blood pressure 100 mm Hg) and normal hematocrit (greater than or equal to 35%). Group II (n = 6) and III (n = 6) dogs were treated with norepinephrine at the beginning of reperfusion to induce hypertension for 4 hours. In addition, group III dogs received hypervolemic hemodilution to a hematocrit of 20% using dextran 40. There were no differences in the time to recovery of electroencephalographic activity among groups. All six group I dogs remained severely disabled; in groups II and III combined, six of the 12 dogs achieved good outcome (p less than 0.01). Some regional histopathologic damage scores at 96 hours were better in groups II and/or III than in group I (neocortex: p less than 0.05 group II different from group I; hippocampus: p less than 0.01 both groups II and III different from group I). Total histopathologic damage scores were similar among the groups. A hypertensive bout with a peak mean arterial blood pressure of greater than or equal to 200 mm Hg beginning 1-5 minutes after the start of reperfusion was correlated with good outcome (p less than 0.01). Our results support the use of an initial bout of severe hypertension, but not the use of delayed hemodilution.

Comment:

LOE3, FAIR ,NEUTRAL.

Dogs in this study had improved neurological functional recovery with hypertensive episode and or hemodilution. There was not a statistically significant difference in the total histological scores between these dogs and the controls. A subanalysis was performed based on the overall performance scores between the good (OPC >2) and poor (OPC<3) outcome dogs to assess if there was indeed a difference in the MAP and HCT between these two groups. The good outcome does had a statistically different MAP from the poor outcome dogs but there was no difference in HCT between the two groups. It is for this reason that the authors attributed the clinical improvement

in neurological function scores to the hypertensive intervention.

7) Sunde K, Pytte M, Jacobsen D, et al. (2007). "Implementation of a standardised treatment protocol for post resuscitation care after out-of-hospital cardiac arrest." *Resuscitation* 73(1):29-39

Abstract

Background: Mortality among patients admitted to hospital after out-of-hospital cardiac arrest (OHCA) is high. Based on recent scientific evidence with a main goal of improving survival, we introduced and implemented a standardised post resuscitation protocol focusing on vital organ function including therapeutic hypothermia, percutaneous coronary intervention (PCI), control of haemodynamics, blood glucose, ventilation and seizures.

Methods: All patients with OHCA of cardiac aetiology admitted to the ICU from September 2003 to May 2005 (intervention period) were included in a prospective, observational study and compared to controls from February 1996 to February 1998.

Results: In the control period 15/58 (26%) survived to hospital discharge with a favourable neurological outcome versus 34 of 61 (56%) in the intervention period (OR 3.61, CI 1.66-7.84, $p=0.001$). All survivors with a favourable neurological outcome in both groups were still alive 1 year after discharge. Two patients from the control period were revascularised with thrombolytic versus 30 (49%) receiving PCI treatment in the intervention period (47 patients (77%) underwent cardiac angiography). Therapeutic hypothermia was not used in the control period, but 40 of 52 (77%) comatose patients received this treatment in the intervention period.

Conclusions: Discharge rate from hospital, neurological outcome and 1-year survival improved after standardisation of post resuscitation care. Based on a multivariate logistic analysis, hospital treatment in the intervention period was the most important independent predictor of survival.

Comment:

LOE 6, GOOD , POSITIVE

Comprehensive postresuscitation strategy for in hospital care of out of hospital cardiac arrest patients. Survival increased from 31% to 56% with the use of a standardized protocol. One of the main interventions in this study was targeted towards coronary arterial revascularization. Myocardial infarction is a very uncommon reason for cardiac arrest in our patient populations, so how this protocol would translate into improved outcomes in our veterinary population is not know. This study does highlight the importance of multiple interventions and attention to how well the protocol is implemented and adhered to.

AS Laerdal Foundation