# WORKSHEET for Evidence-Based Review of Science for Emergency Cardiac Care

**Worksheet author(s)**

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**Clinical question**.

ALS-D-026A "In adult cardiac arrest (asystole, pulseless electrical activity, pulseless VT and VF) (prehospital [OHCA], in-hospital [IHCA]) (P), does the use of calcium alone or combination with other drugs (I) compared with not using drugs (or a standard drug regimen) (C), improve outcomes (eg. ROSC, survival) (O).".

**Is this question addressing an intervention/therapy, prognosis or diagnosis?** The question addresses intervention issue
**State if this is a proposed new topic or revision of existing worksheet**: New topic-

**Conflict of interest specific to this question**

Do any of the authors listed above have conflict of interest disclosures relevant to this worksheet? No one has any C.O.I.

**Search** strategy (including electronic databases searched).

Articles were collected through search in the Databases. The following databases were consulted:
PubMed
Embase
Cochrane Database
AHA Endnote Master Library
Topics where searched through MeSH terms as follows:

| | | | |
|---|---|---|---|
| #29 | Search "Heart Arrest/drug therapy"[Mesh] AND "calcium"[Title/Abstract] Limits: All Adult: 19+ years | 08:20:17 | 18 |
| #28 | Search "Heart Arrest/drug therapy"[Mesh] AND "calcium"[Title/Abstract] | 08:13:35 | 66 |
| #0 | pubmed clipboard | 10:33:26 | 37 |
| #27 | Search #26 AND #24 Limits**:** Humans, All Adult: 19+ years | 10:36:32 | 5 |
| #26 | Search #20 OR #12 Limits: Humans, All Adult: 19+ years | 10:29:19 | 593 |
| #25 | Search #20 OR #12 | 10:26:54 | 1151 |
| #24 | Search "Calcium"[Mesh] | 10:19:41 | 208856 |
| #11 | Search "Calcium/therapeutic use"[Mesh] | 10:17:38 | 6264 |
| #22 | Search #21 AND #11 | 10:15:44 | 34 |
| #21 | Search ("Heart Arrest"[Mesh] OR Arrest, Heart Cardiac Arrest OR Arrest, Cardiac OR Asystole OR Asystoles OR Cardiopulmonary Arrest OR Arrest, Cardiopulmonary) AND "drug therapy"[sh] | 10:09:19 | 3671 |
| #20 | Search ("Heart Arrest"[Mesh] OR Arrest, Heart Cardiac Arrest OR Arrest, Cardiac OR Asystole OR Asystoles OR Cardiopulmonary Arrest OR Arrest, Cardiopulmonary) AND "drug therapy"[Mesh] | 10:06:33 | 887 |
| #16 | Search "Heart Arrest/drug therapy"[Mesh] | 10:02:19 | 891 |
| #19 | Search #17 AND #11 | 10:00:34 | 21 |
| #18 | Search #17 AND #11 AND #14 | 09:56:33 | 1 |
| #17 | Search #16 OR #12 | 09:52:33 | 1133 |
| #14 | Search ("Emergency Service, Hospital"[Mesh] OR "Cardiology Service, Hospital"[Mesh]) | 09:46:34 | 32764 |
| #12 | Search "pulseless electrical activity" OR pulseless VT" | 09:41:36 | 270 |
| #8 | Search "Heart Arrest"[Mesh] AND "Emergency Medical Services"[Mesh] | 09:34:28 | 2069 |

Search was also performed through free text using the following key words: (cardiac arrest OR cpr OR cardiopulmonary resuscitation) AND (calcium OR calcium administration).
For the Cochrane review the following items were used:

| # | Searches | Results |
|---|---|---|
| 1 | heart arrest {No Related Terms} | 497 |
| 2 | calcium {No Related Terms} | 1759 |
| 3 | 1 and 2 | 0 |
| 4 | cardiac arrest {No Related Terms} | 348 |
| 5 | asystole {No Related Terms} | 131 |
| 6 | pulseless {No Related Terms} | 37 |
| 7 | 6 or 4 or 1 or 5 | 984 |
| 8 | 7 and 2 | 4 |
| 9 | from 8 keep 1-4 | 4 |

Additional papers were retrieved through the references cited at the end of each manuscript.

• State inclusion and exclusion criteria

The search yielded a total of 48 manuscripts. From these the following were excluded:

1. no cardiac arrest conditions
2. Editorial articles, abstracts, review articles, comments, guidelines
3. Use of calcium channel blockers not associated with cardiac arrest

• Number of articles/sources meeting criteria for further review:

12

# Summary of evidence

## Evidence Supporting Clinical Question

| | | | | | |
|---|---|---|---|---|---|
| **Good** | | | | | |
| **Fair** | | | | | |
| **Poor** | | | | {Harrison, 1983, 267}[A,B,C] {Kay, 1951, 97}[A,B,C,D] | |
| | **1** | **2** | **3** | **4** | **5** |
| **Level of evidence** | | | | | |

A = Return of spontaneous circulation
B = Survival of event
C = Survival to hospital discharge
D = Intact neurological survival
E = Other endpoint
*Italics = Animal studies*

# Evidence Neutral to Clinical question

| | | | | | |
|---|---|---|---|---|---|
| **Good** | | | | | |
| **Fair** | {Stueven, 1985, 630}[A,BC] {Stueven, 1985, 626}[A,C] | | | | ~~E~~ |
| **Poor** | | {Gando, 1988, 154}[A] | | {Harrison, 1983, 267}[A,B,C] | |
| | **1** | **2** | **3** | **4** | **5** |
| **Level of evidence** | | | | | |

A = Return of spontaneous circulation
B = Survival of event
C = Survival to hospital discharge
D = Intact neurological survival
E = Other endpoint
*Italics = Animal studies*

# Evidence Opposing Clinical Question

| | | | | | |
|---|---|---|---|---|---|
| **Good** | | | | | |
| **Fair** | | {Stueven, 1983, 136}[A,C] | | | |
| **Poor** | | | | | *{Meuret, 1984, 108}*[AE] *{Meuret, 1983, 1153}*[A, E] *(Blecic, 1987, 324)*[A] |
| | **1** | **2** | **3** | **4** | **5** |
| **Level of evidence** | | | | | |

A = Return of spontaneous circulation
B = Survival of event
C = Survival to hospital discharge
D = Intact neurological survival
E = Other endpoint
*Italics = Animal studies*

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

The use of calcium salts for the treatment of cardiac arrest patients was firstly recommended in the 1974 guidelines. This emphasis was provided taking into account the physiological mechanisms by which calcium intervenes in the excitation-contraction coupling. It is likely that some anecdotal report prompted these suggestions but there were no specific references in the 1974 document. This issue was subsequently reinforced in the following 1980 statements and was based on a single human study in which three different salts of calcium (chloride, gluconate and gluceptate) were used in 15 patients undergoing cardiac surgery (White RD, J Thorac Cardiovasc Surg, 1976;72:609) There were no data related to patients in cardiac arrest.

In the first years of the eighties, some studies investigated, both retrospectively and prospectively, the effects of calcium during cardiac arrest and CPR. The drug was strongly de-emphasized in 1986 and in 1992 guidelines that limited its use to the treatment of hyperkalemia, hypocalcemia and calcium channel blocker intoxication (class IIA). In the 2000 guidelines calcium was not taken into account.

This review highlight the paucity of data on the use of calcium during cardiac arrest and CPR. In fact, most of the data refer to studies conducted in the middle of eighties. Following those studies, there were no additional investigations on the use of calcium salts during CPR. A number of review article, however, were published but all referring to the same few studies published in the early eighties.

One LOE 4 study (Kay, 1951, 97) supported the use of the drug but with profound methodological limitation. This study was performed on four pediatric patients who underwent cardiac arrest in the operative room. The Authors simply conclude that ROSC occurred after administration of calcium during the phase of cardiac arrest.

Another LOE 4 study (Harrison, 1983, 267) only partially supported its use in PEA but reported failure of calcium in refractory VF and in asystole. Long term survival (6 months), however, was scant as one patient only of the 7 who arrived alive at the emergency dept survived.

Two LOE 1 studies only were prospectively conducted by the same investigators (Stueven, 1985, 626, Stueven, 1985, 630), one selecting PEA as a subgroup, the second one focused on patients in asystole. In the first study (PEA) only a subgroup of patients with wide QRS had a better ROSC. In the second study (asystole) calcium was associated with worst outcome. No results referring to long term survival or neurological outcome were reported.

One LOE 2 study (Stueven, 1983, 136) and 3 LOE 5 animal studies (Blecic, 1987, 324, Meuret, 1984, 108, Meuret, 1983, 1153) demonstrated, with different and non comparable methods of research, failure of calcium to restore spontaneous cardiac activity.

One additional LOE 2 study (Gando, 1988, 154) was performed to ascertain the relationship between the calcium levels in the blood and the effects of calcium administration in two groups of patients (a total of 30 pts) one who received and the other that did not receive calcium. Despite correction of the ionized calcium levels, there were non differences in ROSC between the two groups.

Hypocalcemia may occur during cardiac arrest and CPR as documented in earlier and more recent animal studies (Cairns CB, Ann Emerg Med 1991;20:1178, Niemann JT, Ann Emerg Med, 1999;34:1) but none of these studies pointed out calcium administration. One additional study (LOE 4,Urban, 1988, 110) had the main goal to investigate the levels of calcemia in two different groups of pts, one out-of-hospital (OOH), the second one in-hospital (IH) cardiac arrest and both received calcium. In the group of OOH-CA there were 12 patients only, 5 VF, 5 asystole, 2 PEA. Three of 5 VF had ROSC and survived while only one in asystole survived. None of PEA were discharged alive from the hospital. In the IH-CA (11 pts) all patients had ROSC and survived. Ten had VF as presenting rhythm and one asystole. The patients in VF were post-CABG or AMI conditions. Their survival occurred regardless calcium administration (given in five patients) and none was hypocalcemic. Both groups had additional pharmacological interventions (epinephrine, bicarbonate) given

in unspecified fashion. Under the physiological point of view, the evidence of acidosis demonstrated in these studies seems to support the competition of idrogen ions with calcium ions thus accounting for the low levels of serum calcium ion.

No cardiac arrest and CPR studies with pre-existing hypocalcemia were found.

In the setting oh hyperkalemia, one single LOE 5 study (Hollmann, 2003, 606) was found. This study failed to demonstrate beneficial effects of calcium to antagonize hyperkalemia. This investigation, however, was conducted in rats and had the main goal to evaluate pre-treatment with calcium prior to induction of hyperkalemia. Absence of $K^+$ measurements, limited number of animals studied, and methodology used do not allow to draw definitive conclusions about correction of hyperkalemic cardiac arrest via the use of calcium.

In the setting of calcium treatment to antagonize the effects of calcium channel blockers, only one LOE 4 study (Andrivet, 1994, 350) reports the beneficial effect of calcium administration during chronic ca-blocker intoxications (diltiazem). However, in this study, the patients had a profound shock state with bradi-asystolic arrest but did not undergo CPR maneuvers. All other studies retrieved from the literature are single case reports (occurred either following verapamil or nifedipine intoxications) in which the patients had several degrees of sinus or atrio-ventricular node dysfunction but without being in cardiac arrest.

**Acknowledgements**:
We gratefully acknowledge dr. Annamaria Falcetta's precious support, responsible of the library service of our Health Unit, for having provided her expertise and for retrieving manuscript not readily available in our sites.

# *Citation List*

| Citation Marker | Full Citation* |
|---|---|
| | |
| {Blecic, 1987, 324} | Blecic S, De Backer D, Huynh CH, Deleuze M, Domb M, Luypaert P, Vincent JL. Calcium chloride in experimental electromechanical dissociation: a placebo-controlled trial in dogs. *Crit Care Med*. 1987;15:324-7.<br><br>Level 5, opposing, poor<br><br>The study tested the effects of calcium chloride randomly administered in comparison to epinephrine and to dextrose (D5W). There was a random assignment in which each drug was given either as first, second or third episode of cardiac arrest. This methodology, by itself, highlights several limitations:<br>- 1: the hemodynamic, respiratory and metabolic conditions of the animals are not the same after recovery from cardiac arrest.<br>2: All animals are substantially acidotic prior to cardiac arrest. The arterial pH and bicarbonate concentration were 7.25 and 13.6 in epinephrine group, 7.26 and 15.0 in calcium group, 7.31 and 16.6. in D5W group. It is no clear the reason of such abnormal pre-arrest condition.<br>3: There are no measurements of calcium levels prior to cardiac arrest and during treatment. Accordingly, despite the randomization, it is not possible to ascertain whether the more negative outcome observed in the calcium group was associated to the levels of hypocalcemia. |
| {Gando, 1988, 154} | Gando S, Tedo I, Tujinaga H, Kubota M. Variation in serum ionized calcium on cardiopulmonary resuscitation. *J Anesth*. 1988;2:154-60.<br><br>Level 2, neutral, poor<br><br>The study was conducted with the primarily goal of ascertaining the values of calcium before and after calcium administration in a series of cardiac arrest patients.<br>There was no randomization. Two groups were identified, one (11 pts) who did receive calcium and group 2 (19 pts) who did not. However, the legend in tab 1 indicates that group 1 was treated with or without calcium whereas group 2 was treated without calcium. There is then no clear assignment of the groups.<br>The statistical method appears weak: there is no clear specification when paired or unpaired t-test was used: Means are reported sometimes with SD and sometimes with SE.<br>There is no indication of any of the treatment during cardiac arrest (down time, presenting rhythm, witness CPR, duration of CPR, pharmacological interventions). Accordingly, the comparison between the two groups is impossible.<br>There are several spelling mistakes. Overall the manuscript is poorly written. |

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| {Harrison, 1983, 267} | Harrison EE, Amey BD. The use of calcium in cardiac resuscitation. *Am J Emerg Med*. 1983;1:267-73.<br><br>Level 4, supporting and neutral, poor<br><br>Retrospective human study on the effects of calcium in out of hospital cardiac arrest patients. Calcium was administered in refractory VF, asystole and PEA either intravenously or intracardiac. Evaluation on the positive effects of calcium administration was the establishment of a pulse or blood pressure immediately after calcium administration.<br>Several weakness points: no control group, lack of down time, administration of multiple drugs (epinephrine, sodium bicarbonate, lidocaine, bretylium, calcium chloride). The author conclude about the beneficial effects of calcium. However, from the data of table 5 it appears that only 1/119 pts in PEA had a long term survival. None of the 52 asystolic patients and none of the 29 pts in VF who received calcium survived. |
| {Kay, 1951, 97} | Kay JH, Blalock A. The use of calcium chloride in the treatment of cardiac arrest in patients. *Surg Gynecol Obstet*. 1951;93:97-102.<br><br>Level 5, human case reports, supporting, poor.<br><br>First human study reporting the effects of calcium during CPR. The study collect four case reports of patients who suffered cardiac arrest episode during anaesthesia in paediatric patients, respectively 5 y.o., 14 m.o., and 18 m.o. the last two ones. No methodology was used. The conditions for which cardiac arrest occurred differed from case to case as well as the treatment and duration of arrest. As a conclusions, the author simply reported that recovery from cardiac arrest was associated to calcium chloride administration. |
| {Meuret, 1983, 1153} | Meuret GH, Schindler HF. [Calcium antagonism - a new pharmacologic principle in resuscitation. Comparison of calcium and calcium antagonists]. *Schweiz Med Wochenschr*. 1983;113:1153-7.<br><br>(Article in German)<br><br>Level 5, opposing, poor.<br><br>The aim of the study is well defined and the randomization appears appropriate. However, the methodology of the animal preparation and the study protocol is poorly described. Only the doses of the drugs are indicated, but not duration of cardiac arrest, start and duration of CPR, how CPR was conducted (manually or by mechanical piston? What was the compression rate?). No measurements of calcium to establish some relationship (if any) to outcome, no data on coronary perfusion pressure or blood gas. |

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| {Meuret, 1984, 108} | Meuret GH, Schindler HF, Scholler KL. [Is calcium indicated in resuscitation? Experimental studies in dogs]. *Anaesthesist*. 1984;33:108-14.<br><br>(Article in German)<br><br>Level 5, opposing, poor<br><br>The study examined the effects of calcium in comparison to those of calcium and adrenaline together in a dog model of anoxic cardiac arrest. Resuscitation maneuvers were performed via a direct massage of the heart. Although there is some methodological weakness due to the double administration thus impeding the evaluation of the calcium administration alone, the results lead to the conclusion that calcium has exerted negative effects on resuscitation rate. |
| {Stueven, 1983, 136} | Stueven H, Thompson BM, Aprahamian C, Darin JC. Use of calcium in prehospital cardiac arrest. *Ann Emerg Med*. 1983;12:136-9.<br><br>Level 2 neutral , fair<br><br>Study oriented to evaluate, retrospectively, the effects of calcium in OOHCA (asystole and PEA) pts. Age, sex, time from onset of complaint to initial treatment, witnessed arrest, treatment before paramedic CPR, significant cardiac history, and cardiac drugs were compared between the groups, either in asystole or in PEA groups. No statistical comparison was made.<br>Results can be summarized as follow:<br>**Asystole**<br>CaCl / No CaCl / Tot.<br>ROSC 8 / 8 / 16<br>NO ROSC 97 / 16 / 113<br>TOT 105 / 24 / 129<br>EER: 97/105 = 0.92 CER: 16/24 = 0.66<br>RR 0.92/0.66 = 1.39<br>ARR = CER – EER = 0.66-0.92 = -0.26<br>RRR = ARR/CER = -0.39<br><br>**PEA**<br>CaCl / No CaCl / Tot.<br>ROSC 10 / 8 / 18<br>NO ROSC 53 / 10 / 63<br>TOT 63 / 18 / 81<br>EER: 53/63 = 0.84 CER: 10/18 = 0.55<br>RR 0.84/0.55 = 1.52<br>ARR = CER – EER = 0.55-0.84 = -0.29<br>RRR = ARR/CER = -0.52<br><br>In both groups, asystole and PEA, both an RR greater than 1 and a negative RRR indicate that the treatment with calcium was associated with |

| | |
|---|---|
| | worst ROSC. |
| {Stueven, 1985, 626} | Stueven HA, Thompson B, Aprahamian C, Tonsfeldt DJ, Kastenson EH. The effectiveness of calcium chloride in refractory electromechanical dissociation. *Ann Emerg Med*. 1985;14:626-9.<br><br>Level 1, neutral , fair<br><br>Prospective, randomized, blinded study investigating calcium versus sodium chloride placebo in refractory EMD (PEA)<br>**P**<br>1a Randomization was performed correctly (vials coded by numbers and colours randomly distributed to the paramedic unit)<br>1b the groups were similar as for as the number is concerned. However, it is not reported whether the groups were similar in age, sex, comorbidity factors, witness CPR.<br>**I**<br>2a Both groups were treated equally with the “intention to treat”. Both received dopamine and isoproterenol without statistical differences.<br>2b Both groups were analyzed in the group to which they entered<br><br>**C**<br>3 The study was double blind. When the code was broken prior to completion of the treatment the patient was excluded.<br><br>**O**<br>Results can be summarized by a 2x2 table:<br><br>\| \| Ca \| NaCl \| Tot. \|<br>\| ROSC \| 8 \| 2 \| 10 \|<br>\| NO ROSC \| 40 \| 40 \| 80 \|<br>\| TOT \| 48 \| 42 \| 90 \|<br><br>EER: 40:48 = 0.83 CER: 40:42 = 0.95<br>RR = 0.83/0.95 = 0.87<br>ARR = (CER – EER) = 0.95 – 0.83 = 0.12<br>RRR = ARR /CER = 0.12/0.95 = 0.13<br>NNT = 1/ARR = 1/0.12 = 8.3<br><br>In the subgroup of patients with wide QRS there were:<br><br>\| \| Ca \| NaCl \| Tot. \|<br>\| ROSC \| 8 \| 1 \| 9 \|<br>\| NO ROSC \| 31 \| 30 \| 61 \|<br>\| TOT \| 39 \| 31 \| 70 \|<br><br>EER: 31:39 = 0.79 CER: 30:31 = 0.97<br>RR = 0.79/0.97 = 0.81<br>ARR = (CER – EER) = 0.97 – 0.79 = 0.18<br>RRR = ARR /CER = 0.18/0.97 = 0.19 |

| | |
|---|---|
| | NNT = 1/ARR = 1/0.18 = 5.6<br><br>From these calculation it appears that calcium promoted a better ROSC as compared to saline placebo. However, the study does not report the effects of the treatment on the other outcomes (survival to event, hospital discharge, intact neurological survival) |
| {Stueven, 1985, 630} | Stueven HA, Thompson B, Aprahamian C, Tonsfeldt DJ, Kastenson EH. Lack of effectiveness of calcium chloride in refractory asystole. *Ann Emerg Med*. 1985;14:630-2.<br><br>Level 1, neutral, fair<br><br>Prospective, randomized, blinded study investigating calcium versus sodium chloride placebo in refractory asystole. Overall, the methodology appears appropriate as in another study of the same group:<br>**P**<br>1a Randomization was performed correctly (the vials were coded by numbers and colours and were randomly distributed to the paramedic unit)<br>1b the groups were similar as for as the number is concerned. Although the description of sex, age witnessed arrest is reported, there is no statistical comparison between the groups to ascertain the absence of statistical differences.<br>**I**<br>2a Both groups were treated equally with the “intention to treat”. Both received dopamine and isoproterenol without statistical differences.<br>2b Both groups were analyzed in the group to which they entered<br><br>**C**<br>The study was double blind. When the code was broken prior to completion of the treatment the patient was excluded.<br><br>**O**<br>Results can be summarized by a 2x2 table:<br><br>Ca NaCl Tot.<br>ROSC 3 1 4<br>NO ROSC 36 33 69<br>TOT 39 34 73<br><br>EER: 36:39 = 0.92 CER: 33:34 = 0.97<br>RR = 0.92/0.97 = 0.95<br>ARR = (CER – EER) = 0.97 – 0.92 = 0.05<br>RRR = ARR /CER = 0.05/0.97 = 0.05<br>NNT = 1/ARR = 1/0.05 = 20 |
| | **Supplementary bibliography** |

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|---|---|
| {Andrivet, 1994, 350} | Andrivet P, Beaslay V, Kiger JP, vu Gnoc C. Complete sinus arrest during diltiazem therapy; clinical correlates and efficacy of intravenous calcium. *Eur Heart J*. 1994;15:350-4.<br><br>Level 5, supporting, poor<br><br>The manuscript reports the effects of calcium chloride administration in patients who had severe node dysfunction leading to complete sinus arrest. Although the study relates to heart arrest condition due to calcium blocker intoxication (diltiazem), none of the patients underwent CPR. Restoration of cardiac function is reported solely due to calcium chloride. This is a case series observation in which there is no control of any variable of the patients studied. Indeed, there is a large variability of the dosage of diltiazem chronically given ranging from 90 to 240 mg/day, of the hemodynamic conditions at admission (some was hypotensive, some had a normal BP), and on the associated therapy (5 pts had amiodarone, 2 had acebutolol). |
| {Hollmann, 2003, 606} | Hollmann MW, Strumper D, Salmons VA, Washington JM, Durieux ME. Effects of calcium and magnesium pretreatment on hyperkalaemic cardiac arrest in rats. *Eur J Anaesthesiol*. 2003;20:606-11.<br><br>Level 5, neutral, fair<br><br>This study investigates the effects of pre-treatment with calcium in hyperkalaemic cardiac arrest rats. Cardiac arrest occurred either as asystole or VF following KCl injection. However, there are no measurements of K prior to KCl administration nor after, so that the levels of blood K are unknown. Accordingly, it is not possible to ascertain the relationship (if any) between blood K and the pharmacological pre-treatment. There were no CPR interventions. |
| {Urban, 1988, 110} | Urban P, Scheidegger D, Buchmann B, Barth D. Cardiac arrest and blood ionized calcium levels. *Ann Intern Med*. 1988;109:110-3.<br><br>Level 4, neutral, fair<br><br>This study was conducted the examine the levels of calcemia in two different groups of patients: the first one (15 pts) related to out-of-hospital cardiac arrest (OOHCA) and the second one (10 pts) to in-hospital cardiac arrest (IHCA). The groups are not comparable due to the several different variables (time of arrest, clinical conditions, treatment, time to intervention).<br>In the first group hypocalcemia and acidosis were documented. However, hypocalcemia, per se, was not associated with worst outcome. A p ns was reported between the two groups.<br>In the IHCA all patients had ROSC and all were successfully discharged. The levels of calcium in this group was close to normal values.<br><br>**OOHCA**: |

Three pts. were excluded because of calcium administration prior to sampling. Five had FV as presenting rhythm, 5 asystole, 2 PEA. Nine of 12 pts in OOHCA received calcium (either 500 or 1000 mg). The results extrapolated from tab. 1 of the paper can be summarized:

| | $Ca^{++}$ given | $Ca^{++}$ not given | Tot. |
|---|---|---|---|
| ROSC | 6 | 0 | 6 |
| No ROSC | 3 | 3 | 6 |
| Total | 9 | 3 | 12 |

EER: 3:9 = 0.33 CER: 3:3 = 1
RR = 0.33/1 = 0.33
ARR = (CER – EER) = 1 – 0.33 = 0.67
RRR = ARR /CER = 0.67/1 = 0.67
NNT = 1/ARR = 1/0.67 = 1.5

| | $Ca^{++}$ given | $Ca^{++}$ not given | Tot. |
|---|---|---|---|
| Hosp. disch. | 4 | 0 | 4 |
| No Hosp. disch | 5 | 3 | 8 |
| Total | 9 | 3 | 12 |

EER: 5:9 = 0.55 CER: 3:3 = 1
RR = 0.55/1 = 0.55
ARR = (CER – EER) = 1 – 0.55 = 0.45
RRR = ARR /CER = 0.45/1 = 0.45
NNT = 1/ARR = 1/0.45 = 2.2

Three patients in VF had ROSC and all had a long term survival. Of the two patients in PEA, one had ROSC but subsequently died, the second one had no ROSC. Among the five patients in asystole, two had ROSC and one had a long term survival.

**IHCA**
One pt was excluded because of calcium administration prior to sampling. Eight had VF as presenting rhythm, one asystole. Nine of 10 pts in IHCA received calcium (either 500 or 1000 mg). The results extrapolated from tab. 1 of the paper can be summarized:

| | $Ca^{++}$ given | $Ca^{++}$ not given | Tot. |
|---|---|---|---|
| ROSC and hosp. disch. | 5 | 4 | 9 |
| No ROSC | 0 | 0 | 0 |
| Total | 5 | 4 | 9 |

EER: 0:5 = 0 CER: 0:4 = 0

Although in OOHCA the results seem to support the administration of calcium, the number of pts treated, especially due to the additional intervention (epinephrine, DF, bicarbonate) do not allow to draw conclusions about the benefit of calcium. In the IHCA pts had ROSC and were all discharged alive regardless the calcium administration.