WORKSHEET for Evidence-Based Review of Science for Veterinary CPCR

1. Basic Demographics

Worksheet author(s)

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2. Clinical question:
MON24:
In dogs and cats with cardiac arrest (P), does the use of a technique for prediction of the likelihood of success of defibrillation (analysis of VF, etc) (I) compared with standard resuscitation (without such prediction) (C), improve outcomes (eg. successful defibrillation, ROSC, survival) (O).

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
Pubmed - (cardiopulmonary resuscitation) AND defibrillation[MeSH Major Topic]) OR ventricular fibrillation analysis[MeSH Major Topic]) AND outcome analysis[MeSH Major Topic]) OR return of spontaneous circulation[MeSH Major Topic]) OR survival[MeSH Major Topic]) OR mortality[MeSH Major Topic]

Pubmed - (cardiopulmonary resuscitation) AND ventricular fibrillation[MeSH Major Topic]) OR defibrillation[MeSH Major Topic]) AND animal[MeSH Major Topic)

CAB - (cardiopulmonary resuscitation) AND (ventricular fibrillation) OR (defibrillation) AND la:(En OR English) AND it:("Journal article")

Pubmed - (Waveform analysis) AND (defibrillation)

CAB - (Waveform analysis) AND (defibrillation)

CAB - (cardiopulmonary resuscitation) AND (English) AND (Journal articles)

4b. Other sources

Wiley Interscience - cardiopulmonary resuscitation in All Fields AND ventricular fibrillation in All Fields OR defibrillation in All Fields

5. Summary of evidence

Evidence Supporting Clinical Question

Good						*Menegazzi, 2004, AD* *Menegazzi, 2003, A* Povoas, 2000, A
Fair			*Kuelz, 1994, A*			*Marn-pernat, 2001, A*
Poor			*Kern, 1988, A, B*			
	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						Li, 2008, A, B
Fair			*Yakaitis, 1980, A*			
Poor						Paradis, 1990, A
	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						Baker, 2008, A, D Jacobs, 2005, A, C
Fair						*Indik, 2009, A, 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

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

In the past 20 years there have been multiple studies examining whether analysis of electrocardiographic data or measures of coronary perfusion can be used to guide the likelihood of success of defibrillation. It is well agreed upon that the ventricular fibrillation(VF) waveform changes from a higher amplitude wavelet (coarse ventricular fibrillation) seen during the initial phase (<3-4 minutes of duration), to a lower amplitude wavelet (fine ventricular fibrillation) with the progression in time. As the amplitude and structure of the VF waveform decreases with the increasing duration of ischemia, it is also seen clinically that there a decreased likelihood of ROSC and survival. It is also known that multiple higher energy countershocks decrease survival, so the goals of these initial studies was to differentiate those patients who are unlikely to respond to an immediate countershock (IC) and therefore provide a different strategy in order to improve survival.

The gold standard in determining the likelihood of response to defibrillation has been shown to be measurement of the coronary perfusion pressure (CPP) (Ref Kern + Paradis). This has been established through laboratory trials although no large scale clinical trial has been performed. The lack of both trials and clinical utility is likely due to the invasiveness and impracticality of cardiac catheterization in critical patients either pre or during cardiac arrest. Due to these difficulties other means for prediction of success have been developed, with the majority focussing on waveform analysis using the ECG (Marn-Pernat, Indik, Li, Menegazzi, Kuelz, Povoas). The goals of waveform analysis have been to determine if there are key components to the VF wavelets that can guide whether there is a high chance of success with single IC at that time. The initial focus to the laboratory investigations was to take the knowledge that larger VF amplitudes are associated with a higher probability of success with IC (kuelz) and to apply this to investigations into both brief and prolonged VF. Due to artifacts produced by movement, chest compressions, electrode placement and recording devices concerns have arisen that direct interpretation of the peak to peak amplitude is likely inaccurate. Because of this multiple different ways to analyze this data, filter the artifacts created by CPR and standardize readings have been created including Hilbert transforms, frequency based measures and scaling exponents. Due to the complexity of these methods their clinical utility has been restricted to the programming of more sophisticated AED's which can determine if the patient has been in prolonged VF or if they are likely to respond to a single IC.

Several initial swine laboratory trials showed a survival benefit to initiating a short period of chest compressions and CPR prior to countershock in cases of prolonged VF (Marn-Pernat, Li Menegazzi, Povoas). This lead to larger randomized clinical trials to determine if waveform analysis and CPR prior to countershock improved outcomes over the current standard of care for IC (Baker, Simpson, Jacobs). The most recent evidence from these large clinical trials did not show any survival benefit to CPR prior to countershock although in one subgroup there may be a potential benefit to those patients in prolonged (> 5 minutes) VF. Due to the scarcity of large randomized clinical trials and the conflicting evidence within these no conclusion on this has been made in human medicine. The latest human guidelines state that CPR should be supplied while setting up and charging the AED or defibrillator, and minimizing the time without chest compressions both pre and post shock is emphasized. Due to the low level of evidence either for or against the practice, no change in the current recommendations was made on delaying defibrillation for a short period of chest compressions in cases of prolonged VF.

7.**Conclusion**

Waveform analysis in association with predicting defibrillation success remains a topic of investigation in human medicine, and holds promise for the future in determining the optimal order of intervention in CPR. Due to the small number of randomized clinical trials in human medicine, and the complete lack of clinical trials in veterinary medicine it is uncertain whether these techniques will improve outcomes in veterinary populations. Based on prior laboratory investigations in dogs and swine however it does appear that coarse VF is more likely to respond with ROSC than fine VF. Further investigations into whether clinical cases of prolonged VF should be treated with a short period of chest compressions prior to defibrillation should be performed before these recommendations can be made.

8. Acknowledgement
Nil

Citation List

Baker P.W, Conway J, Cotton C, Ashby D. T, Smyth J, Woodman R.J, Grantham H, (2008). “Defibrillation or cardiopulmonary resuscitation first for patients with out-of-hospital cardiac arrests found by paramedics to be in ventricular fibrillation? A randomised control trial” Resuscitation 79, 424-431
Summary
Aim: To determine whether in patients with an ambulance response time of >5 min who were in VF cardiac arrest, 3 min of CPR before the first defibrillation was more effective than immediate defibrillation in improving survival to hospital discharge.
Methods: This randomised control trial was run by the South Australian Ambulance Service between 1 July, 2005, and 31 July, 2007. Patients in VF arrest were eligible for randomisation. Exclusion criteria were: (i) <18 years of age, (ii) traumatic arrest, (iii) paramedic witnessed arrest, (iv) advanced life support performed before arrival of paramedics and (v) not for resuscitation order or similar directive. The primary outcome was survival to hospital discharge with secondary outcomes being neurological status at discharge, the rate of return of spontaneous circulation (ROSC) and the time from first defibrillation to ROSC.
Results: For all response times, no differences were observed between the immediate defibrillation group and the CPR first group in survival to hospital discharge (17.1% [18/105] vs. 10.3% [10/97]; P = 0.16), the rate of ROSC (53.3% [56/105] vs. 50.5% [49/97]; P = 0.69) or the time from the first defibrillation to ROSC (12:37 vs. 11:19; P = 0.49). There were also no differences between the immediate defibrillation group and the CPR first group, for response times of ≤ or > 5 min: survival to hospital discharge (50.0% [7/14] vs. 25.0% [4/16]; P = 0.16 or 12.1% [11/91] vs.7.4% [6/81]; P = 0.31, respectively) and the rate of ROSC (71.4% [10/14] vs. 75.0% [12/16]; P = 0.83 or 50.5% [46/91] vs. 45.7% [37/81]; P = 0.54, respectively). No differences were observed in the neurological status of those surviving to hospital discharge.
Conclusion: For patient in out-of-hospital VF cardiac arrest we found no evidence to support the use of 3 min of CPR before the first defibrillation over the accepted practice of immediate defibrillation.
LOE6, Good quality, opposing
Notes: The overall conclusion to this study showed no change in survival if CPR was initiated prior to defibrillation although in a subgroup of patients in which response time of > 5 minutes there was an 18% increase in survival to hospital discharge if CPR was given for 3 minutes prior to defibrillation so it showed a potential benefit in this subgroup.

Indik J, Shanmugasundaram M, Allen D, Valles A, Kern K, Hilwig R, Zuercher M, Berg R, (2009), “Predictors of resuscitation outcome in swing model of VF cardiac arrest: A comparison of VF duration, presence of acute myocardial infarction and VF waveform, Resuscitation 80: 1420-1423,
Introduction: Factors that affect resuscitation to a perfusing rhythm (ROSC) following ventricular fibrillation (VF) include untreated VF duration, acute myocardial infarction (AMI), and possibly factors reflected in the VF waveform. We hypothesized that resuscitation of VF to ROSC within 3 min is predicted by the VF waveform, independent of untreated VF duration or presence of acute MI.
Methods: AMI was induced by the occlusion of the left anterior descending coronary artery. VF was induced in normal (N= 30) and AMI swine (N= 30). Animals were resuscitated after untreated VF of brief (2 min) or prolonged (8 min) duration. VF waveform was analyzed before the first shock to compute the amplitude-spectral area (AMSA) and slope.
Results: Unadjusted predictors of ROSC within 3 min included untreated VF duration (8 min vs 2 min; OR 0.11, 95%CI 0.02–0.54), AMI (AMI vs normal; OR 0.11, 95%CI 0.02–0.54), AMSA (highest to lowest tertile; OR 15.5, 95%CI 1.7–140), and slope (highest to lowest tertile; OR 12.7, 95%CI 1.4–114). On multivariate regression, untreated VF duration (P = 0.011) and AMI (P = 0.003) predicted ROSC within 3 min. Among secondary outcome variables, favorable neurological status at 24 h was only predicted by VF duration (OR 0.22, 95% CI 0.05–0.92).
Conclusions: In this swine model of VF, untreated VF duration and AMI were independent predictors of ROSC following VF cardiac arrest. AMSA and slope predicted ROSC when VF duration or the presence of AMI were unknown. Importantly, the initial treatment of choice for short duration VF is defibrillation regardless of VF waveform.
LOE 6, Fair, Opposing

Jacobs I, Finn J, Oxer H, Jelinek G, (2005) “CPR before difibrillation in out-of-hospital cardiac arrest: A randomized trial, Emergency Medicine Australasia 17: 39-45
Current resuscitation guidelines recommend that defibrillation be undertaken as soon as possible in patients suffering a cardiac arrest where the cardiac rhythm is either ventricular fibrillation (VF) or ventricular tachycardia (VT). Evidence from animal and clinical studies suggests that outcomes may be improved if a period of cardiopulmonary resuscitation (CPR) is given prior to defibrillation. The objective of this study was to determine if 90 seconds of CPR before defibrillation improved survival.
Methods:
Patients suffering non-paramedic witnessed VF/VT cardiac arrest were randomized to receive either 90 seconds of CPR before defibrillation (treatment) or immediate defibrillation (control). The study was carried out in Perth, Western Australia between June 2000 and June 2002. The primary endpoint was survival to hospital discharge with secondary endpoints of return of spontaneous circulation (ROSC) and survival at 1 year.
Results:
A total of 256 patients underwent randomization. Baseline characteristics including response intervals were similar in both groups. Survival to hospital discharge in the CPR first group was 4.2% (5/119) compared with 5.1% (7/137) for the immediate defibrillation group (OR 0.81; 95%CI. 0.25–2.64). No difference in those achieving ROSC was observed between the groups (OR 1.16; 95% CI 0.49–2.80).
Conclusion:
Ninety seconds of CPR before defibrillation does not improve overall survival in patients suffering VF/VT cardiac arrests. Further studies to evaluate various aspects of this treatment strategy are required as published outcomes to date are inconclusive.
LOE6, RTC - good quality, opposing

Note : This paper was a randomized trial although was slightly underpowered as they did not reach the estimated goal number of patients of 390 (they recruited 256 patients in their 2 year trial period). They did otherwise have a well designed trial with no significant benefit noted with CPR given prior to defibrillation.

Kern K, Ewy G, Voorhees W, Babbs C, Tacker W, (1988), “Myocardial perfusion pressure: A predictor of 24-hour survival during prolonged cardiac arrest in dogs, Resuscitation 16: 241-250
Myocardial perfusion pressure, defined as the aortic diastolic pressure minus the right atrial diastolic pressure, correlates with coronary blood flow during cardiopulmonary resuscitation (CPR) and predicts initial resuscitation success. Whether this hemodynamic parameter can predict 24-h survival is not known. We examined the relationship between myocardial perfusion pressure and 24-h survival in 60 dogs that underwent prolonged (20 min) ventricular fibrillation and CPR. Forty-two (70%) animals were initially resuscitated and 20 (33%) survived for 24 h. Myocardial perfusion pressure was significantly greater when measured at 5, 10, 15 and 20 min of ventricular fibrillation in the resuscitated animals than in the non-resuscitated animals ($P < 0.01$). Likewise, the myocardial perfusion pressure was also greater in the animals that survived 24 h than in animals that were resuscitated, but died before 24 h ($P < 0.02$). Myocardial perfusion pressure measured after 10 min of CPR was 11 ± 2 mmHg in animals never resuscitated, 20 ± 3 mmHg in those resuscitated that died before 24 h and 29 ± 2 mmHg in those that survived 24 h ($P < 0.05$). A myocardial perfusion pressure at 10 min of CPR of 20 mmHg or less is an excellent predictor of poor survival (negative predictive VALUE = 96%). Myocardial perfusion pressure is a useful index of CPR effectiveness and therefore may be a useful guide in helping to optimize resuscitation efforts.
LOE 3, poor, supporting

Kuelz K, Hsia P, Wise R, Mahmud R, Damiano R, (1994), “Integration of absolute vetricular fibrillation voltage correlates with successful defibrillation” IEEE Transaction on biomedical engineering, 41: 8, 782-791
Abstract-Previous work has suggested that at higher absolute ventricular fibrillation voltages(AVFV),the heart is more amenable to defibrillation. This study investigated in a canine model whether voltage integration of the AVFV is associated with the defibrillationSuccess rate. The moving average filter was used to process the ventricular fibrillation (VF) waveform recorded from Lead I1 of the electrocardiogram (ECG). In seven animals, defibrillation trials were analyzed using a dc shock (DCS) successful approximately 50% of the time when delivered randomly. For each of a total of 84 DCS (40% successes, 60% failures), the fibrillation waveform just prior to DCS was analyzed. The integration of the AVFV waveform was performed over various sample sizes including 1, 4, 8, 16, 64, and 128 ms, as well as the time equal to the mean VF cycle length. The results suggest that dc shocks delivered at instants of higher values of integrated AVFV over the various window sizes are associated with successful defibrillation. Window sizes less than 16 ms appeared to offer the best discrimination. The integration of AVFV over the entire VF cycle length was significantly higher for successful rather than unsuccessful DCS. This interesting observation is consistent with the clinical observation that “coarse” VF (high AVFV) is easier to defibrillate than “fine” VF (low AVFV). The use of voltage integration of AVFV may have potential implications in the improvement of defibrillation success in implantable devices.
LOE3, fair, supporting

Li Y, Ristagno G, Bisera J, Tang W, Deng Q, Weil M, (2008), “Electrocardiogram waveforms for monitoring effectiveness of chest compression during cardiopulmonary resuscitation”, Critical Care Medicine 36: 211-215
Background: Newer guidelines address the importance of effective chest compressions, citing evidence that this primary intervention is usually suboptimally performed during cardiopulmonary
resuscitation. We therefore sought a readily available option for monitoring the effectiveness of chest compressions, specifically using the electrocardiogram.

Methods and Results: Ventricular fibrillation was induced by coronary artery occlusion and untreated for 5 mins. Male domestic pigs weighing 40 2 kg were randomized to optimal or suboptimal chest compressions after onset of ventricular fibrillation. Optimal depth of mechanical compression in six animals was defined as a decrease of 25% in anterior posterior diameter of the chest during compression. Suboptimal compression, also in six animals, was defined as a decrease of 17.5% in anterior posterior diameter. For each group, the chest compressions were maintained at a rate of 100 per min. After 3 mins of chest compression, defibrillation was attempted with a 150-J biphasic shock. All animals had return of spontaneous circulation after optimal compressions. This contrasted with suboptimal compressions, after which none of the animals had return of spontaneous circulation. Amplitude spectrum area values, representing the electrocardiographic amplitude frequency spectral area computed from conventional precordial leads, like coronary perfusion pressure and end tidal PCO2, were predictive of outcomes.
Conclusion: The effectiveness of chest compressions was reflected in the amplitude spectrum area values. Accordingly, the amplitude spectrum area predictor may be incorporated in current automated external defibrillators to monitor and prompt the effectiveness of chest compression during cardiopulmonary resuscitation.
LOE 6, RCT, good quality, neutral

Marn-Pernat A, Weil M, Tang W, Pernat A, Bisera J, (2001), "Optimizing timing of ventricular defibrilation", Critical Care Medicine 29: 2360-2365,
Objective: Our intent was to evolve a prognosticator that would predict the likelihood that an electrical shock would restore a perfusing rhythm. Such a prognosticator was to be based on conventional electrocardiographic signals but without constraints caused by artifacts resulting from precordial compression. The adverse effects of "hands off" intervals for rhythm analyses would therefore be minimized. Such a prognosticator was further intended to reduce the number of electrical shocks and the total energy delivered and thereby minimize post resuscitation myocardial dysfunction. Design: Observational study.
Subjects: Medical research laboratory of a university-affiliated research and educational institute.
Subjects: Domestic pigs. Interventions: Ventricular fibrillation was induced in an established porcine model of cardiac arrest. Recordings of scalar lead 2 over the frequency range of 4–48 Hz were utilized. The area under the curve representing the amplitude and frequency was defined as the amplitude spectrum area (AMSA). Measurements and Main Results: A derivation group of 55 animals yielded a threshold value of AMSA that uniformly predicted successful resuscitation. A separate group of 10 animals, a validation group, confirmed that an AMSA value of 21 mV·Hz predicted restoration of perfusing rhythm after 7 of 8 electrical shocks and failure of electrical conversion in 21 of 23 electrical shocks, yielding sensitivity and specificity of about 90%. The negative predictive value of AMSA was 95% and statistically equivalent to that of coronary perfusion pressure, mean amplitude, and median frequency. The positive predictive value that would prompt continuation of cardiopulmonary resuscitation without interruption for an unsuccessful defibrillation attempt was greatly improved with AMSA (78%) as compared with coronary perfusion pressure (42%), mean amplitude (32%), and median frequency (29%). Conclusion: AMSA has the potential for guiding more optimal timing of defibrillation without adverse interruption of cardiopulmonary resuscitation or the delivery of unsuccessful high energy electrical shocks that contribute to postresuscitation myocardial injury.
LOE6, Fair, Supportive

Menegazzi J, Callaway C, Sherman L, Hostler D, Wang H, Fertig K, Logue E, (2004), "Ventricular fibrillation scaling exponent can guide timing of defibrillation and other therapies, Circulation 109: 926-931
Background—The scaling exponent (ScE) of the ventricular fibrillation (VF) waveform correlates with duration of VF and predicts defibrillation outcome. We compared 4 therapeutic approaches to the treatment of VF of various durations.

Methods and Results—Seventy-two swine (19.5 to 25.7 kg) were randomly assigned to 1 of 9 groups (n8 each). VF wasinduced and left untreated until the ScE reached 1.10, 1.20, 1.30, or 1.40. Animals were treated with either immediate
countershock (IC); 3 minutes of CPR before the first countershock (CPR); CPR for 2 minutes, then drugs given with 3 more minutes of CPR before the first shock (CPR-D); or drugs given at the start of CPR with 3 minutes of CPR before the first shock (DrugsCPR). Return of spontaneous circulation (ROSC) and 1-hour survival were analyzed with 2 and Kaplan-Meier survival curves. IC was effective when the ScE was low but had decreasing success as the ScE increased. No animals in the 1.30 or 1.40 groups had ROSC from IC (0 of 16). CPR did not improve first shock outcome in the 1.20 CPR group (3 of 8 ROSC). Kaplan-Meier survival analyses indicated that IC significantly delayed time to ROSC in both the 1.3 (P0.0006) and the 1.4 (P0.005) groups.
Conclusions—VF of brief to moderate duration is effectively treated by IC. When VF is prolonged, as indicated by an ScE of 1.3 or greater, IC was not effective and delayed time to ROSC. The ScE can help in choosing the first intervention in the treatment of VF
LOE6, good quality, supporting

Paradis N, Martin G, Emanuel P, Goetting M, Appleton T, Feingold M, Nowak R, (1990), "Coronary perfusino pressure and the return of spontaneous circulation in human cardiopulmonary resuscitation", JAMA 263; 1106-1113
Coronary perfusion pressure (CPP), the aortic-to-right atrial pressure gradient during the relaxation phase of cardiopulmonary resuscitation, was measured in 100 patients with cardiac arrest. Coronary perfusion pressure and other variables were compared in patients with and without return of spontaneous circulation (ROSC). Twenty-four patients had ROSC. Initial CPP (mean +\-SD) was in those with in those without ROSC and 25.6 +\- 7.7mm Hg in those with ROSC. Differences were also found for the maximal aortic relaxation pressure, the compression-phase aortic-to right atrial gradient, and the arterial Po2. No patient with an initial CPP less than 0 mm Hg had ROSC. Only patients with maximal CPPs of 15 mm Hg or more had ROSC, and the fraction of patients with ROSC increased as the maximal CPP increased. A CPP above 15 mm Hg did not guarantee ROSC, however, as 18 patients whose CPPs were 15 mm Hg or greater did not resuscitate. Of variables measured, maximal CPP was most predictive of ROSC, and all CPP measurements were more predictive than was aortic pressure alone. The study substantiates animal data that indicate the importance of CPP during CPR.
LOE6, poor, neutral

Povoas H, Bisera J, (2000), "Electrocardiographic waveform analysis for predicting the success of defibrillation", Critical Care Medicine 28: 11, 210-214
A new electrocardiographic predictor of the likelihood that an electrical shock would restore a perfusing rhythm is described. The intent was to develop a prognosticator that would be displayed during precordial compression. We anticipated that such a predictor would allow more selective timing of electrical shocks and reduce electrical injury to the myocardium caused by repetitiveshocks. In a porcine model of cardiac arrest because of ventricular fibrillation, electrocardiographic recordings of ventricular fibrillation wavelets were analyzed and transformed into an amplitude spectrum area (AMSA). An AMSA value of 21 mVzHz predicted restoration of perfusing rhythm with a positive predictive value equivalent to that of coronary perfusion pressure. More important, the negative predictive value that a shock would fail to reestablish spontaneous circulation was 96%. AMSA, therefore, has the potential for guiding optimal timing of defibrillation
LOE6, Good, supporting

Yakaitis R, Ewy G, Otto C, Taren D, Moon T, (1980) "Influence of time and therapy on ventricular defibrillation in dogs" Critical Care Medicine 8: 157-163

AB Factors that may influence energy requirements for ventricular defibrillation include the duration of fibrillation and the mode of resuscitation. The present study assesses the effect of these influences on the energy needed for defibrillation. Dogs were anesthetized, and arterial blood pressure and Lead II of the ECG were continuously recorded. Ventricular fibrillation was electrically induced in each dog for a period of 1, 3, 5, or 9 min. Three resuscitation techniques were evaluated: precountershock artificial ventilation (AV) and closed-chest cardiac massage (CCCM); precountershock AV/CCCM and epinephrine, 1 mg IV; and countershock without preliminary AV/CCCM or epinephrine. Each animal was shocked with successive doses of 1, 2, 4, and 8 J/kg, ceasing when either electrical conversion occurred or after the maximum dose had been delivered. If defibrillation was unaccompanied by resumption of spontaneous circulation (systolic pressure > 60 mm Hg > 2 min), AV/CCCM was administered for 1 min. In general, the incidence of defibrillation was inversely proportional to the duration of fibrillation. Epinephrine had no significant effect on the energy dose needed for conversion. After 2 min of fibrillation, however, epinephrine became increasingly important for restoration of circulation. The technique of immediate countershock was effective for episodes of fibrillation limited to approximately 3 min. Regardless of therapy, for intervals of fibrillation of up to 9 min, Gompertz data curves indicated that a delivered energy of 4-5 J/kg is the approximate energy dose associated with the maximum achievable incidence of defibrillation within the limits of this experimental protocol
LOE 3, fair, neutral