What is the optimal chest compression-ventilation ratio?
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**Purpose of review**
Despite a more widespread knowledge of basic cardiopulmonary resuscitation maneuvers in the community, the survival rate for patients with cardiac arrest has remained essentially unchanged in the past 30 years. Over the past few decades, many different compression-ventilation ratios have been studied in terms of best coronary and cerebral oxygen delivery, restoration of spontaneous circulation, and neurologic outcome. This article summarizes the recent evidence presented at the International Consensus on Resuscitation Science in January 2005.

**Recent findings**
Recent data from animal and mathematical models suggest a move to a higher compression-ventilation ratio to maximize coronary and cerebral oxygen delivery during cardiac arrest and long-term neurologic outcome. Prospective randomized human data on alternative compression-ventilation ratios are missing and new evidence seems to indicate the inadequacy of both lay and professional rescuers in providing chest compression and ventilating the victim in cardiac arrest. Finally, observational and animal studies highlight the hidden danger of inadvertent hyperventilation during advanced cardiac life support as a reduction of both coronary and perfusion pressure secondary to increased intrathoracic pressure and decreased venous return.

**Summary**
The optimal compression-ventilation ratio is still unknown and the best tradeoff between oxygenation and organ perfusion during cardiopulmonary resuscitation is probably different for each patient and scenario. A discrepancy between what is recommended by the current guidelines and the ‘real world’ of cardiopulmonary resuscitation has resulted in a near flat survival rate from cardiac arrest in the past few years.

**Keywords**
cardiopulmonary resuscitation, compression-ventilation ratio

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**Introduction**
Survival rates from cardiac arrest remain poor despite the development of both cardiopulmonary resuscitation (CPR) and electrical defibrillation as treatment modalities over the past 50 years \[1,2\]. Approximately 1–6\% of patients having out-of-hospital cardiac arrest ultimately survive the event, and although survival rates are somewhat better for in-hospital arrest patients, a recent comprehensive report observed that only 17\% of these patients were discharged alive \[3–6\].

The goal of CPR is to generate sufficient oxygen delivery to the coronary and cerebral circulation while waiting for a definitive restoration of a cardiac rhythm by defibrillation, pharmacologic intervention, or both. Coronary perfusion is a determinant of return of spontaneous circulation (ROSC) and cerebral perfusion is a determinant of neurologic outcome in both in-hospital and out-of-hospital cardiac arrest.

Because cerebral and coronary perfusion are strictly related to chest compression, optimal quantity and quality of chest compression are fundamental to survival from cardiac arrest. This review discusses the up-to-date evidence-based medicine available on the role of chest compression during CPR and its ratio with rescue breathing.

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**The need for chest compression to provide organ perfusion during cardiac arrest**
Chest compressions for CPR are serial, rhythmic applications of pressure over the lower half of the sternum, the circulatory base for basic life support (BLS). Ideally, blood circulating to the lungs by chest compressions, accompanied by properly performed rescue breathing, supports delivery of oxygen to the heart and brain and other vital organs until circulation is restored. Left ventricular stroke volume remains relatively constant during chest compression. As a result, left ventricular dimensions...
decompression phase, as unnecessary intrathoracic pres-

Recently, a greater emphasis has been placed on 

It is commonly accepted, 

There is no significant rate-related difference in the com-

Unfortunately, interpretation of data from animal studies 

Sternal compression force is generally gauged as adequate 

Although this validation of pulses requires at least two 

Sternal compression force is generally gauged as adequate 

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Sure can adversely affect coronary and cerebral perfusion 

Are we performing correct chest compression? No

Although US and international guidelines continue to rec-

Recent laboratory data, however, point toward rescuer fa-

Characteristics of the correct chest compression

Although a straightforward maneuver, chest compression 

Coronary electromagnetic flow probes in animals in cardiac 

Optimal compression-ventilation ratio
Ventilation in basic life support: a tradeoff between oxygenation and organ perfusion during cardiopulmonary resuscitation

Although it is now clear that bystander CPR improves the chances of successful defibrillation and better neurologic outcome survival compared with no CPR [25–30], in recent years the necessity of ventilation during CPR has come under close scrutiny. It is now accepted that during cardiac arrest, mixed venous blood gases reflect tissue oxygenation and acid-base state more accurately than arterial blood gas [31]. Therefore, a minute ventilation that is considerably less than that required in the presence of a normal cardiac output may produce normal arterial blood gases and provide adequate exchange of alveolar gas during CPR.

One cannot attempt to define the ideal C-V ratio for BLS without considering quality and quantity of ventilation required during cardiac arrest. Current experimental and clinical evidence seems to deemphasize the importance of ventilation during the initial period of CPR. Abundant animal data in the early 1990s showed that when cardiac arrest was treated only with chest compressions, without active ventilation, the average arterial blood saturation was maintained at about 70% for up to 10 minutes during CPR [32]. More experimental work showed that large animal survival and neurologic outcome up to 48 hours were not different when ventilation was withheld during resuscitation. These initial studies, although clearly deemphasizing the importance of ventilation during the first few minutes of CPR, were limited by the persistence of an artificial patent airway in the animal, which resulted from the kinked nature of the airway [33,34]. In summary, during longer periods of CPR it has been stated that some ventilatory support is critical for successful resuscitation outcomes [35], but for the first few minutes of dysrhythmic cardiac arrest ventilation is probably not vital.

Ventilation is still beneficial in improving arterial blood gas [34] and 24-hour neurologic outcome [36] in pure asphyxial animal models (clamping of the endotracheal tube), a condition used often to simulate pediatric cardiac arrest but impossible to distinguish in most adult out-of-hospital cardiac arrests. In general, attempting ventilation in adult patients with cardiac arrest [35–37] results in long interruptions in circulation during CPR, which appear potentially to compromise the success of cardiac resuscitation [38,39]. The number of chest compressions per minute decreases when assisted ventilation is included in the model of bystander CPR [40,41]. Moreover, maneuvers aimed at increasing proficiency of ventilation usually decrease the speed of chest compression [42]. The transition from chest compression to ventilation in single-rescuer CPR further decreases the number of chest compressions delivered. In a recent study, in which lay rescuers were videotaped while performing CPR on a mannequin with a C – V ratio of 15 : 2, rescuers compressed the chest only 39 times per minute. In an 8-minute period of standard CPR compared with continuous chest compression CPR (CCC-CPR) on mannequins, the CCC-CPR group received 675 total compressions compared with 308 compressions for the standard CPR group [41]. The ‘need’ for ventilation during CPR is also a deterrent for lay rescuers to perform any rescue at all, because many are reluctant to perform mouth-to-mouth ventilation due to widespread concerns about transmission of infectious diseases [43–45]. In the bystander setting, it is possible that the increased victim survival rates with dispatcher-instructed chest compression alone vs dispatcher-instructed standard CPR reflect the unwillingness of rescuers to perform ventilation due to fear of infection transmission [46]. Finally, a minor potential consequence of rescue ventilation during mouth-to-mouth ventilation is the rescuer hyperventilation syndrome, presumably a hypocapnic state causing subjective fatigue and dizziness, exacerbated by the rescuer taking a deep breath before exhaling in the victim’s mouth [47,48]. In summary, although both ventilation and chest compression theoretically contribute to oxygen delivery, they are antagonizing each other during BLS CPR.

Are we performing correct ventilation? No

A statement from the American Heart Association (AHA) Guidelines 2000 recommends the C-V for one-lay and two-lay rescuer adult CPR to be 15 compressions to two ventilations when the victim’s airway is unprotected for both lay and professional rescuers (airway protection = successful placement of endotracheal tube). The suggested ‘ventilation time’ is 1.5–2 seconds after a deep inspiration. Although this ventilation time target is relatively easy to achieve with an endotracheal tube or an alternative airway in place, the average lay rescuer seems to average the endless time of 8 seconds for each breath provided (16 seconds for two ventilations and 30 seconds for five) when tested using a mannikin, only a few months after taking a CPR class. This observation explains the reported overall 50% gain when a 50 : 5 ratio was compared with 15 : 2 for single-rescuer CPR on mannikins [49]. The implications of increased ventilation time for blood circulation during cardiac arrest are obvious and disastrous. For example, if a layperson requires a 16-second break in the compressions for two mouth-to-mouth ventilations, there will be no circulation for 60 and 35% of the total resuscitation time for the ratios...
15 : 2 and 50 : 2, respectively. The optimal number of ventilations to provide in series is not known. In a manikin study when use of the BVM is compared between a 15 : 2 and 50 : 5 ratio, the predicted 25% reduction of ventilation time in the 50 : 5 group could not be demonstrated [50].

Multiple ventilations in series have the theoretical advantage of progressively improving rescuer efficiency in ventilating, but this advantage must be balanced with the decreased blood flow to the victim and fatigue and hyperventilation of the rescuer. A simplified three-staged approach to CPR has been used to improve the ventilation learning curve and the overall number of chest compressions provided [49,51,52,53] but it has not been widely validated in the BLS training community, where learning retention is dismal. Intuitively, the easier way to achieve lay rescuer skill retention after CPR training would be simply to eliminate ventilation from the teaching, a ‘radical’ approach recently proven effective [54*]. Once the airway is secure (protected by insertion of endotracheal tube), compressions and ventilations can be asynchronous, with recommended ventilation rate of about 12–15 breaths/min. Excitement and overenthusiasm of the professional rescuer at the scene of cardiac arrest can increase the chance of unnecessary hyperventilation. It has become clear by anecdotal observations in intubated patients that very high ventilation frequencies can significantly increase intrathoracic pressure and decrease organ perfusion during CPR [55**]. When ventilation rates of 20/min or higher are provided through an endotracheal tube, it is deadly in an animal model of cardiac arrest [55**].

The effect of high ventilatory rate when the airway is not protected by an endotracheal tube is unclear. The likely presence of an unquantified airway leak is a protective mechanism that decreases the deleterious effect of high ventilatory rate on the cardiovascular system of the victim. No matter how ventilation is provided, a consistent message is needed for BLS and advanced life support (ALS) providers emphasizing that although ventilation can be important, hyperventilation is always harmful.

**Compression-ventilation ratio: the science**

The current closed-chest compression method was introduced into clinical practice 45 years ago [7,56]. Early experimental studies suggested a C-V ratio of 4 : 1 [57–59]. Being apparently a simple technique, CPR became the method of choice for the resuscitation of a patient in cardiac arrest. The first official guideline for the treatment of patients with cardiac arrest was available only in the 1970s [60] and then was updated in 1980 [61] and 1986 [62], focusing on the importance of chest compressions. By 1992, published guidelines reflected a new emphasis on early defibrillation and relegated chest compression to a secondary role [63]. At that time C-V ratios of 5 : 1 and 15 : 2 were the proposed standards for adult CPR, depending on the presence of a single or dual rescuers.

By the year 2000, the AHA Guidelines for Resuscitation suggested a simpler unified 15 : 2 approach for adult CPR by one or two lay rescuers, leaving the 5 : 1 ratio as an alternative only for pediatric resuscitation [64]. Despite recent emphasis on ventilation and administrative deregulation of the use of automatic external defibrillation (AED) by the lay rescuers, the survival rate for patients with cardiac arrest, especially after prolonged arrest times [2–4], has remained essentially unchanged. Although the causes of this poor outcome are still under discussion, the issues of inadequate provision of chest compressions and the most advantageous C-V ratio has been the focus of renewed attention [41].

**Mathematical models**

A simplistic approach to correlation between C-V ratios and oxygen delivery during CPR has been attempted with mathematical speculations in both adult and pediatric cardiac arrest. Intuitively, all these models suggest a move to a higher C-V ratio. Optimal number of chest compressions in adults was between 30–70 : 2, based on ‘standard AHA’ (two seconds/breath) vs ‘real world’ (8 seconds/breath) performance of the rescuer [65]. A 50 : 2 ratio was found to be adequate for single-rescuer and two-rescuer basic CPR [65]. CCC-CPR was associated with the greatest oxygen delivery during the first 2 minutes of CPR but resulted in grossly hypoxic arterial blood after 6 minutes of CPR. A 5 : 1 ratio, despite producing high arterial blood oxygen levels, resulted in lowest oxygen in all the models [65,66]. Ratios of 15 : 2 and 50 : 5 were approximately equivalent in terms of gas exchange. When the model was applied to a speculative ‘asphyxial’ cardiac arrest with an ideal ventilation time (2 seconds per breath), a 20 : 1 ratio resulted in the best compromise of oxygen delivery, arterial oxygen tension, arterial carbon dioxide tension, partial pressure of oxygen in mixed venous blood, and partial pressure of carbon dioxide in mixed venous blood [67] for mouth-to-mouth ventilation and bag-valve mask ventilation (BVM) oxygen.

Recently we have developed a simple mathematical analysis to predict minute time distribution of chest compression and ventilation to the victim in cardiac arrest at different ratios and with one or two rescuers. Two figures are presented that list a number of possible ventilation/compression ratios and the number of compressions and ventilations that can be delivered to a patient in 1 minute (Figs. 1 and 2). Several assumptions are built into the figures that model what occurs during CPR given by humans when it has been measured. For example, the ‘hands-off’ interval has been measured to range from 10–20 seconds per each minute of CPR. The assumptions used in Fig. 1 for one rescuer only are the following: a compression rate
of 100/min, a 2-second time to open the airway followed by 2 seconds for each ventilation provided to the victim. A 2-second exhalation time follows the first breath in and 1 second is ‘wasted’ to return to chest compression.

For two professional rescuers, the only different assumption is an immediate access to the airway. In this latter case the best scenario implies no time wasted to open the airway, a ventilation time of 2 seconds for breath, and a ‘wasted’ time of 1 second to resume chest compressions.

Although theoretically useful to explain pathophysiology of oxygen delivery at different C : V ratios, mathematical models have not been validated by animal studies, in which arterial and venous desaturation during BLS is usually more profound than estimated values [65,66]. A healthy cardiac output and pulmonary status are presumed to preexist the arrest and the lack of calculation for massive pulmonary shunting during CPR makes these models in general overly optimistic [39].

**Animal models**

Data from out-of-hospital cardiac arrest registry confirm better outcome in patients performing 15 : 2 CPR if compared with no CPR. No comparison was made, however, between different C : V ratios in human settings [27–30]. Human studies of CPR have been limited due to ethical concerns. The few available in the literature have been generally biased by the use of general anesthesia and paralysis, where gasping is not allowed. Conversely, most animal models, like the pig, have been limited by misrepresentation of the airway whereas in cardiac arrest, usually straight and open in the intubated animal while likely obstructed in humans [68]. In fact, only in animal models has a free exchange of small amounts of tidal volume associated with chest compression been observed. Nevertheless, most of the information on this subject is extrapolated from animal studies.

Based on the new understanding of physiopathology of compression and ventilation during CPR previously discussed, some researchers have begun to explore other C – V ratios with much longer series of uninterrupted compressions such as 50 : 5 [50,52], 30 : 2 [65,69**,70], 100 : 5 [65,66,69**,70,71], or even CCC-CPR [33,39,72–74]. Sanders et al. [71] in 2002 evaluated 24-hour survival and neurologic outcomes in four models of CPR after 3 minutes of induced ventricular fibrillation without any resuscitation effort by using different C-V ratios: 15 : 2 (standard CPR); CC-CPR (chest compressions only with no ventilations for 12 minutes), 50 : 5-CPR (CPR with a ratio of 50 : 5 compressions to ventilations), and 100 : 2-CPR (4 minutes of chest compressions only followed by CPR with a ratio of 100 : 2 compressions to ventilations) performed for 12 minutes. Although the number of animals was small in this experiment, no statistically significant difference in survival was found among the groups, consistent with other similar studies in the literature [75–77]. Remarkably, neurologic deficit was significantly lower in the 100 : 2-CPR group compared with that in the standard CPR group, as well as that in the CC-CPR group. In this 12-minute CPR model, the standard CPR group received a total of 720 chest compressions compared with 1116 chest compressions in the 100 : 2-CPR group. This advantage in total perfusion over time might have been responsible for the improved neurologic outcome in the 100 : 2-CPR group. The minute ventilation in the CC-CPR group was significantly less than that in the 100 : 2-CPR group but not zero, again showing that ventilation (anecdotally...

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**Figure 1. Mathematical prediction of compression and ventilation time at different ratios**

<table>
<thead>
<tr>
<th>Breath (sec)</th>
<th>C/V</th>
<th>5 Sec</th>
<th>7 Sec</th>
<th>10 Sec</th>
<th>15 Sec</th>
<th>20 Sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>15:2</td>
<td></td>
<td>56/8</td>
<td>47/6</td>
<td>38/5</td>
<td>31/4</td>
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<tr>
<td>15:1</td>
<td></td>
<td>64/4</td>
<td>47/3</td>
<td>38/3</td>
<td>31/2</td>
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</tr>
<tr>
<td>20:1</td>
<td></td>
<td>70/4</td>
<td>55/3</td>
<td>44/2</td>
<td>38/2</td>
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</tr>
<tr>
<td>30:2</td>
<td></td>
<td>75/5</td>
<td>64/4</td>
<td>54/4</td>
<td>47/3</td>
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</tr>
<tr>
<td>50:2</td>
<td></td>
<td>81/3</td>
<td>75/3</td>
<td>66/3</td>
<td>60/2</td>
<td></td>
</tr>
</tbody>
</table>

One-minute cycle and one-layperson rescuer. Grid pattern: best case cardiopulmonary resuscitation (CPR) scenarios. Shaded pattern: more realistic CPR scenarios.

**Figure 2. Mathematical prediction of compression and ventilation time at different ratios**

<table>
<thead>
<tr>
<th>Breath (sec)</th>
<th>C/V</th>
<th>3 (Best case for 1 breath)</th>
<th>4</th>
<th>5 (Best case for 2 breaths)</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
</tr>
</thead>
<tbody>
<tr>
<td>15:2</td>
<td></td>
<td>64/9</td>
<td>60/8</td>
<td>56/8</td>
<td>53/7</td>
<td>47/6</td>
<td>38/5</td>
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<tr>
<td>15:1</td>
<td></td>
<td>75/5</td>
<td>60/4</td>
<td>56/4</td>
<td>56/4</td>
<td>47/3</td>
<td>38/2</td>
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<tr>
<td>20:1</td>
<td></td>
<td>80/4</td>
<td>67/3</td>
<td>63/3</td>
<td>60/3</td>
<td>54/3</td>
<td>44/2</td>
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<td>30:2</td>
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<td>78/5</td>
<td>75/5</td>
<td>72/5</td>
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<td>64/4</td>
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<tr>
<td>50:2</td>
<td></td>
<td>86/3</td>
<td>83/3</td>
<td>81/3</td>
<td>79/3</td>
<td>75/3</td>
<td>67/3</td>
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</tbody>
</table>

One-minute cycle and two professional rescuers. Bag ventilation mask is used. Grid pattern: best case cardiopulmonary resuscitation (CPR) scenarios. Shaded pattern: more realistic CPR scenarios.
An attempt to reproduce a more physiologic model of CPR in human was performed by Dorph et al. [70] in 2003. In this model an impedance threshold valve (Resusci-Valve CPRx, LLC, Minneapolis, MN) was placed to simulate a human airway collapsed and preventing passive inspiratory airway flow generated by thumper precordial compression, with a ventilation time of 2 seconds. A ventilation ratio of 15 : 2 gave better pulmonary gas exchange and cerebral oxygen delivery than both the ratios 50 : 2 and 50 : 5. The arterial oxygen saturation stayed above 80% throughout CPR for 15 : 2, but it dropped below 40% during part of the ventilation – compression cycle in the other two ratios. Conversely, the ratio of 50 : 2 produced 30% more chest compressions per minute than either of the two other methods, with a parallel higher carotid blood flow per minute than 50 : 5. Cerebral Doppler blood flow seemed to be best at 50 : 2, indicating that well-oxygenated blood is of no help if it does not reach the tissues.

When these parameter were projected for 16 seconds instead of 4 seconds per two ventilations, a C-V ratio of 30 : 2 seemed the best compromise. This promising ratio was investigated later in a similar animal model, in which cortical blood flow was accurately measured through a skull window with laser Doppler flowmetry. Continuous chest compression was compared with a 30 : 2 ratio for coronary perfusion pressure, cerebral cortical blood flow, and carotid oxygen delivery. Again, this study would differ from all previous animal models as upper airway inspiration obstruction was simulated with an impedance threshold valve. No hemodynamic difference was found between the two ratios, but oxygen delivery data for 30 : 2 were far superior and closely matched the values for 15 : 2 ratio. If chest compressions were administered alone, the arterial blood was in fact virtually desaturated within 1.5–2 minutes. This decline was significantly attenuated by the interposition of two ventilations with expired air following every 30th compression.

It can be argued that the time for ventilation, a mean of 4.8 seconds with a ratio of 30 : 2, made that group come out too favorably, as a recent report on lay rescuer CPR performance showed a mean break in chest compressions for rescue breathing of 16 seconds for two breaths [39,41]. It is striking, however, how this more realistic animal model resembles the mathematical speculation of Babbs and Kern [65]. Although it was easier to resuscitate the ventilated pigs, all except one pig eventually achieved ROSC. These results differed from the findings of three independent, well-established laboratories where chest compressions only were found as effective as chest compressions plus rescue breathing during the initial 6–12 minutes of CPR [32–35]. The major reason for the difference in results is probably the persistence of an open airway in the previous studies, allowing for ventilation produced by chest compressions alone or in conjunction with spontaneous gasping.

At the time of this writing, it is not possible to know whether the use of an impedance threshold valve to simulate inspiratory airway obstruction, although applied to all animals studied, would have influenced the result through an enhancement of venous return and superior hemodynamic stability [78]. These latest studies do give indications, however, that the ratio of 30 : 2 might be suitable for study in a real life cardiac arrest situation.

**Human observations**

In a recent review of the Pittsburgh laboratory experience, Safar [68] has reported that chest compressions alone failed to produce any tidal volumes in humans with an unsupported head or airway and that spontaneous gasping was not able to produce adequate gas exchange in humans unless the head was tilted backward elevating the shoulders. Despite the limitation of this human volunteer model, anesthetized and paralyzed, a strong message was sent to the scientific community that adequate gas exchange and oxygenation could not be achieved without assisted ventilation in human cardiac arrest victims. The data in human cardiac arrest remained conflicting, however. For example, a study of more than 3000 out-of-hospital cardiac arrests in Europe demonstrated that good-quality chest compressions without ventilations done by lay rescuers were comparable with good-quality chest compressions with ventilations. Long-term survival was 15% in the CC-CPR group and 16% in the compression with ventilation group. Both techniques resulted in better long-term outcome than that seen in patients who received no bystander CPR [79].

**Conclusion**

Optimal C-V ratio is still unknown and further human studies are specifically needed to define the best method for coordinating ventilation and chest compressions during CPR, in terms of survival and neurologic outcome in both patients with protected and those with unprotected airways. Interpretation of animal data is affected by the
lack of uniform methods in different laboratories. In particular the role of ‘involuntary’ ventilation in affecting the outcome of the experiments while providing chest compression to an animal with airways splinted by an endotracheal tube is not clear. Likewise, the true incidence and impact of agonal respiration and airway obstruction in human are not known. A 30 : 2 and 100 : 2 C-V ratio have both been promising to enhance coronary or cerebral oxygen delivery and 24-hour neurologic outcome, respectively, but they cannot be compared head to head at this time because the experiments are radically different.

There is a discrepancy between what is recommended by the current guidelines and the ‘real world’ of CPR that has result in a near flat survival rate in the past few years.

Poor skill retention and fatigue significantly affect rescue performance ventilation and chest compression, but it is clear that a radical but effective way to enhance circulation is to completely eliminate ventilation in the first minutes of CPR for BLS, and limit hyperventilation for ACLS. The recent International Consensus on Science for 2005 will have the difficult responsibility of translating all new available evidence into more beneficial guidelines.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as: *• of special interest **of outstanding interest


19. In-hospital cardiac arrest CPR (real setting) by hospital staff in Chicago, showing that the quality of CPR is often inconsistent with the guideline standards and recommendations.


Staged teaching approach (chest compression only first followed by C — V ratio 50 : 5 and then standard CPR) may improve skill retention and quality of chest compressions assessed 12 months after the training session.


Chest compression performance during standard CPR declined during 18 months whereas there was minimal decline in chest compression performance on repeated testing of uninterrupted CC-CPR.


Two-phase research project: an observational study showing that professional rescuers routinely hyperventilate the patient during CPR, and an animal study in which hemodynamic observations during hyperventilation suggest that high respiratory rate is linked to increased intrathoracic pressure, decreased coronary perfusion, and decreased survival.