Ventilation during CPR

Laurent Brochard
Conflicts of interest

• Our clinical research laboratory has received research grants for clinical trials from the following companies
  – Covidien (PAV+)
  – Dräger (SmartCare)
  – General Electric (FRC)
  – Respironics (NIV)
  – Vygon (CPAP)
BUKIT MERTAJAM: A 65-year-old man, believed to be the country's first person with the rare Lazarus phenomenon, died on Tuesday.

On April 14, this man had stopped breathing and doctors pronounced him dead. But, miraculously, he began breathing again after two-and-a-half hours.
The Lazarus phenomenon

Vedamurthy Adhiyaman\textsuperscript{1}  Sonja Adhiyaman\textsuperscript{2}  Radha Sundaram\textsuperscript{3}

\textit{J R Soc Med} 2007;\textbf{100}:552–557

\section*{SUMMARY}

Even though Lazarus phenomenon is rare, it is probably under reported. There is no doubt that Lazarus phenomenon is a reality but so far the scientific explanations have been inadequate. So far the only plausible explanation at least in some cases is \underline{auto-PEEP} and \underline{impaired venous return}. In patients with PEA or asystole, dynamic hyperinflation should be considered as a cause and a short period of apnoea (30–60 seconds) should be tried before stopping resuscitation. Since ROSC occurred within 10 minutes in most cases, patients should be passively monitored for at least 10 minutes after the cessation of CPR before confirming death.

arrests. We collected information of of arrest, duration of CPR, cardiac stopped and time taken for ROSC, (Table 1).

\section*{Diagnoses at the time of car}

Of the 38 cases described, 13 had eight had obstructive airways dis include ruptured abdominal aortic artery rupture, gastrointestinal haem due to renal failure, trauma, digox overdose with opiates and cocaine. 
AMBU Bag Ventilation
A systematic review of autoresuscitation after cardiac arrest:

K. Hornby, MSc; L. Hornby, MSc; S. D. Shemie, MD

Crit Care Med 2010 Vol. 38, No. 5
Death by hyperventilation: A common and life-threatening problem during cardiopulmonary resuscitation

Tom P. Aufderheide, MD; Keith G. Lurie, MD
No ABC anymore?
"ABC" becomes "CAB"

Circulation is to be addressed first with the initiation of chest compressions. Compression:ventilation ratio of 30:2 should be maintained after the first round of 30 chest compressions.

Push Hard, Push Fast
(high-quality CPR is emphasized)

100 compressions/min at a depth of at least 2 inches.
1. It is unknown whether administration of 100% inspired oxygen is beneficial to people who have sustained SCA; however, there is no evidence that it causes harm in short-term resuscitation. Therefore, the use of 100% oxygen is reasonable during initial resuscitative efforts.

2. Chest compressions cause air to be expelled during the compression phase and oxygen to be passively drawn into the chest during the recoil phase. Therefore, the passive inhalation of oxygen via a nonrebreather facemask is likely to be sufficient for several minutes after the onset of SCA in patients who have a patent airway.

3. Bag-mask ventilation can be challenging to perform correctly and is best done by two trained rescuers. If this technique is used, it is recommended that a tidal volume of approximately 600 mL be delivered over 1 second at a rate of 8 to 10 times per minute.
Ventilation during cardiac arrest

- Very low ventilatory needs
- Gas trapping
- Amplification of pressure effects because of hypovolemia
- Difficult airway access
- Benefits of « gasps »
- Interruption of compression
Interrupting chest compressions for rescue breathing can adversely affect hemodynamics during CPR for VF.
The primacy of basics in advanced life support
Douglas Chamberlain\textsuperscript{a}, Michael Frenneaux\textsuperscript{b} and David Fletcher\textsuperscript{a}

Current Opinion in Critical Care 2009, 15:198–202
No assisted ventilation cardiopulmonary resuscitation and 24-hour neurological outcomes in a porcine model of cardiac arrest

Demetris Yannopoulos, MD; Timothy Matsuura, BS; Scott McKnite, BS; Noah Goodman, BS; Ahamed Idris, MD; Wanchun Tang, MD; Tom P. Aufderheide, MD; Keith G. Lurie, MD
Efficacy of Cardiopulmonary Resuscitation Using Intratracheal Insufflation

LAURENT BROCHARD, GEORGES BOUSSIGNAC, SERGE ADNOT, CATHERINE BERTRAND, DANIEL ISABEY, and ALAIN HARF

Constant flow insufflation of oxygen as the sole mode of ventilation during out-of-hospital cardiac arrest
a. Spring  
b. Bellows  
c. Adiabatic compressor  
d. Intrathoracic pressure measurement  
e. Airway opening  
f. Pencil and milimetric paper  
g. Compressible conducting airway connector
Dynamic thoracic volume change

Lung position after LUCAS 2® positioned

Without ventilation

Dynamic thoracic volume change

C: Compression

D: Decompression

Vol mL

Without ventilation

With CFI

CFI Effect
RESULTS WITH CFI 5

- Without ventilation
- Manual Bag
- CFI 5
- CFI 10

Thoracic volume reduction (ml)

Without ventilation: -400
Manual Bag: -300
CFI 5: -200
CFI 10: -100
RESULTS MV WITH CFI 5

- Dead space ventilation (Estimated physiological dead space = 200ml)
- Calculated alveolar ventilation

<table>
<thead>
<tr>
<th>Condition</th>
<th>L/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without ventilation</td>
<td>10</td>
</tr>
<tr>
<td>Manual Bag</td>
<td>20</td>
</tr>
<tr>
<td>CFI 5</td>
<td>30</td>
</tr>
<tr>
<td>CFI 10</td>
<td>35</td>
</tr>
</tbody>
</table>
RESULTS PRESSURE WITH CFI 5

![Graph showing mean positive and negative lung pressures for different ventilation methods: Without ventilation, Manual Bag, CFI 5, and CFI 10. The graph indicates that CFI 10 has the highest mean positive and negative lung pressures compared to the others.](image-url)
Association Between Arterial Hyperoxia Following Resuscitation From Cardiac Arrest and In-Hospital Mortality

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Alan E. Jones, MD
Nathan I. Shapiro, MD, MPH
Mark G. Angelos, MD
Barry Milcarek, PhD
Krystal Hunter, MBA
Joseph E. Parrillo, MD
Stephen Trzeciak, MD, MPH
for the Emergency Medicine Shock Research Network (EMShockNet)

Investigators

Sudden cardiac arrest is the most common lethal consequence of cardiovascular disease. Even if return of spontaneous circulation (ROSC) from cardiac arrest is achieved, approximately 60% of patients will not survive to hospital discharge.1,2 The high mortality is attributed to the postcardiac arrest syndrome, which involves global ischemia-reperfusion injury, myocardial stunning, and anoxic brain injury.3 The recent success of therapeutic hypothermia

Context Laboratory investigations suggest that exposure to hyperoxia after resuscitation from cardiac arrest may worsen anoxic brain injury; however, clinical data are lacking.

Objective To test the hypothesis that postresuscitation hyperoxia is associated with increased mortality.

Design, Setting, and Patients Multicenter cohort study using the Project IMPACT critical care database of intensive care units (ICUs) at 120 US hospitals between 2001 and 2005. Patient inclusion criteria were age older than 17 years, nontraumatic cardiac arrest, cardiopulmonary resuscitation within 24 hours prior to ICU arrival, and arterial blood gas analysis performed within 24 hours following ICU arrival. Patients were divided into 3 groups defined a priori based on PaO\textsubscript{2} on the first arterial blood gas values obtained in the ICU. Hyperoxia was defined as PaO\textsubscript{2} of 300 mm Hg or greater; hypoxia, PaO\textsubscript{2} of less than 60 mm Hg (or ratio of PaO\textsubscript{2} to fraction of inspired oxygen <300); and normoxia, not classified as hyperoxia or hypoxia.

Main Outcome Measure In-hospital mortality.

Results Of 6326 patients, 1156 had hyperoxia (18%), 3999 had hypoxia (63%), and 1171 had normoxia (19%). The hyperoxia group had significantly higher in-hospital mortality (732/1156 [63%; 95% confidence interval (CI), 60%-66%]) compared with the normoxia group (532/1171 [45%; 95% CI, 43%-48%]; proportion difference, 18% [95% CI, 14%-22%]) and the hypoxia group (2297/3999 [57%; 95% CI, 56%-59%]; proportion difference, 6% [95% CI, 3%-9%]). In a model controlling for potential confounders (eg, age, preadmission functional status, comorbid conditions, vital signs, and other physiological indices), hyperoxia exposure had an odds ratio for death of 1.8 (95% CI, 1.5-2.2).

Conclusion Among patients admitted to the ICU following resuscitation from cardiac arrest, arterial hyperoxia was independently associated with increased in-hospital mortality compared with either hypoxia or normoxia.

JAMA. 2010;303(21):2165-2171

www.jama.com
A Kaplan-Meier survival curve comparing normoxia and hyperoxia groups. The curve shows a significant decrease in survival proportion over time, with a Log-rank P < 0.001. The number of patients at risk decreases over time: for normoxia, from 1171 to 83, and for hyperoxia, from 1156 to 70.

<table>
<thead>
<tr>
<th>Variable</th>
<th>OR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age decile</td>
<td>1.1 (1.1-1.2)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Emergency department origin</td>
<td>1.5 (1.3-1.7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Nonindependent functional status at admission</td>
<td>1.3 (1.1-1.4)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>1.6 (1.3-1.9)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Active chemotherapy</td>
<td>2.8 (1.8-4.6)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>High heart rate in ICU^b</td>
<td>1.9 (1.7-2.1)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hypotension at ICU arrival^c</td>
<td>2.1 (1.9-2.3)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hypoxia exposure</td>
<td>1.3 (1.1-1.5)</td>
<td>.009</td>
</tr>
<tr>
<td>Hyperoxia exposure</td>
<td>1.8 (1.5-2.2)</td>
<td>&lt;.001</td>
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Too much of a good thing?
Thank you
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