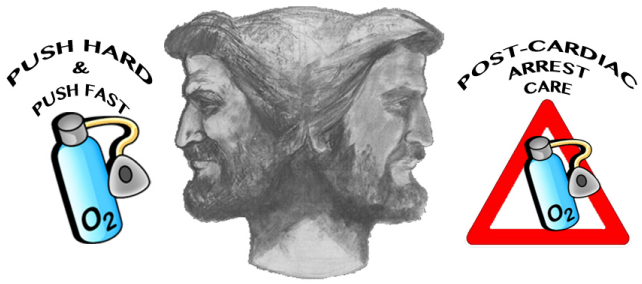
Oxygen therapy: the Janus of cardiopulmonary resuscitation



Anyway Oxygen A 'gogo!

-Sorry?

The oxygen is always good!

-What?

Resolves hypoxia, and in worst

case does no damage!

-Are you kidding? And the damages from hyperoxia?

The conversation, which was rebuilt for fiction, reflects some confusion surrounding the use of O2 therapy in the emergency medicine in both intra- and pre-hospital.
Taking a cue from a recent paper on the use of O2 during cardiopulmonary resuscitation, and since there is data in the literature strong enough, we would get a stake on the use of oxygen, even during cardiopulmonary resuscitation.

The work we mentioned is "Increasing arterial oxygen partial pressure during cardiopulmonary resuscitation is associated with improved rates of hospital admission".

Walter Spindelboeck and the other authors have investigated (retrospectively on a database of Irish OHCA) the impact of O2 administration during CPR in patients victim of non-traumatic cardiac arrest.
During the early stages of ALS was performed an arterial blood sample and the patients have been assigned, according to the PaO2 detected, in three different groups: high, medium, low PaO2.

The primary outcome was hospital admission (and the presence of spontaneous circulation for at least 1 hour); ROSC percentage and survival rate at 28 days were also recorded.
The results, go in the opposite direction compared to what the authors themselves awaited, and show that patients in the group "high O2" have better outcomes than those in the middle and lower group.
Is so confirmed (as recommended by ALS 2010 Guidelines), but with more recent data, that the administration of high doses O2 during CPR improves

survival.
At the resumption of spontaneous circulation on the contrary, all references *(2,3,4),* indicate that the persistent hyperoxia triggers mechanisms potentially detrimental on cellular metabolism, and is therefore harmful, so after ROSC oxygen should be administered by aiming the target (SaO2, EtCO2) and no longer high flow and high FiO2.

Cardiopulmonary resuscitation is configured as a sort of two-faced Janus; the turning point is the restoration of a spontaneous circulation.
During CPR (low pressure period) inside cells and tissues, the O2 tension reaches acceptable levels only with adequate ventilation and maximal oxygenation (100% FiO2 and high flows), while the recovery of the circle by improving DO2 increases in tissues (especially the brain), the risk of hyperoxia with all the damage that we very well know.

ALS patient management and post-cardiac arrest care acquire a qualifyng piece for the "resuscitator".
The attention to details, customization of the simple protocol on the actual patient needings, makes the difference between a good and a "less good" cardiac arrest management.

References:

1. *Increasing arterial oxygen partial pressure during cardiopulmonary resuscitation is associated with improved rates of hospital admission*
2. *Association between arterial hyperoxia following resuscitation from cardiac arrest and in-hospital mortality.*
3. *Relationship Between Supranormal Oxygen Tension and Outcome After Resuscitation From Cardiac Arrest*.
4. *Arterial hyperoxia and in-hospital mortality after resuscitation from cardiac arrest*