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Title : STERNAL COMPRESSIONS BEFORE VENTILATION IN CARDIOPULMONARY RESUSCITATION (CPR)

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Introduction. The CPR Committee of the Dutch Heart Association (Kling, J, personal communication) currently recommends for the management of cardiac arrest, the start of external cardiac compression (ECC) before airway control and ventilation ("CAB sequence"), rather than ventilating the patient first, as is recommended by the American Heart Association ("ABC sequence"). Supporters of CAB reason that, since most sudden cardiac deaths are due to ventricular fibrillation oxygenated blood remains in the arterial system. Since arterial blood may comprise 20% of total blood volume, this could be circulated before the more difficult and time-consuming airway control and ventilation measures are initiated. We conducted the following study to characterize the decline in arterial PO_2 and pH during 5 min of cardiac arrest without CPR, and subsequently during ECC without ventilation for 1 min.

Methods. 13 dogs were anesthetized with halothane and N_2O/O_2 66%/33%, immobilized with pancuronium and ventilated by IPPV (TV 15 ml/kg, f varied to maintain $PETCO_2$ at 30-40 torr). The thoracic aorta was catheterized via the femoral artery for withdrawal of arterial blood. 5 min prior to cardiac arrest, ventilation with room air was begun. Immediately prior to ventricular fibrillation (VF) a blood gas sample was drawn. When the ventilator was disconnected and the tracheal tube clamped at the end of expiration, VF was induced electrically. Arrest was allowed to persist for 5 mins, after which a second blood gas sample was drawn. At this point ECC was started manually (1½-2 inch strokes, 80 compressions per minute) without ventilation, with the tracheal tube remaining clamped (simulating clinical airway obstruction). Blood gas samples were drawn after 30 and 60 sec of ECC, then 4 rapid ventilations with air were administered, and ECC continued with 2 IPPV with air after every 15 compressions. After 60 sec of CPR with ventilation, a final blood gas was drawn.

Results. Control blood gas values were within normal limits, pHa 7.34±0.04 (mean± SD); PaO_2 74.5±18.0 torr and $PaCO_2$ 36.5±6.2 torr. Calculated bicarbonate and base excess were 18.8±1.5 and -5.8±1.3 mEq/L respectively. 5 min of arrest without CPR led to significant hypoxemia and acidemia: PaO_2 at that point was 59.1±8.9 torr ($p<0.001$, using the two-tailed t test for paired samples). pHa was 7.29±0.04 ($p<0.001$). 30 sec of ECC without ventilation failed to produce a significant change in PaO_2 (56.4±10.1 torr) ($p>0.05$), however, pHa decreased to 7.26±0.05 ($p<0.01$). There was a further decrease in PaO_2 and pHa at 60 sec of ECC without ventilation, to 7.24±0.06 ($p<0.001$) and PaO_2 53.3±12.5 torr ($p<0.05$). The addition of ventilation to ECC for 60 sec produced significant improvement of all values seen after 60 sec of ECC alone. pHa was elevated to 7.31±0.07 ($p<0.001$), and PaO_2 to 68.7±15.5 torr ($p<0.001$). After 5 min of cardiac arrest, $PaCO_2$ was 42.0±6.0 torr ($p<0.001$). It was exacerbated by ECC without ventilation for 30 sec, raising $PaCO_2$ to 46.4±5.1 ($p<0.05$) and further at 60 sec of ECC without ventilation, raising $PaCO_2$ to 49.8±7.1 torr ($p<0.05$). One min of ECC with ventilation reversed this trend ($PaCO_2$ 35.7± 5.7) ($p<0.001$).

Discussion. Although the "asphyxiation" of arterial blood during cardiac arrest without CPR and its further deterioration during ECC without ventilation were mild in this study, the contention that PaO_2 does not decline rapidly when the blood is stagnant in the arterial system, and can be safely pumped without ventilation, is refuted by these data. Even 5 min of stasis produced some deterioration of blood gas values. We also have demonstrated that ECC without ventilation further worsens acidemia, hypoxemia and hypercarbia and that this progressive decline can be rapidly reversed by adding ventilation. In man, ECC alone produces circulation[1], but not ventilation[2], and therefore must be accompanied by airway control and ventilation in some form. While the CAB sequence may pump oxygenated blood without ventilation for the first 30-60 sec in cases of sudden witnessed cardiac arrest, particularly when ECC is started immediately, "good" arterial blood cannot be assumed after unwitnessed arrest and after asphyxia.

Conclusions. Teaching of the CAB sequence seems unwise. Asphyxiated arterial blood may be circulated. Teaching CAB also makes training aids obsolete, without proven clinical value. It would deemphasize universal learning of airway control and ventilation, important also in coma without cardiac arrest. Currently taught ABC causes only a 30 sec delay in ECC.

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References.

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