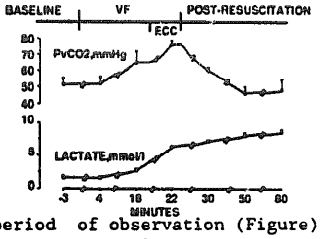
Tuesday, March 5, 1991
4:00PM–5:30PM, Room 254, West Concourse
Cardiopulmonary Resuscitation

4:00

MIXED VENOUS CO_2 VS. LACTATE AS AN INDICATOR OF PERFUSION FAILURE

Bruce Johnson, Max Harry Weil, Frank Maldonado, Wanchun Tang, Jose Bisera; University of Health Sciences/The Chicago Medical School, North Chicago, IL

Previous studies demonstrated that both blood lactate and venous PCO_2 serve as reliable indicators of perfusion failure during circulatory shock and cardiac arrest. In the present study, we compared changes in the PCO_2 of mixed venous blood (P_vCO_2) with that of arterial and mixed venous blood lactic acid following successful cardiac resuscitation. In five domestic pigs weighing between 25 and 35 kg, ventricular fibrillation (VF) was induced and untreated for 20 min. Each of the animals was then successfully resuscitated with extracorporeal circulation (ECC) followed by DC countershock. Progressive increases in mixed venous blood PCO_2 and lactate were observed for approximately 22 min after the onset of VF. Within 7 minutes after successful resuscitation, however, mixed venous blood PCO_2 returned to baseline values whereas lactic acid concentrations continued to increase during the subsequent 60 min post resuscitation period of observation (Figure). This study therefore demonstrates that mixed venous blood PCO_2 has a much better frequency response as an indicator of perfusion deficit with a half-time (t_{50}) of 7 minutes. This contrasts with lactic acid with a t_{50} of greater than 90 minutes.

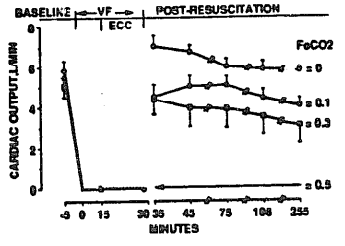


4:15

POST-RESUSCITATION MYOCARDIAL DYSFUNCTION FOLLOWING INDUCED HYPERCARBIC ACIDOSIS

Frank A. Maldonado, Max Harry Weil, Wanchun Tang, Bruce Johnson; University of Health Sciences/The Chicago Medical School, North Chicago, IL

In the present study, we investigated whether the hypercarbia which accompanies cardiac arrest compromises post-resuscitation cardiac function. Ventricular fibrillation (VF) was electrically induced in 12 pentobarbital anesthetized domestic pigs. After 15 min of VF, veno-arterial extracorporeal circulation (ECC) was initiated such as to increase coronary perfusion pressure to 70 mm Hg with oxygenator FO_2 of 0.5. Animals were then randomized to receive oxygenator CO_2 ($FoCO_2$) of 0, .1, .3 or .5. Each of the animals were successfully resuscitated by DC countershock after an interval of 3 to 15 min of ECC, except that no viable rhythm could be reestablished in animals with $FoCO_2$ of 0.5. We further observed a striking reduction in cardiac output following successful resuscitation which was related to the severity of myocardial hypercarbia during cardiac arrest (Figure). These observations lend additional support to prior observations that high levels of myocardial CO2 during cardiac arrest are detrimental. In this instance, they compromise myocardial function and survival after successful resuscitation.



4:30

EFFECT OF INTERPOSED ABDOMINAL COUNTERPULSATION ON RESUSCITATION OUTCOME DURING ASYSTOLE AND ELECTROMECHANICAL DISSOCIATION

Jeffrey B. Sack, M.D., Michael B. Kesselbrenner, M.D., F.A.C.C., Anwar A. Jarrad, M.D., David Bregman, M.D., F.A.C.C. Seton Hall University, School of Graduate Medical Education, Paterson, N.J.

Interposed abdominal counterpulsation during CPR (IAC-CPR) has been shown to increase coronary blood flow and diastolic perfusion pressures. Improving diastolic coronary perfusion pressure has been thought to be a requirement for successful resuscitation during periods of asystole and electromechanical dissociation (EMD). We hypothesized that IAC-CPR during cardiac arrest would improve resuscitation outcome when asystole or EMD was the mechanism of cardiac arrest. We prospectively studied 86 in-hospital cardiac arrests. Patients were randomized to receive either IAC-CPR or standard-CPR (STD-CPR). Our endpoint was return of spontaneous circulation (ROSC), as defined by the presence of a palpable pulse plus systolic BP > 80 mmHg for greater than 3 minutes. There were 38 males and 48 females with a mean age of 66.8 yrs. The mechanism of arrest was asystole in 60 patients and EMD in 26 patients. The overall resuscitation rate was 30.2%. Of the patients who underwent IAC-CPR, 18/42 (42.9%) had ROSC, while in the STD-CPR group, only 8/44 patients (18.2%) had ROSC (p = 0.013). In the IAC-CPR group, 9/27 patients (33.3%) had ROSC during asystole, while only 6/33 patients (18.2%) in the STD-CPR group (p = NS) experienced ROSC. When EMD was the initial mechanism of arrest, 9/15 patients (60.0%) in the IAC-CPR group and only 2/11 patients (18.2%) from the STD-CPR group had ROSC (p = 0.033).

We conclude that IAC-CPR, with its augmentation of diastolic perfusion pressure, is an easily applied manual technique and capable of generating spontaneous circulation in a significantly larger percentage of patients than STD-CPR during asystole and EMD.

4:45

DETERMINANTS OF VASCULAR PRESSURE GENERATION DURING CARDIOPULMONARY RESUSCITATION: THE EFFECT OF RISE TIME

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It has been proposed that performing chest compressions that quickly reach peak force (short rise time (RT)) can enhance blood pressure(P) and flow(F) generation during cardiopulmonary resuscitation (CPR), presumably by increased direct compression of the heart. An alternative hypothesis, however, is that the shorter RT improves P and F by promoting airway collapse and air trapping, thus increasing intrethoracic pressure (ITP). In order to study the effect RT on P and F generation and air trapping, we performed CPR in 7 dogs at the AHA recommended rate of 90 compressions/minute with short(<100ms) and long(>100ms) RT.

Short RT significantly increased peak aortic(Ao) pressure (98±24 vs 88±14mmHg, p<0.001), peak right atrial(RA) pressures (101±29 vs 77±29, p<0.001) and peak carotid flow(CF) (338±192 vs 211±134ml/sec, p<0.001) as compared to long RT. Conversely, the volume of air expired during compression was significantly less at the short RT (16±16 vs 34±31ml, p < 0.05), indicating trapping of air in the lungs at the shorter RT. In contrast to the peak P and F, the mean systolic Ao pressure (61±10 vs 62±15), total CF (37±12 vs 38±12ml/min), and mean diastolic Ao-RA gradient (21±17 vs 26±17) were not significantly different for a short or long RT.

Thus, significantly increased peak P and F are generated with a short RT, which may be mediated via increased air trapping with short RT and not direct compression. Despite increased peak pressures, there is no improvement in the mean pressure which is the major determinant of vital organ perfusion.