CARBON-DIOXIDE ELIMINATION DURING CLOSED CHEST CARDIAC MASSAGE

Case Report

BY

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SUMMARY

Acid-base balance was accurately measured before, during and after external cardiac massage in a case of ventricular fibrillation developing shortly after induction of anaesthesia for herniorrhaphy. Results showed that the acidosis which occurred during cardiac arrest was purely metabolic in origin, and that carbon dioxide elimination by the lungs during intermittent positive pressure respiration in a patient undergoing external cardiac massage was sufficient to lower the Pco₂ below normal. The management of the case is described.

Cardiac arrest causes metabolic acidosis as a direct result of tissue anoxia due to reduced tissue perfusion. The reduction of peripheral blood flow occurs simultaneously with a reduced pulmonary blood flow. It is possible that carbon-dioxide retention might occur during cardiac arrest despite intermittent positive pressure respiration, due to extreme ventilation/perfusion ratio upset and that respiratory acidosis could further reduce the pH. Moreover, closed-chest cardiac massage might itself have a deleterious effect on pulmonary blood flow due to direct pressure on the pulmonary vessels.

We had the rare opportunity of measuring acid-base balance accurately immediately before, during and after cardiac arrest in the following case.

CASE REPORT

A 72-year-old man was hospitalized for an inguinal hernia repair. His general condition was fair. He suffered from arterial hypertension (170/105 mm Hg) and a pre-operative electrocardiogram showed right bundle branch block and a few extrasystoles. Physical examination and X-ray of the chest indicated mild emphysema of the lung.

Premedication consisted of pethidine 75 mg and atropine 0.5 mg given intramuscularly 1 hour before operation. Anaesthesia was induced using thiopentone 250 mg; this was followed by suxamethonium 50 mg, after which the lungs were inflated and the trachea intubated with difficulty. The circuit employed was a circle system with carbon dioxide absorption and the fresh gas flow was 10 l/min.

* The acid-base measurements were made using the Radiometer Astrup Micro-Equipment A.M.E. Ic on arterialized capillary blood.

Immediately after intubation the pulse was found to be absent and no heart sounds were heard. Cardiac arrest was presumed and the patient was immediately placed in the steep Trendelenburg position and external cardiac massage was begun. IPPR was continued with oxygen 100 per cent.

**FIG. 1**

Electrocardiographic tracing obtained after cardiac arrest had been diagnosed.

An electrocardiogram showed ventricular fibrillation (fig. 1) and repeated unsuccessful attempts to defibrillate were made using an external DC defibrillator. Twenty minutes after the onset of cardiac arrest the pH and Pco₂ were measured (table I). These indicated metabolic acidosis and 150 m.eqiv of sodium bicarbonate was rapidly injected intravenously. Despite vigorous external cardiac massage, pulses in the large peripheral vessels were not palpable and a solution of angiotensin amide (Hypertensin; Ciba) 1 mg/1000 ml was slowly infused intravenously. After a few minutes it was possible to feel a carotid pulse concomitant with the cardiac massage. Thirty-seven minutes after cardiac arrest was first diagnosed, defibrillation was accomplished using the maximal current of 400 watt sec. External cardiac massage had to be continued for a further 13 minutes, the peripheral circulation being inadequate as judged by peripheral pulses and blood pressure. The patient was rapidly digitalized using digoxin 1 mg; this was injected slowly intravenously.

Because of prolonged decreased renal perfusion and the threat of acute renal failure 25 g of mannitol (25 per cent) was infused; we hoped that this would also decrease cerebral oedema which might develop due to prolonged cerebral anoxia.
Fifty minutes after the onset of cardiac arrest, cardiac massage was discontinued. After a further 35 minutes respiration became adequate, 80 minutes having elapsed since induction of anaesthesia. The patient regained consciousness 2 hours and 10 minutes after cardiac arrest and was extubated. At first he was irrational and difficult to control but his mental condition improved rapidly and he was completely normal 24 hours after the event. He was discharged from hospital 10 days later.

<table>
<thead>
<tr>
<th></th>
<th>Before arrest</th>
<th>During arrest</th>
<th>After arrest</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.325</td>
<td>7.320</td>
<td>7.320</td>
</tr>
<tr>
<td>Pco₂ (mm Hg)</td>
<td>42</td>
<td>33</td>
<td>43</td>
</tr>
<tr>
<td>Standard bicarbonate (m.equiv/L)</td>
<td>20.5</td>
<td>18</td>
<td>20.5</td>
</tr>
<tr>
<td>Actual bicarbonate (m.equiv/L)</td>
<td>21</td>
<td>16.5</td>
<td>21.5</td>
</tr>
<tr>
<td>Base excess (m.equiv/L)</td>
<td>-4</td>
<td>-8</td>
<td>-4.5</td>
</tr>
</tbody>
</table>

The pre-operative sample was obtained 10 minutes before induction of anaesthesia. The second sample was obtained about 20 minutes after the onset of ventricular fibrillation, during hyperventilation with oxygen, and whilst external cardiac massage was being performed. The third sample was obtained about 150 minutes after the onset of cardiac arrest, that is about 15 minutes after extubation and after the resumption of spontaneous breathing.

**COMMENT**

As is seen from the results (Table I), the acidosis that developed during the period of cardiac arrest and external cardiac massage was purely metabolic and was characterized by a fall in standard bicarbonate and actual bicarbonate. Both perfusion and ventilation of the lungs were sufficient to eliminate carbon dioxide as is shown by the decreased Pco₂. Unfortunately, oxygen tension of the arterial blood was not measured due to the technical difficulties involved in arterial puncture during external cardiac massage.

**ACKNOWLEDGEMENT**

We are grateful to Dr. R. R. Peer under whose service the patient was hospitalized for permission to publish this case report.

**ELIMINATION DE L'ANHYDRIDE CARBONIQUE DURANT LE MASSAGE CARDIAQUE A THORAX FERME**

**SUMMAIRE**

La balance acide-base a été mesurée avec exactitude avant, pendant et après le massage cardiaque externe dans un cas de fibrillation ventriculaire, se développant peu de temps après l'induction d'anesthésie pour une hémiorrhaphie. Les résultats ont montré que l'acidose, se manifestant durant l'arrêt du cœur, était d'origine purement métabolique, et que l'élimination de l'anhydride carbonique par les poumons au cours d'une respiration à pression positive intermittente chez un patient subissant un massage cardiaque externe, était suffisante pour réduire le Pco₂, dessexus de la normale. Le traitement du cas est décrit.

**KOHLENDIOXYD-ABGABE WÄHRENDE DER HERZMASSAGE BEI GESCHLOSSEMEN BRUSTKORB: BERICH'T ÜBER EINEN EINZELFALL**

**ZUSAMMENFASSUNG**

In einem Fall von Ventrikularfibrillation, die kurz nach der Anästhesie-einleitung in einem Hämorrhagiefall auftrat, wurden genaue Messungen der Basen-Säuren-Bilanz vor, nach und während einer äußerer Herzmassage durchgeführt. Es ergab sich hierbei, daß die während des Herzstillstandes vorhandene Azidose ausschließlich stoffwechselbedingt war; ferner zeigte sich, daß die Kohlendioxydabgabe der Lungen während der intermittierenden Positivdruckatmung in einem unter äußerer Herzmassage befindlichen Patienten zur Erniedrigung des Pco₂—Betrages unter den Normalwert hinreichte. Die zur Beherrschung des Zwischenfalles vorgenommene Behandlung sowie die dabei durchgeführten Untersuchungen werden geschildert.

**BOOK REVIEW continued from page 58**

Good defence could be made in support of the many pages devoted to descriptive anatomy and detailed accounts of the techniques of regional block (the latter will undoubtedly provide a superb source of reference for many years).

The discussions of natural childbirth and psycho-phylaxis, although somewhat prolix, provide an admirable review of a contemporary debate. Bonica leans over backwards to be fair to the proponents of these techniques, but some trenchant criticism is levelled, with most of which the reviewer agrees.

There are errors of fact. For example: in the U.K. and also in the U.S.A., a stillborn is not defined as "a foetus with an observable heart beat, but who makes no attempt to breathe" (p. 217); "analgesic concentration of even the potent (inhalation) agents produces no significant depression in the newborn, regardless of the duration of administration" (p. 203)—this is a serious mis-statement of fact, and is indeed contradicted on several occasions in the ensuing text; the figures quoted for maximum inspiratory and expiratory airflows in the pregnant woman (Tables 1–4) are less than half of the actual values. However, this volume is not to be faulted for failing to present, and rationally to interpret and apply, the vast amount of data referable to its theme; rather, by being excessively "too much of a good thing", it has assumed a dinosaur quality, destined, one fears, to become a fossil monument to the all-encompassing knowledge of its author instead of an evolving symbol of his dynamic interest.

In this country, the price will be prohibitive for most individual doctors and for all but the best-endowed libraries.

J. Selwyn Crawford