

Measurement of end-tidal carbon dioxide concentration during cardiopulmonary resuscitation

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SUMMARY

End-tidal carbon dioxide concentrations were measured prospectively in 12 cardiac arrest patients undergoing cardiopulmonary resuscitation (CPR) in an accident and emergency department.

The end-tidal carbon dioxide (CO₂) concentration decreased from a mean (\pm SD) of $4.55 \pm 0.88\%$ 1 min after chest compression and ventilation was established, to values ranging from $2.29 \pm 0.84\%$ at 2 min to $1.56 \pm 0.66\%$ following 8 min of CPR. Spontaneous circulation was restored in five patients. This was accompanied by a rapid rise in end-tidal CO₂ which peaked at 2 min ($3.7 \pm 1.08\%$). Changes in end-tidal CO₂ values were often the first indication of return of spontaneous cardiac output.

There was a significant difference in the end-tidal CO₂ in patients undergoing CPR before return of spontaneous circulation ($2.63 \pm 0.32\%$) and patients who failed to develop spontaneous output ($1.64 \pm 0.89\%$) ($p < 0.001$).

We conclude that measurement of end-tidal CO₂ concentration provides a simple and non-invasive method of measuring blood flow during CPR and can indicate return of spontaneous circulation.

INTRODUCTION

Difficulties in monitoring the effectiveness of efforts at resuscitation have recently been highlighted (Skinner, 1989). Palpation of the carotid or femoral pulse is the usual method of monitoring circulation but may not represent forward flow. Most studies investigating blood flow during external cardiac massage have used invasive techniques in animal preparations. The relevance of animal studies to resuscitation

in humans has been questioned, and invasive monitoring is usually impracticable in the emergency situation (Chamberlain *et al.*, 1987). A non-invasive monitor which reflects blood flow during precordial compression would not only provide a useful clinical tool, but facilitate clinical trials in humans to evaluate the effect of changes in CPR protocols.

End-tidal carbon dioxide concentration has been shown to correlate with cardiac output and coronary blood flow in experimental animals during cardiopulmonary resuscitation (Sanders *et al.*, 1985; Weil *et al.*, 1985). Subsequently, in critically ill patients, measurement of end-tidal CO₂ provided a practical and non-invasive method for monitoring blood flow generated by precordial compression (Falk *et al.*, 1988). Clearly, caution is required before extrapolating from studies carried out in an intensive care environment to the more common situation of unexpected cardiac arrest.

The aim of this study was to assess the clinical applicability of end-tidal carbon dioxide measurements in patients undergoing cardiopulmonary resuscitation in an accident and emergency department.

SUBJECTS AND METHODS

Adults with non-traumatic, normothermic cardiac arrest, admitted to the Accident and Emergency Department of the Edinburgh Royal Infirmary were studied. All patients had sustained cardiorespiratory arrest in the pre-hospital setting and had received only basic life support prior to arrival. On admission, patients were treated according to the guidelines of the UK Resuscitation Council (1984). Electrical DC counter shock and anti-arrhythmic agents were used as appropriate. All patients were intubated and ventilated with 100% oxygen and mechanical chest compression was administered using a Michigan Instruments Thumper, Model 1004, functioning at a ratio of 5 chest compressions to one ventilation (Little *et al.*, 1974). CPR was performed with sufficient force to compress the chest 2.5–3 inches (80–100 lbs) and peak inspiratory pressure was set at 30 cms of H₂O. During CPR, four patients were given 8.4% sodium bicarbonate at a mean dose of 100 mEq between time 3 and 16 min (mean 8.4 min).

The concentration of expired carbon dioxide was measured continuously with an infra-red absorption carbon dioxide analyser (Model 5200, Ohmeda, Louisville, USA) calibrated to a 5% carbon dioxide standard producing an accuracy of ± 1.5 mmHg (from 0–75 mmHg). The CO₂ values were automatically compensated for pressure and temperature variations. The sampling adaptor was sited between the endotracheal tube and the ventilatory circuit tubing.

All end-tidal CO₂ values are reported as the percentage of carbon dioxide in exhaled gas (mean \pm SD). Data were analysed using a two tailed t-test for paired and unpaired data and differences considered significant when $P < 0.05$.

RESULTS

The study population comprised 8 males and 4 females with a mean age of 69 ± 17 years. The presenting dysrhythmia was asystole in five patients, ventricular fibrillation in five patients and two patients had electro-mechanical dissociation. Five patients developed return of spontaneous circulation, four of whom were discharged from the Accident and Emergency Department (Table 1). The end-tidal CO₂ concentration after 1 min of CPR was $4.55 \pm 0.88\%$, but declined to $2.29 \pm 0.84\%$ at 2 min, $2.25 \pm 1.03\%$ at 4 min and $1.66 \pm 0.56\%$ at 8 min or more.

Coincident with return of spontaneous circulation in five patients, the end-tidal CO₂ significantly increased from $1.8 \pm 0.92\%$ to $3.38 \pm 1.06\%$ within one min ($P < 0.05$). After peaking at 2 min ($3.7 \pm 1.08\%$), the end-tidal CO₂ declined to $3.65 \pm 1.43\%$ at 4 min and $3.02 \pm 0.52\%$ at 8 min in these patients.

In patients given sodium bicarbonate, the end-tidal CO₂ concentration increased from $1.5 \pm 0.49\%$ to $1.8 \pm 0.54\%$ but this rise does not reach statistical significance. The difference between the end-tidal CO₂ concentration in patients without return of spontaneous circulation ($N = 7$, $1.64 \pm 0.89\%$) and the end-tidal concentration in patients prior to regaining spontaneous output ($N = 5$, $2.63 \pm 0.32\%$) was significant ($P < 0.001$). Examples of end-tidal CO₂ recordings in three cases are illustrated in Figs. 1 and 2.

DISCUSSION

End-tidal carbon dioxide concentration is dependent on carbon dioxide production, alveolar ventilation and pulmonary blood flow (Powles & Campbell, 1978). If alveolar ventilation is held constant and carbon dioxide production is assumed to

Table 1. Clinical data in 12 patients. VF denotes ventricular fibrillation and EMD electromechanical dissociation. Four patients received 8.4% sodium bicarbonate during CPR.

Case	Age	Sex	Primary rhythm	Duration of CPR (Minutes)	Na HCO ₃ Administered (mEq)	Discharged from A&E
MB	69	F	VF	20	—	No
WF	68	M	Asystole	11	50	No
CD	75	F	Asystole	10	—	No
WS	68	M	Asystole	5	—	No
JA	73	M	VF	14	—	No
HL	70	F	EMD	31	200	No
UM	69	M	Asystole	6	—	No
PC	65	M	Asystole	5	—	Yes
JA	78	M	VF	9	50	Yes
HF	50	F	VF	3	—	Yes
JL	68	M	VF	12	100	No
SA	19	M	EMD	8	—	Yes

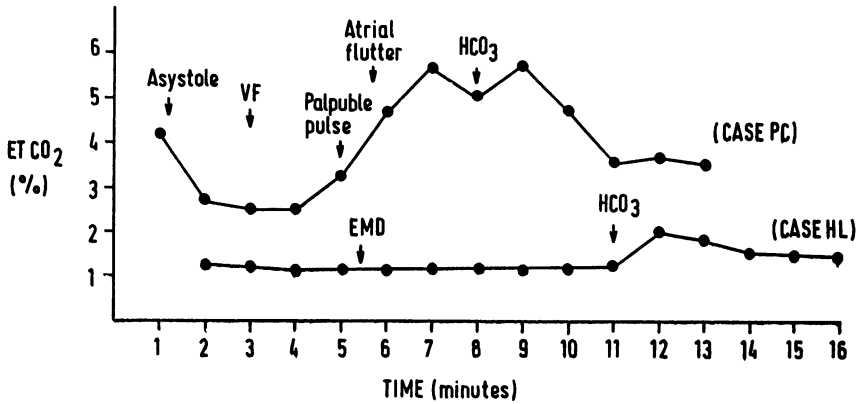


Fig. 1. End-tidal CO₂ concentration during resuscitation. Case PC and HL.

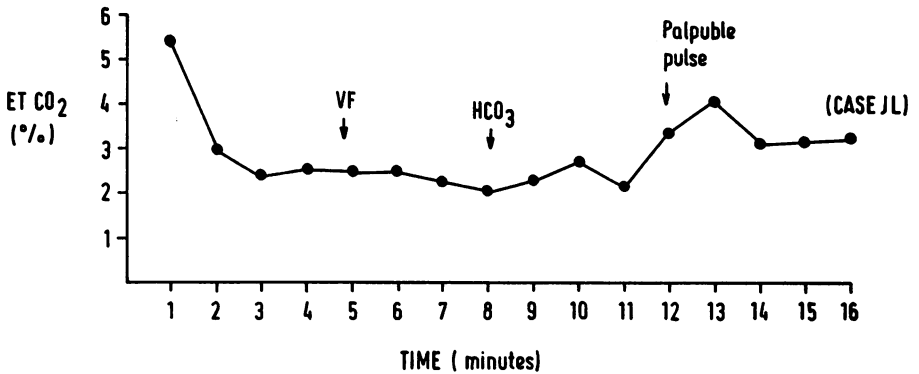


Fig. 2. End-tidal CO₂ concentration during resuscitation. Case JL.

be constant, end-tidal CO₂ values reflect changes in systemic and pulmonary perfusion. The return of spontaneous circulation in this study was associated with a two-fold increase in the end-tidal CO₂ despite constant mechanical ventilation. The low end-tidal CO₂ values demonstrated during precordial compression, reflect the poor forward blood flow achieved with the closed technique. If delivery of CO₂ to the lungs falls as a result of diminished pulmonary perfusion, CO₂ excretion necessarily decreases, even if ventilation is adequate. Closed chest CPR results in reduction of cardiac output to about 25–30% of pre-arrest values (Del Guercio *et al.*, 1965) and therefore the low pulmonary blood flow becomes the rate limiting factor in CO₂ elimination. If spontaneous circulation returns, or if the pulmonary blood flow increases during CPR, CO₂ delivery to the lungs increases, ventilation perfusion mismatch decreases and the end-tidal CO₂ will rise.

Significant arterio-venous CO₂ differences have been reported during cardiopulmonary arrest and resuscitation with a predominant venous respiratory acidaemia and a paradoxical arterial alkalaemia (Weil *et al.*, 1986). The decreased pulmonary blood flow results in the accumulation of CO₂ in the venous circuit, even with adequate ventilation. Carbon dioxide delivery to the lungs is readily eliminated due to the high ventilation/perfusion ratio, resulting in arterial alkalaemia. In this study, the end-tidal CO₂ values were high at the commencement of CPR and in the first few minutes after return of spontaneous circulation, as a consequence of the rapid elimination of accumulated CO₂ in the venous circuit. There was a significant difference between the end-tidal CO₂ concentration in patients undergoing CPR before return of spontaneous circulation and patients who failed to develop spontaneous output. These results support previous observations that end-tidal CO₂ values in resuscitated animals were significantly higher than in animals in which resuscitative efforts were unsuccessful suggesting that measurement of end-tidal CO₂ has predictive value (Sanders *et al.*, 1985; Grundler *et al.*, 1986).

The concept that end-tidal CO₂ reflects pulmonary blood flow has certain limitations. End-tidal CO₂ values can be affected by carbon dioxide production, ventilation and pulmonary perfusion. If manual ventilation is used instead of a mechanical device, care must be taken to provide a constant ventilation rate and tidal volume. The assumption that CO₂ production is constant during resuscitation may be fallacious. However, it is unlikely that CO₂ production decreases immediately after cardiac arrest (Gutipati *et al.*, 1986). End-tidal CO₂ changes related to variation in carbon dioxide production appear insignificant compared with the perfusion changes. In the present study, following a 'wash-out' effect with high end-tidal CO₂ values shortly after patients were established on the Thumper, end-tidal CO₂ concentrations decreased slightly during CPR. Upon return of spontaneous circulation, values increased several fold. In addition, although sodium bicarbonate administration has been shown to result in acute increases in end-tidal CO₂ concentration, the rise is less than that seen with return of spontaneous circulation and of much shorter duration (Falk *et al.*, 1988; Garnett *et al.*, 1987).

During cardiopulmonary resuscitation, an abrupt increase in end-tidal CO₂ values, under conditions of constant ventilation and in the absence of recent sodium bicarbonate administration, provides the earliest evidence of return of spontaneous circulation. Therefore, chest compression need not be interrupted in order to establish whether spontaneous circulation has been restored.

We conclude that end-tidal CO₂ measurement can provide a useful non-invasive monitor of blood flow produced by precordial compression during cardiac arrest.

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