Conjunctival oxygen monitoring during cardiopulmonary resuscitation

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SUMMARY

The conjunctival oxygen tension (C_jO_2) sensor is a non-invasive, continuous index of oxygen delivery in the haemodynamically unstable patient. Human and animal studies have indicated that C_jO_2 reflects cerebral blood flow and oxygenation. Simple insertion, rapid stabilization and reaction time < 60 s allow use in the initial stages of cardiopulmonary resuscitation (CPR) where invasive monitoring is often impracticable.

C_jO_2 was monitored to assess cerebral oxygenation during CPR of 15 patients in cardiac arrest in the accident and emergency department (A&E). Patients who arrested out of hospital with delay to advanced cardiac life support and died had $C_jO_2 < 20$ mmHg (normal C_jO_2 50–60 mmHg) on arrival in A&E. CPR with closed chest cardiac massage (closed CPR) produced no improvement in C_jO_2 . Patients who arrested in ventricular fibrillation (VF) in A&E, and survived with no neurological deficit had $C_jO_2 > 20$ mmHg during CPR. However, further episodes of VF produced an immediate fall in C_jO_2 which continued, despite closed CPR, until restoration of spontaneous cardiac output (RSCO) determined by a palpable carotid pulse. In survivors with delay from arrest to CPR the rise in C_jO_2 with RSCO did not occur for up to 10 min.

This study suggests that closed CPR has no value in maintaining or improving cerebral oxygenation during cardiac arrest. Further studies are required to determine the precise relationship of C_jO_2 to cerebral blood flow and oxygenation during CPR using open and closed techniques of cardiac massage. Open chest cardiac massage (open CPR) has been shown to produce near normal cerebral perfusion and if patients are to survive prolonged resuscitation neurologically intact guidelines for open CPR must be reviewed.

INTRODUCTION

Neurological outcome in survivors of cardiac arrest is determined by the duration of cardiac arrest, oxygen delivery to the brain during CPR and post resuscitation factors.

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Improved techniques in CPR have failed to produce a consistently successful method of producing normal neurological outcome after cardiac arrest of more than 5 min duration. Safar (1986) has described the post-resuscitation syndrome of secondary cerebral perfusion failure, cerebral re-oxygenation injury and cerebral intoxication after ischaemic damage to extra-cerebral organs, for example liver and kidneys, for which many therapeutic regimes are being investigated including barbiturates, calcium antagonists and iron chelators, but none are yet proven. Advanced training of ambulance crews and public education initiatives, for example the Save a Life campaign, have attempted to reduce the duration of cardiac arrest; however, once CPR is initiated, monitoring of oxygen delivery to ensure optimal cerebral oxygenation has only been possible in a few units employing early invasive techniques not normally available during the initial phase of CPR for patients in cardiac arrest in A&E. Non-invasive methods include transcutaneous and conjunctival sensors. Conjunctiva has several advantages over other non-invasive routes of monitoring tissue oxygenation. When placed against the superior palpebral conjunctiva the sensor is $< 30 \mu$ away from the capillary bed. This eliminates abnormal values which may be obtained with transcutaneous systems as a result of skin heating causing increases in blood flow, oxygen diffusion and oxygen dissociation. A variable barrier of skin and adipose tissue may also influence oxygen measurements.

The use of the conjunctival capillary bed for measuring arterial oxygen tension was first described by Kwan & Fatt (1971). Early commercially available models were flawed by complex and time-consuming preparation of the sensor. Improved technology incorporated in the present model simply requires immersion of the sensor in normal saline for 1 h prior to use.

In haemodynamically stable patients there is a linear relationship between C_iO_2 and arterial oxygen tension. This relationship is lost in haemodynamically unstable patients, where C_iO_2 reflects oxygen delivery and tissue oxygenation (Shoemaker *et al.*, 1984). In shock there is a redistribution of blood flow from the peripheral to the central circulation with an estimated 77% shunt of blood from superficial areas beneath the sensors to vital organs (Abraham *et al.*, 1984).

The common vascular supply of the palpebral conjunctiva and brain indicates that C_iO_2 may be an index of cerebral perfusion. The palpebral conjunctiva is predominantly supplied by the internal carotid artery via the medial palpebral branches of the ophthalmic artery and the lateral palpebral branches of the lacrimal artery (Warwick & Williams, 1973). The superior palpebral artery does, however, anastomose laterally with the zygomatico-orbital branch of the superficial temporal artery. Van der Zee (1985) demonstrated the effect of varying ventilation on C_iO_2 in rats. Temporary apnoea, hypoventilation and clamping of the common carotid arteries produced immediate falls in C_iO_2 . Hyperventilation produced an initial increase in C_iO_2 , reflecting improved cerebral oxygenation but subsequent vasoconstriction associated with a low $PaCO_2$ of 20 mmHg produced a fall in C_iO_2 . Nisam (1986) monitored C_iO_2 in normal healthy hyperventilating adults and reported similar significant falls in C_iO_2 associated with a $PaCO_2$ of 20 mmHg. Shoemaker & Lawner (1983) described fluctuations in C_iO_2 reflecting altered cerebral circulation with occlusion and manipulation of the carotid vessels in patients during carotid artery surgery. These reports support the use of C_iO_2 monitoring as a non-invasive method of evaluating cerebral oxygenation.

MATERIALS AND METHODS

The C_iO_2 sensor (TO₂M 2000 monitoring system—Biomedical Sensors Ltd, High Wycombe, England) is a Clark oxygen electrode and thermistor mounted on an acrylic conformer (Figure 1). This is stored in normal saline prior to use and placed beneath the eyelids so that the electrode is adjacent to the lateral aspect of the superior palpebral conjunctiva. The sensor stabilizes rapidly after insertion and has a reaction time of 45 s, rapidly reflecting changes in C_iO_2 . The normal C_iO_2 is 50–60 mmHg.

Fifteen patients in cardiac arrest occurring out of hospital or in A&E were monitored during CPR in A&E. The sensor was inserted within 60 s of arrival in the department of cardiac arrest, when that occurred in the department, and C_iO_2 recorded every 60 s. Resuscitation was performed using Resuscitation Council UK guidelines with closed chest cardiac massage, endotracheal intubation and drug administration via central lines.

Fig. 1 Conjunctival oxygen sensor.

RESULTS

Fifteen patients have been studied. Three cases are reported, which illustrate findings consistent throughout the study.

Case 1

A 74-year-old female collapsed at home. Basic CPR was performed by ambulance personnel en route to hospital. On arrival in A&E she was in cardiac arrest and the initial recorded rhythm was a pulseless bradycardia, which decayed to asystole. Standard CPR was performed and C_iO_2 monitored (Figure 2). There was a rapid fall in

Fig. 2 C_jO₂ during CPR in case 1.

C_jO₂, as the sensor stabilized, to <20 mmHg. CPR produced no increase in C_jO₂. Resuscitation was stopped after 22 min.

Case 2

A 68-year-old male collapsed in the street. A bystander performed mouth-to-mouth respiration but no cardiac massage. CPR was instigated by the ambulance crew, trained in defibrillation but no other facet of advanced cardiac life support. The presenting rhythm was ventricular fibrillation (VF) and defibrillation was performed twice en route to hospital but no stable rhythm obtained. On arrival in A&E approximately 10 min after collapse he was in cardiac arrest with VF. C_jO₂ recorded during resuscitation is shown in Figure 3. Initial values were low and showed no improvement with closed CPR. After 7 min a palpable carotid pulse returned and at 10 min spontaneous respiratory efforts were made. There was, however, a further delay of 8 min before C_jO₂ began to rise. At 19 min cardiac output was lost due to recurrent VF and CPR recommenced. This produced no improvement in C_jO₂, which only rose when spontaneous cardiac output returned, i.e. a palpable carotid pulse. A gradual rise in C_jO₂ followed. The patient survived, to be discharged home with a minimal residual neurological deficit.

Case 3

An 82-year-old male presented with an inferior myocardial infarction and arrested in VF in A&E. Immediate CPR was commenced and C_jO₂ recorded (Figure 4). During resuscitation there were four episodes of VF requiring defibrillation and closed CPR.

Fig. 3 C_iO_2 during resuscitation of a survivor of cardiac arrest (case 2).

Fig. 4 C_iO_2 during resuscitation of case 3.

Two of these, at 4 and 15 min, were brief producing only plateaux in the rise of C_iO_2 . Two episodes at 8 and 18 min were prolonged. CPR failed to correct the fall in C_iO_2 produced by loss of cardiac output. This patient survived neurologically intact.

DISCUSSION

Monitoring cerebral blood flow (CBF) and oxygenation in humans during CPR requires sophisticated invasive techniques presenting major practical difficulties and there is as yet no such data in the literature. The C_jO_2 sensor provides a non-invasive index of CBF appropriate for use in the critically ill patient in A&E. In this study C_jO_2 during cardiac arrest was neither maintained nor improved by closed chest cardiac massage. Return of spontaneous cardiac output indicated by palpable carotid pulsation produced immediate improvement in C_jO_2 where the duration of arrest was <5 min. With delay to CPR or prolonged arrest this rise in C_jO_2 was delayed. Palpable carotid pulse may not therefore guarantee satisfactory cerebral oxygenation in this group, due to sludging and vasospasm in the cerebral circulation and variable blood flow from the common carotid artery to supply the face (Krause *et al.*, 1986).

Optimal outcome after cardiac arrest is survival with no or minimal neurological deficit. This is achieved by rapid restoration of spontaneous cardiac output most consistently in those patients who arrest in VF in A&E. Figure 4 illustrates how cerebral oxygenation as reflected by C_jO_2 changes during such an event. RSCO elevated and maintained $C_jO_2 > 20$ mmHg, with an overall upward trend in C_jO_2 to a peak of 63 mmHg. Recurrent episodes of VF produced a plateau or fall in C_jO_2 , despite immediate closed CPR, which was only corrected with RSCO. Excellent neurological outcome in this patient ensued, however, as the episodes of VF were either very brief (<2 min) or if prolonged (<5 min) C_jO_2 had been restored > 30 mmHg prior to arrest. The brain was therefore oxygenated and could tolerate a short period of hypoxia with full recovery.

Survival with minimal neurological deficit may still be possible with prolonged downtime and CPR. Factors influencing neuronal survival, including the critical duration of downtime, are still to be determined although the brain may be more resilient to hypoxia than previously believed. Case 2 (Figure 3) arrested 10 min before arrival in A&E. He received modified bystander CPR, that is expired air resuscitation but no cardiac massage until the arrival of the ambulance crew who continued massage but were not trained to intubate and ventilation was achieved using a bag and mask. C_jO_2 remained < 20 mmHg for a further 16 min after arrival in A&E although a palpable carotid pulse was restored after 7 min. He survived with minimal neurological deficit despite 26 min elapsing from collapse to $C_jO_2 > 20$ mmHg. It is impossible to be sure of the exact state of cerebral oxygenation prior to arrival in A&E and there may have been some cardiac output with the rhythms following defibrillation en route to hospital. Subsequent RSCO within 6 min of arrival supports the possibility of a responsive viable although hypoxic myocardium, and presumably similar conditions in the brain. The persistent low C_jO_2 with palpable carotid pulse may have been due to cerebral sludging and vasospasm, or, diversion of carotid blood flow to the face. Under these circumstances low C_jO_2 would correctly reflect low cerebral oxygenation and patient survival with minimal neurological deficit only possible if the brain can tolerate prolonged hypoxia. Alternatively the low C_jO_2 may have resulted from peripheral vasoconstriction associated with prolonged cardiac arrest or vasoactive drugs, for example adrenaline, used during resuscitation. In these circumstances C_jO_2 will be a significant underestimate of true cerebral oxygenation, which may in fact be adequate to maintain neuronal

viability. The impact of any vasoconstrictive effect in this case had disappeared however at 18 min, when the single later episode of VF occurred. C_iO_2 then followed the course of case 2 with a simultaneous fall as cardiac output was lost, continuing although closed CPR was instituted immediately, and only rising with RSCO. The effect of adrenaline on C_iO_2 requires further investigation.

Jehle *et al.*, (1985) monitored C_iO_2 during and after CPR of dogs in cardiac arrest. C_iO_2 values 30 min after return of a spontaneous circulation reflected ultimate neurological outcome, and C_iO_2 during CPR was a predictor of cardiac and cerebral resuscitability.

There are few reports of the use of the C_iO_2 monitor during CPR in humans. Abraham *et al.*, (1984) monitored C_iO_2 in 11 patients during CPR. In two patients, one in cardiogenic shock preceding asystole and the other normotensive before a pulseless idioventricular rhythm, C_iO_2 fell 2–3 min before cardiac arrest. Both patients died and C_iO_2 remained < 20 mmHg throughout resuscitation, closed CPR producing no improvement in C_iO_2 .

Rutherford *et al.* (1987) monitored one patient in the post-resuscitation phase after out-of-hospital cardiac arrest. In the 60 min after arrest C_iO_2 rose steadily and neurological status improved. Both declined over the following 3 h although blood pressure remained stable (104 ± 18 mmHg) and $\text{PaO}_2 > 103$ mmHg, reflecting experimental observations following global brain ischaemia with, initially, localized areas of poor perfusion followed by transient global hyperaemia before a progressive decline in cerebral perfusion. C_iO_2 post arrest ranged from 27 to 70 mmHg but declined to a level < 20 mmHg during the final 45 min before death. C_iO_2 fell with or before changes in mean arterial pressure and preceded clinically apparent deterioration.

To maintain brain viability in cardiac arrest the critical cerebral blood flow (CBF) is 10–15 ml/min/100g (normal 60 ml/min/100g) approximately 20% of normal (Symon, 1985). Jackson *et al.* (1984) reported cortical CBF in dogs in cardiac arrest with closed CPR to be 9.8% of pre-arrest flow, and continued to fall with increasing time from arrest to initiation of CPR and with prolonged CPR. The use of high (0.2 mg/kg/min) dose adrenaline in swine after 10 min CPR increased CBF from 7 to 13 ml/min/100g to the cortex and > 29 ml/min/100g to caudal CNS structures (Brown *et al.*, 1986). This is due to α adrenergic effect producing shunt from extracerebral to intracerebral vessels although β -receptor mediated dilation of cerebral microvasculature may contribute. Sharff *et al.* (1984) found in swine that the falling cardiac output is redistributed, with a high percentage cephalad preserving CBF initially. This fell significantly over 10 min and reduced survival rates and poor neurological function followed prolonged CPR, emphasizing the need for rapid re-establishment of spontaneous rhythm and circulation. $\text{CBF} < 10\%$ normal is associated with more severe biochemical, cellular and functional injury than zero perfusion (Jackson *et al.*, 1984). Low CBF may be more harmful than no flow and exacerbate neurological injury.

In humans closed CPR generated cardiac output 25% and CBF 3–15% of pre-arrest values if commenced immediately after cardiac arrest (Krause *et al.*, 1986). This fell with prolonged CPR as thoracic compression raised central venous and intracranial pressure reducing cerebral perfusion pressure. Delay of 5 min from arrest to initiation of CPR resulted in no detectable cerebral perfusion. Krause concluded that closed CPR is effective for only 4–6 min in enhancing resuscitation or the chances of survival, and

must be initiated immediately and followed by definitive resuscitation within 6–8 min.

Open CPR has been repeatedly shown to produce CBF approaching physiological values and far higher than the critical 20% of normal flow required to maintain brain viability. Bircher & Safar (1985) compared neurological outcome in dogs after 30 min CPR. All animals receiving open CPR recovered normal neurological function but resuscitated with closed CPR (with standard CPR or simultaneous ventilation and compression) were brain dead. Stajduhar *et al.* (1983) performed open CPR in dogs and after 1 h achieved perfusion rates 42–105% of the pre-arrest flow (mean CBF 71%, mean arterial pressure 39%) with near normal EEG recordings. He concluded that open CPR should be considered for selected cases of long arrest times and ineffective external CPR. Del Guercio *et al.* (1965) agreed providing that circumstances and personnel permit a choice of method. They reported three patients who underwent open CPR, after closed CPR had failed, up to 45 min after cardiac arrest. Cardiac output was doubled and the mean circulation time significantly improved to near the normal physiological range. Present American Heart Association guidelines (1986) recommend open-chest cardiac massage in non-traumatic cardiac arrest for anatomic deformity of the chest or severe emphysema which may preclude adequate chest compressions and rarely for failure of adequately applied closed chest compression and refractory VF. Krause *et al.* (1986) suggest the early use of open CPR for those patients who do not respond promptly to defibrillation and airway control. Closed CPR has been described as at best a holding manoeuvre to permit defibrillation to be achieved (Robertson, 1988).

CONCLUSION

The results of this study indicate that closed CPR in humans neither maintains nor improves cerebral oxygenation in the cardiac arrest victim. If successful neurological outcome is to be achieved in cases of out-of-hospital cardiac arrest or prolonged in-hospital CPR, the role of open chest cardiac massage must be reviewed and the population for whom this manoeuvre is appropriate more carefully defined.

The short initial stabilization time and rapid reaction time of the C_jO_2 monitor allows use in the early phase of CPR where invasive monitoring is often impracticable. It is valuable adjunct in monitoring the rapidly changing physiological state during CPR and in the critical early post-resuscitation phase where changes in oxygen delivery detected by C_jO_2 monitoring may be the earliest parameter of a deteriorating clinical picture. Further studies are required to quantify the relationship between C_jO_2 and cerebral oxygenation during cardiac arrest and how this is influenced by open or closed chest cardiac massage; delay to or prolonged CPR; and the effect of vasoactive drugs used during resuscitation, in particular adrenaline.

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