Advances in Experimental Medicine and Biology
Volume 428

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CONTINUOUS MEASUREMENT OF CEREBRAL OXYGENATION BY NIRS DURING INDUCTION OF ANAESTHESIA

A. T. Lovell,[1] H. Owen-Reece,[1] C. E. Elwell,[2] M. Smith,[1] and J. C. Goldstone[1]

[1]Division of Academic Anaesthesia
Department of Surgery
University College London Medical School
The Middlesex Hospital
Mortimer St., London, W1N 8AA, United Kingdom
[2]Department of Medical Physics and Bioengineering
University College London
Shropshire House, 11-20 Capper St., London, WC1E 6JA, United Kingdom

1. INTRODUCTION

Continuous intraoperative monitoring of cerebral oxygenation is not routine because existing techniques are either invasive, require a prolonged period of equilibration or involve the use of ionizing radiation. The potential of near infrared spectroscopy (NIRS) as a non invasive tissue oxygenation monitor was first outlined by Jöbsis (Jöbsis, 1977). NIRS enables the continuous measurement of oxyhaemoglobin (HbO_2) and deoxyhaemoglobin (Hb). To date most of the studies that have used NIRS during anaesthesia have considered either the effects on cerebral dynamics of extreme manoeuvres, such as clamping of a carotid artery and induction of ventricular fibrillation (Kirkpatrick et al., 1995; Mason et al., 1994; Williams et al., 1995; Levy et al., 1995), or have been confined to measuring cerebral blood flow (CBF) or cerebral blood volume (CBV) (Owen-Reece et al., 1994; Owen-Reece et al., 1996).

There are no reports on the effects of administration of drugs at clinically used doses, a manoeuvre likely to have far smaller effects than clamping major blood vessels supplying the brain or inducing circulatory arrest.

2. AIM

To determine whether NIRS measurements of cerebral oxygenation alter during induction of anaesthesia.

Oxygen Transport to Tissue XIX, edited by Harrison and Delpy
Plenum Press, New York, 1997

3. METHODS

Twelve healthy ASA I or II patients scheduled to undergo minor surgery were studied. All patients were unpremedicated.

Δ[HbO_2] and Δ[Hb] were recorded using a NIRO 500 spectrophotometer (Hamamatsu Photonics, Japan) with optodes located in the temporoparietal region in order to avoid interogating the frontal sinuses. An intraoptode spacing of 4 cm was used, and the optodes were shielded from ambient light. A sampling interval of 0.5 sec was used and the changes in Δ[HbO_2] and Δ[Hb] were calculated with a previously established algorithm (Wray et al., 1988; Essenpreis et al., 1993). A differential pathlength factor of 6.26 was used (Duncan et al., 1995). Beat to beat arterial saturation was recorded using a modified Novametrix 520A pulse oximeter attached to an ear lobe. End tidal carbon dioxide tension was monitored with a mainstream capnograph (Novametrix 7000A) directly attached to a tightly fitting facemask. Data were continuously recorded and sampled synchronously with the NIRS data. Blood pressure and heart rate were monitored non invasively with a Datex Cardiocap, sampling every 60 seconds.

3.1. Procedure

All measurements were made with the patients in a supine position. All patients breathed room air for the first four minutes of the study. Then propofol, an intravenous induction agent, was administered intravenously at a rate of 10 mg sec^{-1} until loss of verbal contact was achieved. At this stage ventilation was gently assisted with a circuit fed with 2.5 l min^{-1} O_2 and 7.5 l min^{-1} N_2O to maintain E_TCO_2 constant at its preinduction value. Data was recorded for a further 3 minutes before anaesthesia was deepened and surgery allowed to proceed.

3.2. Data Analysis

We averaged the data into 15 second epochs. All the data from the baseline epochs were averaged together. Statistical analysis was carried out using SPSS 4.0. Analysis was by a repeated measures ANOVA model with post hoc paired t-tests with Bonferroni's correction, a corrected p value < 0.05 was taken as significant. All results are expressed as mean ± SD.

4. RESULTS

The demographics of the patients are shown in Table 1.

There were no significant differences in SpO_2 or E_TCO_2 or heart rate at any stage of the study compared to baseline values of 98.6±1.28 %, 5.0±0.46 kPa and 79±10 min^{-1}, respectively. The fall in blood pressure following induction of anaesthesia is shown in Table 2. This shows that mean arterial pressure undergoes an immediate 11% reduction reaching a plateau 15% below baseline three minutes after induction of anaesthesia. The changes in Δ[HbO_2] and Δ[Hb] are shown in figure 1. The Δ[Hb] signal did not significantly change following induction. However there was a highly significant increase in Δ[HbO_2], $p<0.000002$. Further examination of the results showed that from 75 seconds following commencement of induction all values were significantly different from baseline. The changes in Δ[HbO_2] and Δ[HbTot], the sum of Δ[HbO_2] and Δ[Hb] are shown in

Table 1. Patient demographics

Age	38 ± 13(20 - 63) yr
Weight	69.8 ± 12.2 kg
Sex	7 Female 5 Male
Haemoglobin	13.2 ± 0.89 g dl-1
Propofol dose	215 ± 44 mg(3.09 ± 0.47 mg kg-1)
Time to loss of consciousness	52 ± 20 sec

figure 2. The changes in Δ[HbO$_2$] are mirrored by Δ[HbTot]. Following induction of anaesthesia Δ[HbTot] also significantly increased, $p < 0.00002$. Further examination showed, that like Δ[HbO$_2$], Δ[HbTot] was significantly different from baseline from 75 seconds following commencement of induction until the end of the study.

5. DISCUSSION

NIRS was able to detect changes in Δ94Symbol”[HbO$_2$] and Δ94Symbol”[HbTot] associated with the induction of anaesthesia. The rise in both Δ94Symbol”[HbO$_2$] and Δ94Symbol”[HbTot] occurs very shortly after the loss of consciousness. These changes were at a time when no intravenous fluids were administered and so are unlikely to be related to changes in haematocrit. Since the haematocrit was constant, the Δ94Symbol”[HbTot] signal must represent an increase in the volume of blood in the field of observation. From scalp occlusion studies, NIRS signals do not originate solely from the scalp. The increase in Δ94Symbol”[HbTot] in this study may in part reflect an increase in cerebral blood volume (CBV). The changes in Δ94Symbol”[HbO$_2$] occurred at a time when the arterial saturation did not change. It seems likely that this increase in CBV was due to an increase in oxy-haemoglobin within the brain. The deoxy-haemoglobin signal did not significantly change. This data suggests an increase in the saturation of the brain. The NIRS signal has contributions from arterial, venous and capillary compartments and it is impossible with NIRS to determine whether the observed change occurred to an equal extent in all compartments.

In this study there may be many potential reasons for cerebral haemodynamics to change. Although mean arterial pressure fell during this study, it remained within the range of autoregulation and is less likely to contribute to the change in NIRS signals.

To explain our findings that CBV increased, we hypothesize that the brain either dilated or cerebral blood flow (CBF) increased. It is unlikely that CO_2 tension changes affected CBF in this study as it was held constant at each subject baseline.

Table 2. Changes in blood pressure following induction of anaesthesia

Time (min)	Systolic (mmHg)	Mean (mmHg)	Diastolic (mmHg)
Baseline	124 ± 22	90 ± 17	73 ± 15
1	110 ± 18*	80 ± 15*	64 ± 13*
2	110 ± 15*	80 ± 13*	65 ± 13
3	105 ± 14*	76 ± 12*	61 ± 11*

* $p < 0.05$ compared to baseline

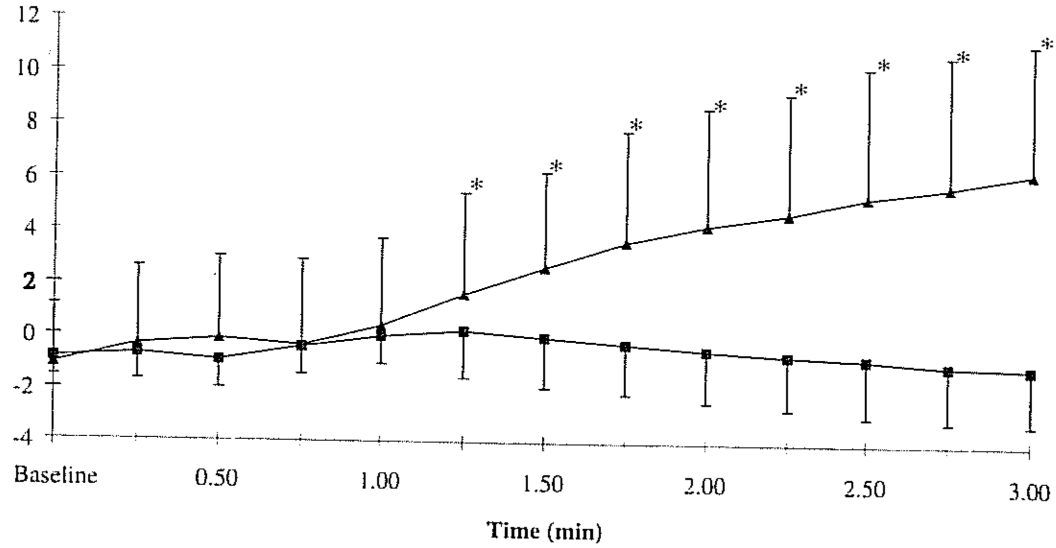

Figure 1. Changes in cerebral $\Delta[HbO_2]$ and $\Delta[Hb]$ following induction of anaesthesia with Propofol. s $\Delta[HbO_2]$ n $\Delta[Hb]$ * $p < 0.05$ compared to baseline.

Several investigators have demonstrated that N_2O vasodilates the cerebral circulation (Reinstrup et al., 1994), and in order to maintain anaesthesia during this study this drug was used.

The actions of intravenous anaesthetic agents on CBF are complex. Whilst cerebral vasoconstriction predominates clinically, direct application of thiopentone has demonstrated dilatation of pial arteries (Michenfelder, 1988). The net effect will be related to the balance between direct dilatation and vasoconstriction secondary to a fall in oxygen consumption ($CMRO_2$). It is known that propofol depresses $CMRO_2$ by 50% (Alkire et al., 1995).

We hypothesize that the increase in CBV that we observed following induction of anaesthesia was due to the direct vasodilating properties of propofol. It must take a period of time for the $CMRO_2$ to fall following propofol administration, and we suspect that only after this time would a vasoconstrictor response be seen. We did not observe the second phase of this response, but the timing of this study was limited by the desire to observe the

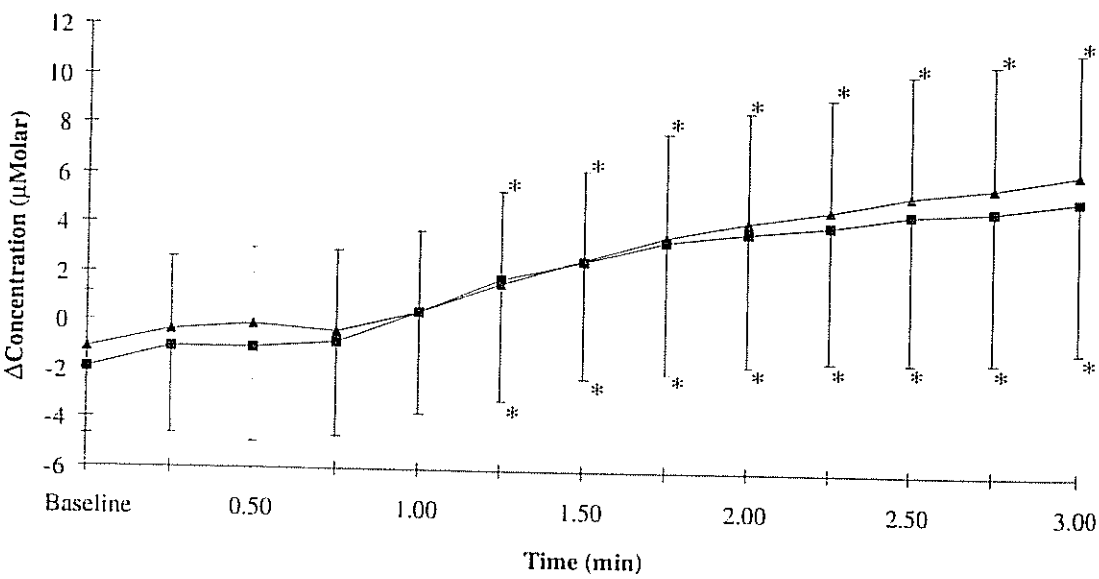

Figure 2. Changes in cerebral HbO_2 and HbTot following induction of anaesthesia with Propofol. s $\Delta[HbO_2]$ n $\Delta[HbTot]$ * $p < 0.05$ compared to baseline.

effects of a single administration of propofol. Single shot administration of propofol in unpremedicated patients only produces a brief duration of anaesthesia, thus limiting the duration of the study.

The relationship between the increase in CBV measured in 94Symbol"mMolar terms and that measured in ml $100g^{-1}$ has previously been described (Wyatt et al., 1990). This technique requires a gradual change in arterial saturation, and our study was designed to keep arterial saturation constant.

6. CONCLUSIONS

Cerebral oxygenation and cerebral blood volume increases very rapidly following induction of anaesthesia with propofol. These changes are temporally associated with the onset of anaesthesia.

Although NIRS was able to detect changes in cerebral blood volume associated with the induction of anaesthesia, the clinical significance of this finding is unlikely to be important. Care must be taken in interpreting changes in NIRS data under anaesthesia to ensure that anaesthetic depth is held constant.

ACKNOWLEDGMENT

This work has been supported by a grant from the Wellcome Trust.

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