Use of Transcranial Cerebral Oximeter as Indicator for Bifrontal Decompressive Craniectomy

S El-Watidy, A El-Dawlatly, Z Jamjoom, E El-Gamal

Citation

S El-Watidy, A El-Dawlatly, Z Jamjoom, E El-Gamal. *Use of Transcranial Cerebral Oximeter as Indicator for Bifrontal Decompressive Craniectomy*. The Internet Journal of Anesthesiology. 2003 Volume 8 Number 2.

Abstract

Objectives: The timing of bifrontal decompressive craniectomy (BDC) in patients with intractable intracranial hypertension (IH) is crucial, and the decision to do surgery is based primarily on invasive neuromonitoring. In this report the authors show the efficacy of a non-invasive, near infrared transcranial cerebral oximeter (TCCO) in the management of a patient with post-traumatic IH.

Clinical Presentation: A 14-year-old male patient who had severe head injury following road traffic accident (RTA). His Glasgow Come Score (GCS) was 6/15. Brain computerized tomography (CT) scan showed multiple brain contusions and diffuse brain edema. He developed a state of IH that did not respond to standard medical treatment. We have used TCCO for neuromonitoring, its readings showed marked difference between the two cerebral hemispheres and this correlated well with the clinical and radiological findings.

Intervention: Because of the decreasing trend of cerebral oxygen saturation and pupillary changes (anisocoria) BDC was performed. The timing of surgery was appropriate as no brain infarction occurred. Following surgery, TCCO readings were normal and the patient recovery was dramatic and relatively quick.

Conclusion: TCCO may be an efficient Neuromonitoring tool in determining the time for surgical interference in patients with IH following RTA.

INTRODUCTION

IH is a state of severely raised intracranial pressure (ICP) which does not respond to routine medical treatment. Patients most often will die or survive with severe disability. BDC is the last resort therapy in these instances. The decision to do surgical decompression is based primarily on invasive monitoring of the ICP. TCCO using near infrared light (700-900 wavelengths) is a non-invasive neuromonitoring modality used for monitoring cerebral oxygen saturation (rSO_2 %) in head injury patients ([1], [2]).

The aim of this report is to shed light on the adequacy and efficacy of TCCO as a sole indicator for decompressive craniectomy in patients with post-traumatic IH.

CASE REPORT

A 14-year-old male patient presented to the emergency department shortly after RTA. Upon arrival, he was unconscious, spontaneously breathing (20/min), heart rate 90/minute, blood pressure 113/83 mmHg, temperature 36.7C, and tissue oxygen saturation 97%. Neurologically his Glasgow coma scale (GCS) was 9/15; his pupils were 3mm equal, and reactive to light. He has a big occipital scalp laceration and large subgaleal hematoma. Systemic review was unremarkable. Shortly after admission to the emergency department, the patient's condition worsened to GCS 6/15. The left pupil became larger than the right and both had sluggish reaction to light. Immediately, the trachea was intubated and the lungs were hyperventilated. Mannitol 20% (0.5 gm/kg i.v bolus) was administered. A CT of the brain scan showed multiple brain contusions in the cerebellum, left frontal and temporal lobes, generalized brain edema, more on the left side causing compression of the lateral and third ventricles, effacement of cerebral sulci, and encroachment on basal cisterns. (see Fig 1.)

Figure 1

Figure 1: Initial CT brain scans showing left frontal contusion and diffuse brain edema with effacement of basal cisterns.

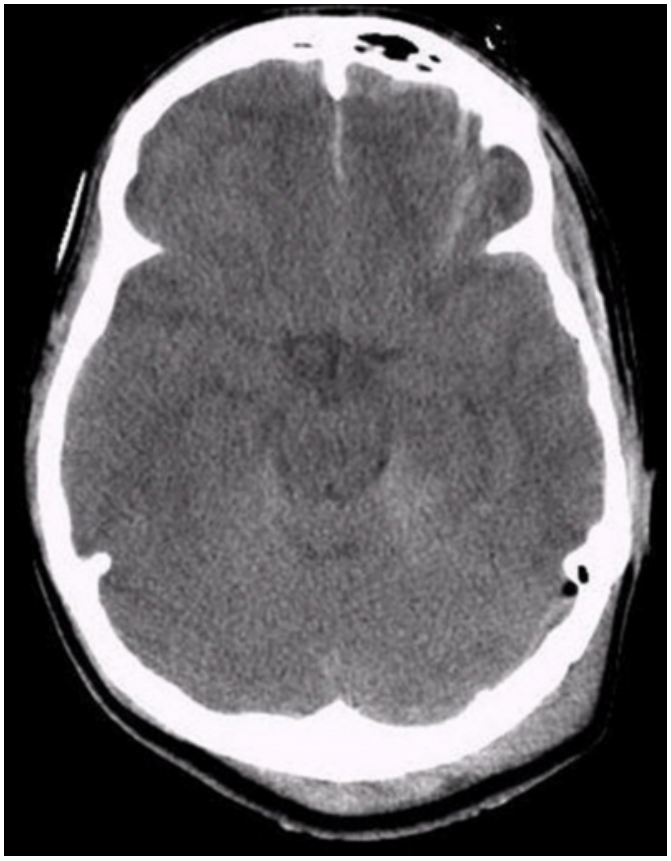
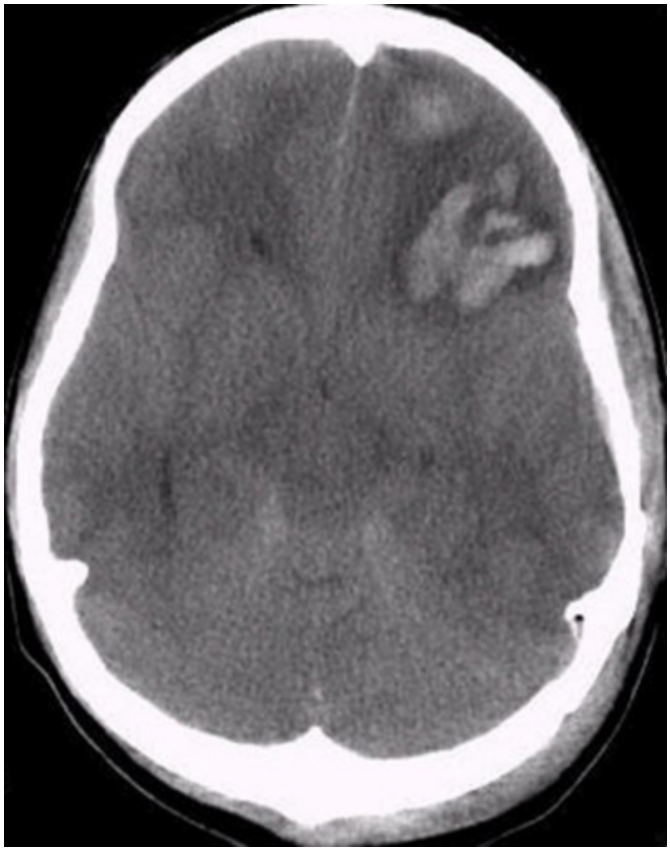


Figure 2

Figure 2: CT brain scan 8 hours after admission showing well developed brain contusion, midline shift, and complete obliteration of basal cisterns.



The patient was admitted to the intensive care unit and received standard treatment for such cases: sedation and muscle relaxation, normothermia, mild hyperventilation to keep PCO_2 between 30-35 mmHg, mannitol, and dopamine infusion was titrated to keep the mean BP 90 mmHg. TCCO neuromonitoring was used which showed difference in the initial readings from both cerebral hemispheres: left 57% and right 72%. The patient responded to mannitol and the pupils came down to 2mm and became equal. We decided to continue the same medical treatment and postpone surgical decompression. TCCO readings in the first 6 hours were more or less similar to the initial readings with more saturation in the right hemisphere.

About 8 hours after admission there was an attack of bradycardia associated with reduced oxygen saturation from cerebral hemispheres, right 65% and left 50%. Repeat CT of the brain scan showed more apparent brain contusions surrounded with edema, more swelling of the left hemisphere with more shift of midline structures, and complete obliteration of the basal cisterns. (see Fig. 2)

Immediately following CT scan, both pupils blown up with the left bigger than right. An extra bolus of mannitol was given and the patient was quickly taken to the theatre to undergo BDC. We have used the technique described by Kielberg and Parieto ($_3$) with some modifications. The patient head was kept slightly elevated on a horseshoe headrest. The bicoronal skin incision runs at a variable distance behind the coronal suture, the skin was raised together with the pericranium, and the temporalis fascia in one flap. The temporalis muscle was detached and retracted posteriorly and inferiorly on both sides. Two burr holes were done over the midline, the first one above the nasion (high enough to avoid opening the frontal air sinus), and the second one at variable distance behind the coronal suture (depending on the amount of bony decompression). Multiple burr holes were then made along the planned line of bone cuts. We use either craniotome or Gigli saw to cut the bone. After elevating the bone flap we performed wide bilateral subtemporal bony decompression. The dura was opened on both sides of the superior sagittal sinus which was cut between ligatures together with the falx cerebri to allow

simultaneous external herniation of both hemispheres without the risk of brain incarceration. Dural cuts extend laterally to the base of the middle cranial fossa and posteriorly parallel to the sinus.

Following inspection of the subdural space, evacuation of subdural clots and removal of lacerated brain, we closed the resultant dural defect using pericranial flaps. We closed skin in two layers leaving a subgaleal drain. Due to reflection of skin flap it is technically difficult to continue monitoring cerebral oxygen saturation during surgery. Immediately following decompressive craniectomy, the pupils came down to 2 mm and became equal but not reactive to light. The cerebral oxygen saturation showed dramatic improvement (right hemisphere 85% and left one 76%). Mannitol and dopamine were continued for three more days and then tapered off. Nasogastric tube feeding was started on the second day and PCO_2 was maintained around 35 mmHg. Neurologic recovery following BDC was quick and dramatic.

Two weeks later the trachea was extubated. The patient was able to open his eyes spontaneously and obeyed verbal commands at times. GCS was 9-10/15 and his pupils were equal and reactive to light. He had transient right arm weakness and dysphasia for three weeks. Follow-up CT brain scans (Fig 3, 4) showed the resolution of brain contusions, brain enema, and absence of brain infarction (Fig 5).

Figure 3

Figure 3: CT brain scan, one day after BDC, showing external herniation of the brain and reappearance of basal cistern, note the amount of bone removal.

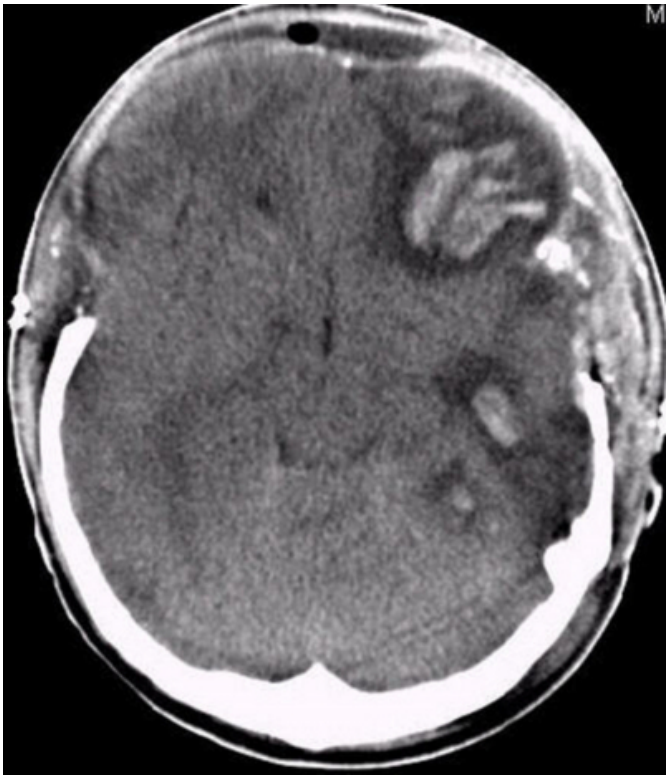


Figure 4

Figure 4: CT brain scan, 3 weeks after BDC showing complete resolution of brain contusions and brain edema and absence of brain infarction.

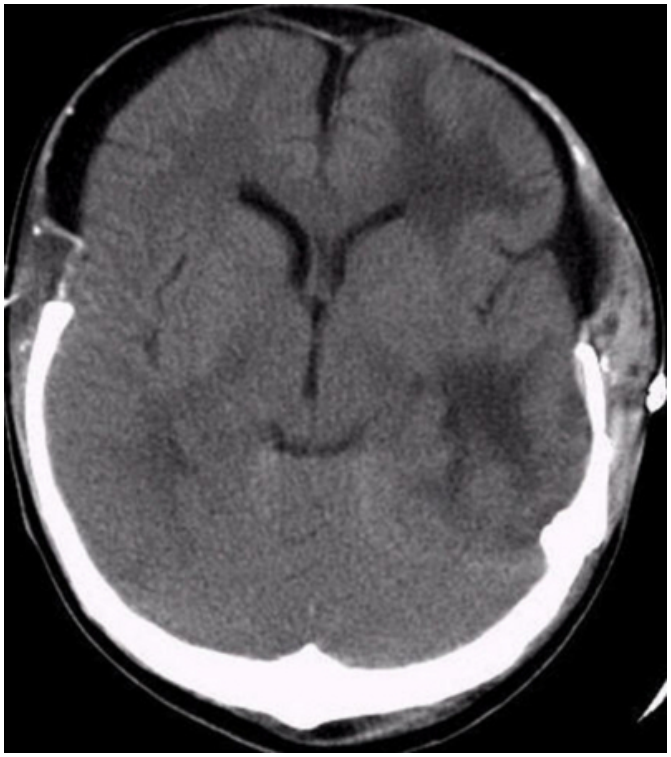
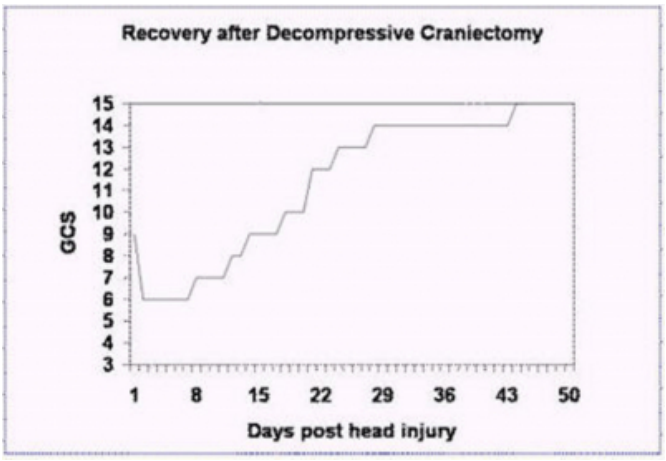


Figure 5

Figure 5: A graph representing patient recovery in six weeks time.



DISCUSSION

The standard protocol of intensive care treatment of patients with post traumatic IH entails invasive monitoring of ICP, brain tissue oxygen partial pressure ($PtiO_2$), jugular bulb oxygen saturation (SjO_2), and in some centers radioactive xenon cerebral blood flow measurements, brain temperature measurement and microdialysis. The cerebral Oximeter INVOS 3100, Somanetics ([4]), is a non-invasive monitor of the regional blood oxygen saturation in microvascular structures of the brain. It uses near infrared light source (wavelengths 700-900mm) and two detectors in skin pads that are fixed firmly with adhesive straps to the skin of the forehead to detect regional blood oxygen saturation. Saturations ≥ 75 suggest adequate cerebral perfusion pressure (CPP) and those < 55 suggest inadequate CPP ([5], [6]). When all medical measures fail in controlling intracranial pressure, brain infarction ensues and the patient most likely will die or end up with severe disability. Decompressive craniotomy is the last resort therapy in these instances ([7], [8], [9]). Different types of decompressive craniectomy are described in the literatures; most commonly used ones are wide subtemporal decompression, hemicraniectomy, and BDC. We prefer to use BDC as it is more physiologic and provides rapid relief and normalization of ICP and simultaneous bifrontal external herniation of the brain that alleviates the subfalcine and transtentorial brain herniation.

Following BDC, we quickly taper off the medical treatment (hypothermia, barbiturate coma, osmotic diuretics, and prolonged hyperventilation) to avoid its potential complications. The decision to do decompressive craniectomy after failure of medical treatment in patients with IH is based primarily on invasive monitoring of the ICP which is supposed to determine the timing for decompressive craniectomy. Proper timing of decompressive craniectomy by itself is an important predictor of the outcome. Ideally it should be performed before brain infarction and secondary brain damage occurs ([10], [11]).

To the best of our knowledge there are no report on the use of TCCO as a sole indicator for decompressive craniectomy. The authors in this report present their experience using this modality alone for neuromonitoring in a patient with post-traumatic IH. The initial readings of TCCO correlated well with the CT findings of multiple brain contusions and edema of the left hemisphere. This caused more reduction of CPP appeared as reduced oxygen saturation during monitoring and clinically as signs of lateralization. The abrupt reduction of cerebral oxygen saturation 8 hr later was quite significant and it was associated with pupillary changes and radiological evidence of transtentorial and subfalcine herniation. The decision to interfere surgically was based on both the reduction of cerebral oxygen saturation to levels suggestive of inadequate CPP and the simultaneous clinical deterioration (anisocoria and pupillary dilation not responding to mannitol infusion). The timing of surgery was

appropriate as no brain infarction was seen on postoperative CT brain scans. Immediately following BDC, the signs of brain herniation were reversed (small and equal pupils), and TCCO readings were suggestive of adequate CPP.

CONCLUSIONS

In conclusion, TCCO may be efficient and reliable non-invasive neuromonitoring. It indicates critical levels of reduced CPP that precedes brain infarction, and it may be a reliable indicator for BDC in patients with intractable intracranial hypertension.

References

1. Van Santbrink H, Mass A.IR, Avezaat C.JJ (1996). Continuous monitoring of partial tissue oxygen in patients with severe head injury. Neurosurgery 38: 21-31.
2. Olsen KS, Svendsen LB, Larsen FS (1996). Validation of Transcranial Near-Infrared Spectroscopy for Evaluation of Cerebral Blood Flow Autoregulation. J Neurosurg Anethesiol 8 (4): 280-285.
3. Kjelberg RN, Parieto A, Jr (1971). Bifrontal Decompressive Craniotomy for Massive Cerebral Edema. J Neurosurg 34 (4): 488-93.
4. Somanetics INVOS 3100 Cerebral Oximeter (1994). Neurosurgery 34 (5): 935.
5. Dunham CM, Sosnowski C, Porter JM (2002). Correlation of Non Invasive Cerebral Oximetry with Cerebral Perfusion in Severe Head Injured Patient: A pilot study. J Trauma 52:40-46.
6. El-Dawlatly AA, Alsalman M, Rabee H, Abuzaid A, Khyshzai RK (2001). Carotid Endarterectomy: A study of Cerebral Oxygenation. MEJ Anaesth 16: 155-160.
7. Whitfield PC, Patel H, Hutchinson PJ, Czosnyka M, Parry D, et al (2001). Bifrontal Decompressive Craniectomy in the Management of Post Traumatic Intracranial Hypertension. Br J Neurosurg 15 (6): 500-7.
8. Pollin RS, Shaffrey ME, Bogaev CA, Tisdale N, Germanson T, et al (1997). Decompressive Bifrontal Craniectomy in the Treatment of Severe Refractory Brain Edema. Neurosurgery 41 (1): 84-94.
9. Kontopoulos V, Foroglou N, Patsalas J, Magras J, Foroglou G, et al (2002). Decompressive Craniectomy for the Management of Patients with Refractory Hypertension: Should it be reconsidered?. Acta Neurochir 144: 791-796.
10. Morgalla MH, Krasznai L, Buchhloz R, Bitzer M, Deusch H, et al (1993). Repeated Decompressive Craniectomy after Head Injury in Children: Two Successful Cases as result of Improved Neuromonitoring. Surg Neurol 43 (6): 583-90.
11. Jaeger M, Soehle M, Meixensberger J (2003). Effects of Decompressive Craniectomy on Brain Tissue Oxygen (PtiO2) in Patients with Intracranial Hypertension. J Neurol Neurosurg Psychiatry 74 (4): 513-5.

Author Information

Sherif El-Watidy, FRCS(SN), MD
Assistant Professor, Neurosurgery Division, College of Medicine, King Saud University

Abdelazeem El-Dawlatly, MD
Associate Professor, Department of Anesthesia, College of Medicine, King Saud University

Zain A. Jamjoom, MD
Professor of Neurosurgery, College of Medicine, King Saud University

Essam El-Gamal, FRCS(SN)
Assistant Professor, Neurosurgery Division, College of Medicine, King Saud University