Decompressive craniectomy following brain injury: factors important to patient outcome

Patrick O. Eghwrudjakpor* and Akaribari B. Allison

Department of Surgery, University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria

Background: Decompressive craniectomy (DC) is often performed as an empirical lifesaving measure to protect the injured brain from the damaging effects of propagating oedema and intracranial hypertension. However, there are no clearly defined indications or specified guidelines for patient selection for the procedure.

Aims: To evaluate outcome determinants and factors important in patient selection for the procedure.

Methods: We reviewed the literature on DC, including single case reports and reported case series, to identify factors affecting outcome following the procedure, as well as its pitfalls and associated complications.

Results: Glasgow coma score of 8 and above, age less than 50 years and early intervention were found to be among the most significant determinants of prognosis.

Conclusion: Improving patient selection for DC may be expected to further improve the outcome following the procedure in severely brain-injured patients.

Keywords: decompressive craniectomy; indications; intracranial hypertension; outcome; patient selection

Received for publication: 27 July 2009; Accepted in revised form: 20 October 2009; Published: 7 January 2010

ecompressive craniectomy (DC) refers to the removal of an area of skull in order to enhance the potential volume of the intracranial compartment. It was first described by Kocher in the treatment of post-traumatic brain oedema which was refractory to conventional medical treatment in 1901 (1). Since then, interest in the procedure has either increased or decreased at various times. At present, however, it is commonly accepted as a means of rapidly relieving intracranial hypertension associated with a number of clinical conditions. The procedure however remains controversial owing to unresolved issues (2).

Despite the renewed interest in DC during the last decade, there are as yet no clear-cut guidelines regarding the indications for, or optimal timing of the procedure (3,4).

In this paper, we briefly review aspects of DC including: its documented benefits, the outcome determinants and the associated complications using the following search terms: 'decompressive craniectomy', 'outcome of', 'complications of', 'indications for', 'infarction', 'brain injury' and 'cranioplasty'.

Cerebral oedema and intracranial hypertension

Cerebral oedema and intracranial hypertension are among the most fundamental pathophysiological processes

occurring in several neurologic conditions including: sub-arachnoid haemorrhage (SAH), traumatic brain injury (TBI), cerebral infarction, cerebral blood flow abnormalities, inadequate oxygen delivery and energy failure. The impaired cerebral perfusion resulting from the increased pressure precipitates further increases and accounts for the vicious cycle leading to cell injury and death. A major goal in the treatment of these patients is, therefore, the interruption of the vicious cycle by controlling the brain swelling and maintaining the intracranial pressure (ICP) below target. Failure to interrupt this cycle is thought to be a significant contributor to poor outcome in the patients – many of whom will either die or survive with severe disability (with mortality exceeding 80% being reported in some series) (5).

Several modes of intervention have been applied in cases of intracranial hypertension. Most of them are effective and include therapies like the use of osmotic diuretics (such as mannitol or hypertonic saline), sedation, high-dose barbiturates, mild hyperventilation, moderate hypothermia, maintenance of oxygenation and drainage of cerebrospinal fluid by ventriculostomy (5). Clinical evidence, however, shows that these measures are not always effective, and as a result the vicious cycle continues to propagate. In such situations, more

aggressive methods of treatment are indicated. Furthermore, some of the conventional measures have been associated with significant side effects. For example, whereas mannitol is known to cause adverse effects like pulmonary congestion, convulsions, rebound intracranial hypertension, paradoxical increase of ICP (3) as well as fluid and electrolyte disorders; barbiturates have been reported to cause hypotension and depressed cardiac function while rebound increase in ICP has been known to occur following hyperventilation.

DC is thought to be a potential option in these instances (5), with the decision to intervene preferably being based on invasive monitoring of the ICP (6, 7).

Decompressive craniectomy (DC)

Surgical decompression as a means of relieving ICP is an old neurosurgical concept (3). Mainly, it involves raising a bone flap, duraplasty, cerebrospinal fluid drainage and removal of any intracranial mass lesions. The modern concept of decompression for TBI was introduced by Harvey Cushing in the early 20th century (8). DC refers to the removal of an area of skull bone with the aim of converting the 'closed' intracranial compartment into an 'open' one.

Interest in the procedure has fluctuated through the years partly due to a number of unresolved issues including: whether the results justify the treatment as well as the associated complications (such as the increased tendency of brain injury to occur at the craniectomy site) (9). There has also been concern about the functional outcome in surviving patients. Nevertheless, there has been a revival of interest during the last couple of decades; and it has come to gain wide acceptance as a salvage procedure in the treatment of refractory intracranial hypertension in a number of clinical conditions which are accompanied by massive oedema and brain swelling (10-12). The adverse effects of intracranial hypertension are due to compression of the brain as well as impairment of cerebral blood flow. DC reduces this pressure and enhances blood flow; and it has been shown that the larger the craniectomy, the greater the reduction of the ICP (14).

Clinical data show that DC is a safe and effective primary surgical procedure. Its role in the treatment of patients with intracranial hypertension associated with post-traumatic brain swelling is, however, still controversial (3, 12).

Even though the optimum size of the craniectomy is still a subject of controversy, clinical evidence shows that sub-optimal bone windows increase the chances of brain injury and thereby contribute to poor outcome. A craniectomy of at least 12 cm is recommended (15). However, the size of the bone flap should be tailored to meet the individual need. In their retrospective study of 263 patients with severe TBI that were treated with large DC (135 patients) or routine DC (128 patients), Li et al. (13) compared the treatment outcome and postoperative complications of the two treatment methods during a sixmonth follow-up period. They found that whereas large DC is superior to routine DC in improving the outcome of severe TBI and effectively reducing the chances of reoperation, it is also associated with a higher incidence of delayed complications such as intracranial haematoma and contralateral subdural effusion (13).

Other controversial aspects of DC include: the functional outcome following the procedure as it relates to patient selection criteria as well as surgical timing; its benefits in the treatment of patients with massive infarction of the territory of the middle cerebral artery (MCA) territory (16); whether the craniectomy should be unilateral or bilateral; and whether or not durotomy or duraplasty should be performed.

Despite the controversies, several studies have documented beneficial effects due to its performance. The advantages DC has over more conservative approaches to ICP control are thought to be due to the rapid and generally permanent decline in ICP, maintenance of neurologic status and the ability to obtain a neurologic examination after it is performed (8). Some studies have found that DC also improves cerebral perfusion pressure and cerebral blood flow in head-injured patients (17, 19).

Clinical data also indicate that DC reduces mortality, improves functional recovery, reduces duration of stay in intensive care unit and improves the Barthel Index Score, especially when it is performed early (3, 19–30). Guerra et al. reported that up to 65% of their patients who underwent DC for diffuse brain swelling refractory to medical management made a good recovery at one year (1). In experimental models of TBI and ischaemic stroke, it has been demonstrated that DC minimises posttraumatic ICP increase, improves cerebral perfusion, significantly reduces secondary brain damage and improves survival and functional outcome. These effects are thought to be the result of increases in collateral circulation, reductions in tissue oedema and improvements in oxygenation and energy metabolism in injured tissues (9, 31).

Children

Studies have shown that the majority of severely braininjured children in whom early DC was performed benefited from the procedure as demonstrated by the prompt control of the ICP, improvement in radiological findings and good neurological recovery. It is suggested that the procedure has advantage over non-surgical methods of treatment among children (32-35).

Indications

In spite of the fact that there are numerous reports in the literature supporting good clinical outcome after DC,

there are no clearly defined indications for, or optimal timing of the procedure (3, 4, 9). DC has most commonly been performed in patients with TBI (5, 36) and cerebral infarction (37) associated with intractable intracranial hypertension. Other indications, which have mostly been described in single case reports or small case series, include meningitis (38), subdural empyema, encephalitis (39), acute disseminated encephalomyelitis (40), encephalopathy due to Reye syndrome (41), toxoplasmosis (42), and cerebral venous and dural sinus thrombosis (43).

Various workers have based their decision to operate on different premises. Reddy et al., for example, based their decision to perform DC on the presence of mass effect with midline shift on neuroimaging and the impairment of consciousness to Glasgow Coma Scale (GCS) of 12 and below (3). Albanèse et al., on the other hand, performed early DC (within 24 hours) among their head-injured patients, if they had a GCS score of less than 6 and showed clinical signs of cerebral herniation (which were correlated with abnormalities on computed tomography scan – such as the presence of haematoma or brain swelling). Similarly, they used as indications for 'late' decompression (i.e. more than 24 hours) an intractable intracranial hypertension – with ICP of above 35 mm Hg, absence of pupillary reflexes and CT abnormalities (12).

Outcome determinants

Clinical data show that outcome in many of the survivors of DC is acceptable (44–47). Using the Glasgow Outcome Score Extended (GOSE), functional outcome was generally adjudged as good in several studies. Evidence from some of these studies indicates that one of the most important determinants of outcome is the timing of the procedure: with good outcome correlating with early surgery (generally within 48 hours). Younger patients generally fare better, with age greater than 50 years being associated with a poorer outcome. GCS score of 8 and above is associated with favourable outcome, while mortality rates and the incidence of residual disabilities are much higher in patients with admission GCS of 5 and below (3, 5, 48, 49) (see Table 1).

Other factors that have been associated with poor outcome include: polytrauma and significant pupillary abnormalities (anisocoria or mydriasis). It is, however, believed that of all these factors, the only one that is statistically related to bad prognosis is GCS at the time of admission.

Pitfalls and complications

Despite the documented benefits of DC, a number of workers have expressed concern as to whether the procedure has always been performed only on patients that actually needed it, or whether it has also been performed in cases that probably would have benefited from medical treatment alone. This is pertinent in view of the fact that the risk of complications following the procedure is comparatively high, with some studies reporting as much as 50% complication rate postoperatively (50).

A very important drawback of DC is the increased risk of brain injury. Honeybul reports the case of a middle-aged man who had a DC following TBI as a result of a fall. The patient was reported to be making good recovery when he fell a second time and injured the unprotected craniectomy site. As a result, he suffered further cerebral injury and subsequently died (51). The case highlights the need to view these patients as particularly high risk and emphasises the importance of measures aimed at protecting the brain after the procedure.

The fact that a minimum of two surgical procedures are required – the first being the actual removal of the bone flap and the second to repair the defect (cranioplasty) – is also a potential cause of concern (8, 52) since the latter has also been associated with a number of complications.

A major specific complication that has been associated with DC is the syndrome of sinking skin flap described by Yamaura and Makino. It is characterised by progressive neurological deterioration with the depression of the skin at the site of the cranial defect, and develops within a few weeks to several months after large external cerebral decompression. These authors opine that the neurological deterioration may be due solely to the effect of the concavity of the skin flap with consequent distortion of the underlying brain which is subjected to the atmospheric pressure through it (2).

Other reported complications of DC include: contralateral subdural effusions (53), infections (such as meningitis or brain abscess) and hydrocephalus (10). Persistent vegetative state is probably one of the most devastating outcomes following DC (54). It is thus necessary that in taking the decision to operate, the risk of complications should be weighed against the potential benefits of the procedure in the context of the life-threatening circum-

Cranioplasty, which is commonly indicated for large cranial defects following DC, is also associated with several complications including extradural haematoma, infections and instability of the implant (52, 55, 56), among others. Cranioplasty is generally performed three months after the DC. Clinical data, however, reveal that the rate of complications is reduced when it is performed early. Thus, there is at present, a tendency to perform it within 5–8 weeks of the craniectomy (57).

Ongoing trials

There are at present two prospective randomised controlled trials aimed at providing Class I evidence on the role of DC in the treatment of intracranial hypertension following severe TBI. The DECompressive CRAniectomy

Table 1. Decompressive craniectomy following brain injury: factors and considerations in patient selection that have been found to be important to patient outcome.

1.	Failed pharmacotherapeutic intervention	Sustained intracranial hypertension which does not respond to conservative strategies carries a bad prognosis, with mortality exceeding 80% being reported in some series. Decompressive craniectomy (DC) is often performed as a final option in the treatment of such cases (1, 5).
2.	Timing	Early DC (within 48 hours of injury) has been associated with good functional outcome. Reports indicate that neurological recovery is comparatively inferior among patients in whom surgery was delayed (26, 61).
3.	Brain herniation	DC should be performed before the development of neurological features of brain herniation. Evaluation of the functional recovery of patients, using the Glasgow Outcome Scale and Barthel Index, showed that patients who underwent DC before the occurrence of brain herniation had comparatively more satisfactory outcome than those in whom the procedure was performed after onset of herniation (62, 63).
4.	Glasgow Coma Scale (GCS) score	Score should be at least 8. Lower GCS scores appear to be associated with a poorer outcome. Studies indicate that most of the mortalities were among patients that had GCS of 4–6 at the time of craniectomy; whereas the overwhelming majority of the survivors were those who had higher GCS scores (8 and above). Reddy et al. reported 88% survival among their patients who had a preoperative GCS of 8 and above, and 27% survival among those with GCS less than 8 (3, 5, 18, 58).
5.	Patient's age	Should be less than 50 years. Age is perhaps one of the key factors in taking the decision whether or not to perform DC. Patients in younger age groups tend to do better after surgery, with age greater than 50 years being associated with a poorer outcome. The incidence of complications is also higher above this age (1, 18, 32, 58, 64, 66).
6.	Primary brainstem injury	There should be no primary brainstem injury. The chances of survival following DC in patients with primary brainstem injury are greatly reduced and as such several authors consider this a contraindication to this form of intervention (1, 66).
7.	Abnormal pupillary findings	Clinical data show that recurrent or persistent absence of pupil reflexes indicates a poor neurological outcome (5, 18).
8.	Intracranial pressure	Should preferably be less than 40 mm Hg at the time of decompression. Clinical data show that patients with sustained ICP of more than 40 mm Hg did comparatively poorly after DC as compared to those whose ICP was lower at the time of surgery (26).
9.	Midline shift	The degree of midline shift in the initial computed tomography has been found to correlate well with the quality of outcome following DC. Preoperative midline shift greater than 1 cm is believed to be a significant predictor of poor outcome (18, 65).

(DECRA) Trial is a multi-centre prospective randomised trial designed to evaluate the effect of early DC on neurological function in patients with severe TBI. It is based on the theory that early DC can improve long-term neurological outcome in patients with severe TBI and intracranial hypertension which is refractory to conventional management (58). Randomised Evaluation of Surgery with Craniectomy for Uncontrollable Elevation of ICP(RESCUEicp) is another prospective, randomised international multi-centre trial aimed at providing Class I evidence as to whether DC is effective for the management of patients with refractory intracranial hypertension following TBI as compared with medical management alone (59).

A major limitation of this review is that standard data meta-analysis techniques could not be applied; and like several other publications on DC in the literature, it tended to be biased in favour of publications with good outcome. There was non-uniformity in several aspects of the studies evaluated, many of which were based on uncontrolled retrospective data and small case series. Some of the areas of variation in the reports were the differences in detail in the surgical procedures performed as well as their indications: e.g. the choice of hemicraniectomy instead of bilateral craniectomy, durotomy or duraplasty, GCS score that was deemed acceptable, etc. all of which without doubt impacted (at least to some extent) the interpretation of the results.

Conclusion

DC is commonly performed as an empiric lifesaving measure in an attempt to protect the brain from the damaging effects of propagating oedema and intracranial hypertension (60). Improving patient selection and optimising timing of the procedure may be expected to further improve outcome in severely brain-injured patients. An important way of achieving this is to have clearly defined guidelines that may be applied in every case for which the procedure is envisaged. Table 1 shows the factors and considerations in patient selection and timing of DC that have been found to be important to patient outcome.

Conflict of interest and funding

The authors have not received any funding or benefits from industry to conduct this study.

References

- 1. Guerra WK, Gaah MR, Dietz H, Mueller J, Piek J, Fritsch MJ. Surgical decompression for traumatic brain swelling: indications and results. J Neurosurg. 1999; 90: 187-96.
- 2. Yamaura A, Makino H. Neurological deficits in the presence of the sinking skin flap following decompressive craniectomy. Neurol Med Chir (Tokyo). 1997; 17: 43-53.
- 3. Reddy AK, Saradhi V, Panigrahi M, Rao TN, Tripathi P, Meena AK. Decompressive craniectomy for stroke: indications and results. Neurol India. 2002; 50: 66-9.
- 4. Kalia KK, Yonas H. An aggressive approach to massive MCA infarction. Arch Neurol. 1993; 50: 1293-7.
- 5. van Veen E, Aerdts S, van den Brink W. Decompressive (hemi)craniectomy for refractory intracranial hypertension after traumatic brain injury. Critical Care 2006; 10: p. 458 doi:10.1186/cc4805.
- 6. Morgalla MH, Krasznai L, Buchholz R, Bitzer M, Deusch H, Walz GU, et al. Repeated decompressive craniectomy after head injury in children: two successful cases as result of improved neuromonitoring. Surg Neurol 1993; 43: 583-90.
- 7. Jaeger M, Soehle M, Meixensberger J. Effects of decompressive craniectomy on brain tissue oxygen (PtiO2) in patients with intracranial hypertension. J Neurol Neurosurg Psychiatry. 2003;
- 8. Polin RS, Ayad M, Jane JA. Decompressive craniectomy in paediatric patients. Crit Care. 2003; 7: 409-10.
- 9. Hutchinson P, Timofeev I, Kirkpatrick P. Surgery for brain oedema: decompressive craniectomy. 2007. Available from: http://www.medscape.com/viewarticle/559009_2 [cited 13 July 2009].
- 10. Piek J. Decompressive surgery in the treatment of traumatic brain injury. Curr Opin Crit Care. 2002; 8: 134-8.
- 11. Figaji AA, Fieggen AG, Peter JC. Early decompressive craniotomy in children with severe traumatic brain injury. Childs Nerv Syst. 2003; 19: 666-73.
- 12. Albanèse J, Leone M, Alliez JR, Kaya JM, Antonini F, Alliez B, et al. Decompressive craniectomy for severe traumatic brain injury: evaluation of the effects at one year. Crit Care Med. 2003; 31: 2535-8.
- 13. Li G, Wen L, Yang X, Zheng X, Zhan R, Liu W. Efficacy of large decompressive craniectomy in severe traumatic brain injury. Chin J Traumatol (English Edition). 2008; 11: 253-6.
- 14. Skoglund TS, Eriksson-Ritzen C, Jensen C, Rydenhag B. Aspects on decompressive craniectomy in patients with traumatic head injuries. J Neurotrauma 2006; 23: 1502-59.
- 15. Wagner S, Schnippering H, Aschoff A, Koziol JA, Schwab S, Steiner T. Suboptimum hemicraniectomy as a cause of additional cerebral lesions in patients with malignant infarct of the MCA. J Neurosurg. 2001; 94: 693-6.
- 16. Kazuhiko K. Decompressive craniectomy for massive infarction of middle cerebral artery territory. Neurol Surg. 2001; 29: 831–5.

- 17. Seppelt I. Intracranial hypertension after traumatic brain injury. Indian J Crit Care Med 2004; 8: 120-6.
- KIlInçer C, Asil T, Utku U, BalcI K, HamacIoglu MK. Decompression craniotomy. J Neurosurg 2007; 107: 1276–1278.
- 19. Kunze E, Meixensberger J, Janka M, Sorensen N, Roosen K. Decompressive craniectomy in patients with uncontrollable intracranial hypertension. Acta Neurochir. 1998; 71: 16-8.
- 20. Engelhard K, Müller-Forell W, Werner C. Therapy of head trauma. Anaesthesist. 2008; 57: 1219-31.
- 21. Aarabi B, Hesdorffer DC, Ahn ES, Aresco C, Scalea TM, Eisenberg HM. Outcome following decompressive craniectomy for malignant swelling due to severe head injury. J Neurosurg. 2006; 104: 469-79.
- 22. Moulin DE, Lo R, Chiang J, Barnett HJ. Prognosis in middle cerebral artery occlusion. Stroke 1985; 16: 282-4.
- 23. Heinsius T, Bogousslavsky J, Van Melle J. Large infarcts in middle cerebral artery territory: aetiology and outcome patterns. Neurology. 1998; 50: 341-50.
- 24. Hacke W, Schwab S, Horn M, Spranger M, De Georgia M, von Kummer R. Malignant MCA territory infarction. Arch Neurol 1996; 53: 309-5.
- 25. Andrews BT, Pitts LH. Functional recovery after traumatic transtentorial herniation. Neurosurgery. 1991; 29: 227-31.
- 26. Polin RS, Shaffrey ME, Bogaev CA, Tisdale N, Germanson T, Bocchicchio B, et al. Decompressive bifrontal craniotomy in treatment of severe refractory post traumatic cerebral oedema. Neurosurgery 1997; 41: 84-94.
- 27. Oppenheim C, Samson Y, Manaï R, Lalam T, Vandamme X, Crozier S, et al. Prediction of malignant MCA infarction by DWI. Stroke 2000; 31: 2175-81.
- 28. Mori K, Aoki T, Yamamoto T, Horinaka N, Maeda M. Aggressive decompressive treatment in patients with massive hemispheric embolic of infarction associated with severe brain swelling. Acta Neurochir 2001; 143: 483-91.
- 29. Kapadia FN, Masurkar VA, Sankhe MS, Gursahani RD. An audit of decompressive craniectomies. Indian J Crit Care Med. 2006; 10: 21-4.
- 30. Williams RF, Magnotti LJ, Croce MA, Hargraves BB, Fischer PE, Schroeppel TJ, et al. Impact of decompressive craniectomy on functional outcome after severe traumatic brain injury. J Trauma Inj Infect Crit Care. 2009; 66: 1570-6.
- 31. Zweckberger K, Stoffel M, Baethmann A, Plesnila N. Effect of decompression craniotomy on increase of contusion volume and functional outcome after controlled cortical impact in mice. J Neurotrauma. 2003; 20: 1307-4.
- 32. Hejazi N, Witzmann A, Fae P. Unilateral decompressive craniectomy for children with severe brain injury. Report of seven cases and review of the relevant literature. Eur J Pediatr. 2002; 161: 99-4.
- 33. Jagannathan J, Okonkwo DO, Dumont AS. Outcome following decompressive craniectomy in children with severe traumatic brain injury: a 10-year single-centre experience with long-term follow up. J Neurosurg Pediatr. 2007; 106: 268-75.
- 34. Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, et al. A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. Child. s Nerv Syst. 2001; 17: 154-62.
- 35. Simma B, Tscharre A, Hejazi N, Krasznai L, Fae P. Neurologic outcome after decompressive craniectomy in children. Intensive Care Med. 2002; 28: 1000.
- 36. Faleiro RM, Faleiro LCM, Caetano E, Gomide I, Pita C, Coelho G, et al. Decompressive craniotomy: prognostic factors and complications in 89 patients. Arq Neuro-Psiquiatr. 2008; 66:
- 37. Vahedi K, Hofmeijer J, Juettler E, Vicaut E, George B, Algra A, et al. Early decompressive surgery in malignant infarction of the

- middle cerebral artery: a pooled analysis of three randomised controlled trials. Lancet Neurol 2007; 6: 215-22.
- 38. Baussart B, Cheisson G, Compain M, Leblanc PE, Tadie M, Benhamou D, et al. Multimodal cerebral monitoring and decompressive surgery for the treatment of severe bacterial meningitis with increased intracranial pressure. Acta Anaesthesiol Scand. 2006; 50: 762-5.
- 39. Schwab S, Junger E, Spranger M, Dorfler A, Albert F, Steiner HH, et al. Craniectomy: an aggressive treatment approach in severe encephalitis. Neurology 1997; 48: 412-7.
- 40. Refai D, Lee MC, Goldenberg FD, Frank JI. Decompressive hemicraniectomy for acute disseminated encephalomyelitis: case report. Neurosurgery 2005; 56: 871-2.
- 41. Ausman JI, Rogers C, Sharp HL. Decompressive craniectomy for the encephalopathy of Reye's syndrome. Surg Neurol. 1976;
- 42. Agrawal D, Hussain N. Decompressive craniectomy in cerebral toxoplasmosis. Eur J Clin Microbiol Infect Dis. 2005; 24: 772–3.
- 43. Keller E, Pangalu A, Fandino J, Konu D, Yonekawa Y. Decompressive craniectomy in severe cerebral venous and dural sinus thrombosis. Acta Neurochir (Suppl). 2005; 94: 177-83.
- 44. Howard JL, Cipolle MD, Anderson M, Sabella V, Shollenberger D, Li PM, et al. Outcome after decompressive craniectomy for the treatment of severe traumatic brain injury. J Trauma. 2008; 65: 380-5.
- 45. Pfefferkorn T, Eppinger U, Linn J, Birnbaum T, Herzog J, Straube A, et al. Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. Stroke 2009: 40: 3045-50.
- 46. Aarabi B, Hesdorffer DC, Ahn ES, Aresco C, Scalea TM, Eisenberg HM. Outcome following decompressive craniectomy for malignant swelling due to severe head injury. J Neurosurg. 2006; 104: 469-79.
- 47. Williams RF, Magnotti LJ, Croce MA, Hargraves BB, Fischer PE, Schroeppel TJ, et al. Impact of decompressive craniectomy on functional outcome after severe traumatic brain injury. J Trauma Inj Infec Crit Care 2009; 66: 1570-6.
- 48. Stevier T, Ringlab P, Hacke W. Treatment options for large hemispheric stroke. Neurology 2001; 57: 561-8.
- 49. Georgiadis D, Schwarz S, Aschoff A, Schwab S. Hemicraniectomy and moderate hypothermia in patients with severe ischemic stroke. Stroke 2002; 33: 1584-8.
- 50. Yang XF, Wen L, Shen F, Li G, Lou R, Liu WG, et al. Surgical complications secondary to decompressive craniectomy in patients with a head injury: a series of 108 consecutive cases. Acta Neurochir. 2008; 150: 1241-8.
- 51. Honeybul S. Decompressive craniectomy: a new complication. J Clin Neurosci. 2009; 16: 727-9.
- 52. Chang V, Hartzfeld P, Langlois M, Mahmood A, Seyfried D. Outcomes of cranial repair after craniectomy. J Neurosurg. 2009. Available from: http://www.ncbi.nlm.nih.gov/pubmed/ 19612971 [cited 12 October 2009].
- 53. Yang XF, Wen L, Li G, Zhan RY, Ma L, Liu WG. Contralateral subdural effusion secondary to decompressive craniectomy performed in patients with severe traumatic brain injury:

- incidence, clinical presentations, treatment and outcome. Med Princ Pract. 2009; 18: 16-20.
- 54. Stiver SI. Complications of decompressive craniectomy for traumatic brain injury. J Neurosurg. 2009; 26. Available from: http://thejns.org/doi/full/10.3171/2009.4.FOCUS0965?cookieSet =1 [cited 24 September 2009].
- 55. Budde M, Fritsch MJ, Mehdorn HM. Complications and revision rate of cranioplasty following decompressive craniectomy; 2007. Available from: http://www.egms.de/en/meetings/ dgnc2007/07dgnc065.shtml [cited 20 September 2009].
- 56. Gooch MR, Gin GE, Kenning TJ, German JW. Complications of cranioplasty following decompressive craniectomy: analysis of 62 cases. Neurosurg Focus. 2009; 26: 1-7.
- 57. Liang W, Xiaofeng Y, Weiguo L, Gang S, Xuesheng Z, Fei C, et al. Cranioplasty of large cranial defect at an early stage after decompressive craniectomy performed for severe head trauma. J Craniofac Surg. 2007; 18: 526-2.
- 58. National Trauma Research Institute, Australia. The DECRA trial: early decompressive craniectomy in patients with severe traumatic brain injury; 2009. Available from: http://clinicaltrials. gov/show/NCT00155987 [cited 27 September 2009].
- 59. Corteen E, Timofeev I, Kirkpatrick P, Hutchinson P. The RESCUEicp study of decompressive craniectomy: implications for practice. Br J Neurosci Nurs. 2007; 3: 428-33.
- 60. Rengachary SS, Batnitzky S, Morantz RA, Arjunan K, Jeffries B. Hemicraniectomy for acute massive cerebral infarction. Neurosurgery 1981; 8: 321-8.
- 61. Bullock MR, Chesnut R, Ghajar J, Gordon D, Hartl R, Newell DW, et al. Surgical management of traumatic parenchymal lesions. Neurosurgery 2006; 58: 25-46.
- 62. Mori K, Nakao Y, Yamamoto T, Maeda M. Early external decompressive craniectomy with duroplasty improves functional recovery in patients with massive hemispheric embolic infarction: timing and indication of decompressive surgery for malignant cerebral infarction. Surg Neurol. 2004; 62: 420-30.
- 63. Ziai WC, Port JD, Cowan JA, Garonzik IM, Bhardwaj A, Rigamonti D. Decompressive craniectomy for intractable cerebral oedema: experience of a single centre. J Neurosurg Anaesthesiol. 2003; 15: 25-32.
- 64. Schneider GH, Bardt T, Lanksch WR, Unterberg A. Decompressive craniectomy following traumatic brain injury: ICP, CPP and neurological outcome. Acta Neurochir. 2002; 81: 77-9.
- 65. Meier U, Ahmadi S, Killeen T, Al-Zain FT, Lemcke J. Long term outcomes following decompressive craniectomy for severe head injury. Acta Neurochir. 2009; 102: 29-31.
- 66. Meier U, Lemcke J, Reyer T, Gräwe A. Decompressive craniectomy for severe head injury in patients with major extracranial injuries. Acta Neurochir (Suppl). 2006; 96: 373-6.

*Patrick O. Eghwrudjakpor

Department of Surgery University of Port Harcourt Teaching Hospital Port Harcourt, Nigeria. Email: patejakpor@yahoo.com