Case Report
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Frontal Cortex Electrical Changes during Infratentorial Neurosurgery: A Case Series

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Abstract

The posterior fossa is usually discussed as a separate entity from supratentorial structures, with no distinct correlation described, between infratentorial manipulation and changes in supratentorial cortical electrophysiology. We report a series of four cases of infratentorial tumour surgeries showing abrupt slowing of frontal cortical electrical activity intraoperatively which were ruled out from being artefactual. In three of the cases, the changes were transient with no lasting effects on postoperative outcome. In one case, patient had prolonged recovery after anaesthetic withdrawal and poorer neurological outcome. A mechanism involving reticular formation and its effect of cortical electrical activity has been proposed based on previous neurophysiology literature.

Keywords: Frontal cortex, Infratentorial, Neurosurgery

1. Introduction

Frontal cortex electrical activity is usually used as a guide for monitoring the depth of hypnosis during the intraoperative period. The changes observed in the electrical activity with alterations in the depth of anaesthesia are usually gradual and predictable, which are exploited by the current hypnosis monitors. Abrupt changes are unusual and are mostly electrical or mechanical signal artefacts not recognized by the monitoring algorithms or acute damaging influences on the frontal cerebral cortex such as ischaemia, hypoxia, and seizure activity. In this case series, we report four patients who manifested with abrupt onset frontal cortical delta activity during posterior fossa surgeries, without any identifiable causative factors.

Informed written consent from two adult patients and relatives of one adult and one adolescent patient were obtained for publishing this report.

2. Case Description

Case 1

A 30-year-old female, presenting with complaints of difficulty in hearing in the right ear was diagnosed with a right cerebello-pontine angle schwannoma. She was scheduled for a retromastoid suboccipital craniectomy and tumour decompression with facial nerve monitoring. Anaesthetic protocol was modified to exclude neuromuscular blockade and Entropy™ monitor was applied on contralateral forehead to monitor and titrate the depth of anaesthesia. Anaesthesia was maintained with 50% N2O and sevoflurane titrated to maintain State Entropy value between 40 and 50. During tumour decompression a sudden decline in both Response and State Entropy values to a trough of 12 – 15 was noted (Figure 1A), without any explanatory change in the anaesthetic depth or physiological parameters (blood pressure, temperature and haemoglobin oxygen

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saturation). The electroencephalograph (EEG) of Entropy monitor showed visible flattening and slowing of EEG waveform. (Figure 1B) Surgeon was notified, however, due to idiopathic nature of the observation, surgery was continued. The EEG and Entropy reverted spontaneously to pre-event values within 10 minutes. (Figure 1A) Rest of the surgery was uneventful. Post-surgery, the patient was conscious and responding to verbal commands without any motor deficits and hence trachea was extubated after elective ventilation for 36 hours.

Case 2

This patient was an adult male, aged 30 years who presented with complaints of unilateral hearing loss and unsteadiness of gait, and was diagnosed with a left vestibular schwannoma on a magnetic resonance imaging (MRI) scan. Anaesthetic protocol was similar to the previous case, except for substitution of sevoflurane with isoflurane. Intraoperative EEG and Entropy changes were similar to the previous case with respect to its timing of occurrence (during tumour decompression), duration, non-association with alterations in physiological parameters and spontaneous reversion. The intensity of decline was higher in this patient with Entropy values declining to 7. (Figure 1 C, D) Postoperative recovery was uneventful and the trachea was extubated in the operating room after surgery.

Case 3

This patient was a 21-year-old male with similar complaints, diagnosis, operative procedure and anaesthetic technique. During the decompression of tumour, there was a sudden increase in the Entropy value to 65 without any artefactual alterations in EEG waveform, which sustained for 75 seconds followed by decline to burst suppression range. (Figure 1E, F) The burst suppression ratio increased to more than 65% and was sustained at that level throughout the surgery despite decreasing isoflurane dial concentration to 0.5%. There were no changes in physiological parameters throughout this episode. Following completion of surgery, the patient remained unresponsive with entropy values ranging from 18 – 22 even after 30 minutes of anaesthetic withdrawal. Bipolar ipsilateral frontal EEG was applied separately which showed similar waveform with delta band ratio (DBR) of 92% and spectral edge frequency (SEF) of 4.7 Hz. The patient was ventilated in the intensive care unit (ICU). He gradually improved over the next 7 days to M5

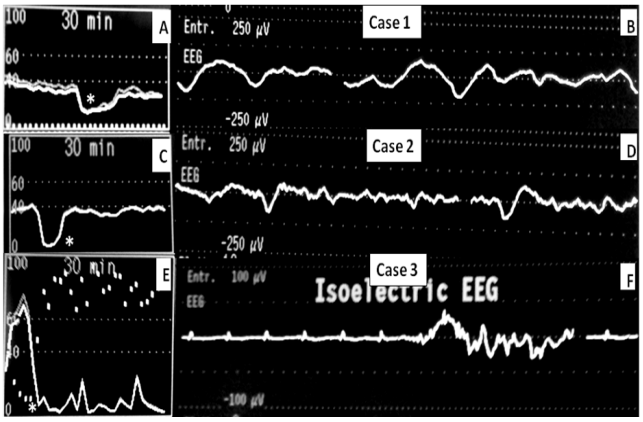

Figure 1. Showing trends of Entropy changes during surgery (A, C, E) and EEG waveforms during these events (B, D, F). White asterisks on images A, C, E denote the timing of capture of the adjacent waveform.

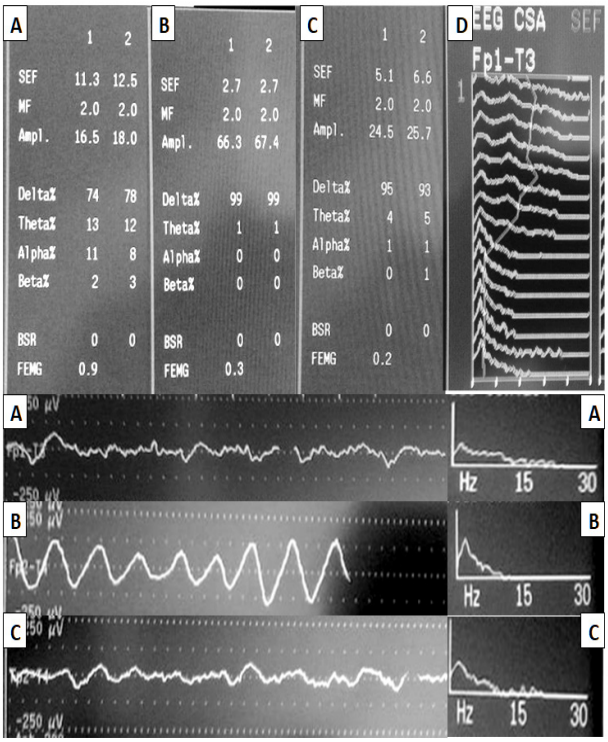

Figure 2. Showing quantitative EEG parameters, EEG waveform and power spectral density (PSD) during pre-event (A), Event (B) and post-thiopentone (C) in patient 4. 2D indicates compressed spectral array (CSA) with abrupt change in PSD during the event. Grey vertical line overlying the PSD graphs represents spectral edge frequency (SEF).

status on the Glasgow coma scale (GCS) and extubation was carried out after 2 weeks.

Case 4

This patient was a 17-year-old male presenting with unsteadiness of gait and headache, and diagnosed to have a midline posterior fossa mass lesion on a MRI scan. He was scheduled for decompression of the tumor through a midline suboccipital craniectomy. Anaesthetic technique included induction with thiopentone and maintenance with 50% N2O and sevoflurane 1.5%. Bilateral fronto-temporal bipolar EEG montages were applied for anaesthetic titration to maintain SEF between 10 and 14 Hz. During tumor decompression, a sudden bilateral change in the EEG character was noted from a low-amplitude fast activity to a high-amplitude slow sinusoidal oscillation. The SEF reduced from 12 Hz to 2.5 Hz, DBR increased from 75% to 99% and amplitude from 17 μV to 65 μV. (Figure 2 A, B) No causative etiology could be discerned from prevalent physiological parameters or anaesthetic depth. Thiopentone 100 mg was administered which led to amelioration of the sinusoidal oscillations and reversal of the changes in the EEG parameters. (Figure 2 C) Postoperative neurological examination did not reveal any deficits and hence the trachea was extubated in the operating room.

3. Discussion

Abrupt changes in the EEG waveform and processed EEG parameters during surgery usually signify either mechanical disruption of electrode contact to the scalp (via blood soakage) or a myriad of electrical signal interferences. Mechanical disruption usually leads to increased impedance at the electrode-skin interface and is picked up with impedance testing. In all our patients, impedance was within normal range for Entropy processing or ranged from 2-5 kΩ in EEG during these events. Electrical signal interference, being usually high frequency and high amplitude signals, are either filtered out by the low pass filters or diagnosed as artefact by artefact detection algorithms and discarded, or is incorporated into the EEG signal being processed and would cause increases in entropy values due to increased irregularity of the signal. No electrical signal contamination should, conceptually, cause a reduction in the entropy values. The visible EEG waveform changes mirrored the entropy changes in our patients and can thus be deemed to be true events, rather than artefactual.

As no identifiable changes occurred in the physiological parameters during these events and also because of the spontaneous reversal of the changes in the first three patients, the possible causative factor was thought to be surgical. However, posterior fossa surgery leading to cortical EEG changes has not been described till date in literature. A recent case report describes discordance between the GCS and the bispectral index in a patient with traumatic brain injury induced midbrain contusion due to cortical EEG preservation[1]. However, neurophysiology literature extensively describes the influence of brainstem, specifically, the reticular formation (RF) in arousal and activation of cerebral cortex.[2] Two distinct pathways, a thalamic and an extrathalamic via hypothalamus have been described, via which cortical activation can be achieved by high frequency stimulation of the RF or the subcortical relay stations[2]. Most such studies have been conducted in animal models and distinct neurotransmitter bundles have been delineated, with cholinergic and nor-adrenergic fibres promoting high frequency activity and GABA-ergic fibres having an inhibitory influence on adjacent cholinergic fibres and cortical neurons promoting slow wave activity[3]. Based on our observations in four patients during posterior fossa neurosurgery, we hypothesize that the observed changes in the frontal electrical activity may be caused by transient mechanical disruption of the RF (akin to neurapraxia), during surgical manipulation. The transient increase in entropy values in the patient 3 is likely from the mechanical stimulation of the RF followed by neurapraxic decline.

The initial changes in patient 4 were similar to the previous ones; however, the high frequency activation after thiopentone administration does not lend itself to probable explanation. Paradoxical excitation (PE) of cortical EEG after low dose anaesthetic administration is a well known phenomenon. McCarthy *et al* explained the phenomenon using neuronal network models as emergence of high frequency interneuron anti-synchrony due to an interaction of synaptic $GABA_A$ current and intrinsic membrane slow potassium current which causes the post-synaptic neuron to be in an excited state even during the inter-spike interval[4]. These changes were explained in context of low dose propofol, and may be extrapolated to other anaesthetics such as thiopentone, displaying the paradoxical excitation phenomenon. We

hypothesize a similar mechanism for explaining the events in patient 4.

The clinical implications of these observations are unknown given the transient nature of these changes and absence of impact on the postoperative neurological outcomes in three of the four patients. However, prolonged recovery after surgery and poor clinical outcome in patient 3 who demonstrated persistent slowing of frontal cortical activity despite the use of minimal anaesthetic reflects the likely utility of such changes in predicting postoperative outcomes. Unlike three patients who had uneventful recovery after transient changes, this patient had irreversible change in the electrical activity after the event. Though the differentiation between transient and irreversible change increases the confidence in making prediction about postoperative outcomes, such conclusions based on small observations should be made with caution. However, since such events are few in clinical neuroanaesthesia practice, studies to decipher the exact mechanisms and make definitive predictions would be difficult to design.

4. Conclusion

EEG based functional cortical monitoring plays a significant role in neuroanaesthesia practice both to monitor the adequacy of anaesthesia and also to detect critical events during important phases of neurosurgery. These monitoring tools add value not only during supratentorial surgeries but also have a role during infratentorial surgeries as demonstrated in this report. A prospective study with a large sample is essential to strengthen or refute these observations and define the role of such monitoring in routine clinical neuroanesthesia practice.

5. References

1. Chakrabarti D, Ramesh VJ, Manohar N. Brainstem Contusion: A Fallacy of GCS-BIS Synchrony. Journal of Neurosurgical Anesthesiology. 2016; 28:429-30. https://doi.org/10.1097/ANA.0000000000000215 PMid:26274623
2. Jones BE. Arousal Systems. Front. Biosci., 2003; 8:s438-s451. https://doi.org/10.2741/1074 PMid:12700104
3. Jones BE. Activity, modulation and role of basal forebrain cholinergic neurons innervating the cerebral cortex. Prog Brain Res. 2004; 145:157-69. https://doi.org/10.1016/S0079-61230345011-5.
4. McCarthy MM, Brown EN, Kopell N. Potential network mechanisms mediating electroencephalographic beta rhythm changes during propofol-induced paradoxical excitation. J Neurosci. 2008; 28:13488-504. https://doi.org/10.1523/JNEUROSCI.3536-08.2008 PMid:19074022 PMCid:PMC2717965